Inconsistency in Reaction Time: Normal Development and Group Differences Between Those with Attention Deficit / Hyperactivity Disorder and Controls

by

Benjamin Robert Williams
B.A., University of Western Ontario, 1997
M.A., Brock University, 1999

A Dissertation Submitted in Partial Fulfillment of the Requirements for the Degree of

DOCTOR OF PHILOSOPHY

in the Department of Psychology

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Abstract

Moment-to-moment fluctuation in performance (i.e. across-trial inconsistency) was examined in 2-choice reaction time data in three separate samples of participants: healthy controls ranging in age from 6 to 81 years, and both children and adolescents with Attention Deficit / Hyperactivity Disorder (ADHD). A methodological approach was employed that allowed for the examination of inconsistency while controlling for the effects of practice, fatigue, and group differences in mean level of performance. Among healthy controls, a U-shaped curve defined the relationship between age and inconsistency with optimal performance found in those aged approximately 20 years old. In children (aged 6-12 years old), those with ADHD were significantly more inconsistent than controls, and inconsistency was related to ratings of symptoms of ADHD both at home and school. Group differences were also observed in adolescents (aged 12-17 years old) with ADHD, however, in general, differences were observed only in those participants who also had reading difficulties (RD). Inconsistency was also examined separately in each end of the reaction time distribution. Evidence for a specific effect selectively affecting the slow portion of the distribution was found across all three
samples. However, in addition, there was also evidence for effects that were general to both the fast and slow portions of the distribution (in the normal population in those age 6 to 20 years), as well as evidence for an effect selectively affecting the fast portion of the distribution (in adolescents with and without ADHD). The findings indicate that in addition to traditional outcomes of interest (e.g. mean level of performance), moment-to-moment fluctuation in cognitive performance is an important phenomenon which should be taken into account in future research in developmental psychology and psychopathology.
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Dedication

To Kim, my partner in life: You have given to me in such abundance that I will never be able to repay you.
General Introduction

Within research in psychology, it is typically assumed that outcome measures reflect relatively stable characteristics of a person. As a consequence, mean level of performance, or even a single sample of performance is employed as the primary outcome of interest, and within-person variation in performance over short intervals is typically regarded as noise or error. However, Nesselroade (1991a) advocates a view of human development and psychological functioning that is characterized by change. This variability is manifest not only in relatively enduring changes such as those reflective of development and learning, but also in short-term, rapid changes that are more or less reversible. Nesselroade (1991b) refers to the latter as intraindividual variability, which he indicates constitutes “a coherent, interpretable steady-state “hum” that describes the base condition of the individual (p.94)” If systematic sources of variation can be observed in intraindividual variability, it cannot be dismissed as error, and a richer understanding of psychological functioning is obtained by including this aspect of human nature in our research.

Stability and variability have been defined and labeled in many ways (e.g. Alwin, 1994; Cattell, 1957; Fiske & Rice, 1955; Nesselrode & Featherman, 1997), and on occasion, the same label has been applied to different types (e.g. Christensen et al., 1999; Shammi, Bosman, & Stuss, 1998). The conceptualization adopted for the current undertaking is provided below and is consistent with that used by others (e.g. Christensen et al., 1999). Briefly, Hultsch and colleagues identified three types of variability: diversity, dispersion, and inconsistency (Hultsch & MacDonald, 2004; Hultsch, MacDonald, & Dixon, 2002). Diversity refers to between-person variation; that is,
differences between persons on a single task measured at one time. The other two types refer to variability within persons. Dispersion refers to within-person variability in performance across different tasks measured on a single occasion. This type of variability resembles the notion of scatter or profile analysis as discussed within the clinical literature. Inconsistency involves within-person variability in performance on a single task measured on multiple occasions, either across testing sessions or across separate trials within the same testing session. Moreover, inconsistency is defined as that part of within-person variability that cannot be accounted for by systematic time-related changes such as practice effects or fatigue.

Inconsistency is the focus of this paper. Following Nesselroade’s (1991a) conception of intraindividual variability, inconsistency refers to relatively short-term changes in behaviour (e.g. states, moods, transient fluctuations in performance) that are more or less reversible and occur more rapidly than relatively enduring intraindividual changes typically construed as learning or development.

Reaction time (RT) provides an appropriate context for the examination of inconsistency. In addition to examining mean performance over a number of trials, a measure of inconsistency, such as the intra-individual standard deviation (ISD), can also be derived. Inconsistency in RT appears useful in distinguishing various special populations from healthy individuals. For example, individuals with mild dementia exhibit higher levels of inconsistency than both healthy adults and those with arthritis suggesting that inconsistency may reflect neurological compromise rather than deterioration of general health (Hultsch, MacDonald, Hunter, Levy-Bencheton, & Strauss, 2000; Strauss, Macdonald, Hunter, Moll, & Hultsch, 2002).
The examination of inconsistency presents a number of methodological challenges. First, inconsistency must be disentangled from systematic effects that accompany repeated measurement such as practice and fatigue (Fiske & Rice, 1955). Moreover, group differences in mean level of performance can present a confound for measures of inconsistency (e.g. ISD). For example, one might expect the ISD of a RT to be larger in older adults simply because their mean group RT is larger. In order to account for these challenges, all observable systematic effects in level of performance are removed before inconsistency is examined (Hultsch & MacDonald, 2004; Hultsch et al. 2000; Hultsch et al., 2002).

*Inconsistency and Development*

Inconsistency in RT has been useful in examinations of basic issues in development. Nesselroade (1991a) conceived of a developmental fabric consisting of a dynamic weave of intraindividual change and intraindividual variability. That is, an individual’s performance at any one point in time should be understood within the context of long-term enduring change over the lifespan, as well as short-term or momentary fluctuations in performance. As such, there may be individual differences in inconsistency. Consistent with this notion, a number of studies with adults have shown that higher levels of inconsistency are associated with late, as compared to early, adulthood (Salthouse, 1993; Fozard, Verberruysen, Reynolds, Hancock, & Quilter, 1994; Shammi, Bosman, & Stuss, 1998; Anstey, 1999; Hultsch et al. 2002). However, the developmental course is unclear, particularly in childhood (Li & Lindenberger, 1999). Moreover, age group comparisons in adulthood have resulted in some equivocal results; Shammi et al. (1998) found that age group differences in inconsistency in choice RT
disappeared after mean response time was controlled, and West, Murphy, Armilio, Craik, and Stuss (2002) reported no group differences between younger and older adults in inconsistency on a basic choice reaction-time task. To date, there has been one study that has examined inconsistency across the life span and demonstrated that it follows a u-shaped curve with increases in age through childhood associated with lower inconsistency and increases in age through adulthood associated with higher inconsistency (Li et al., 2004). However, this study did not take into account the methodological issues associated with repeated measurement such as practice and fatigue. Accordingly, a primary goal of this dissertation is to examine inconsistency across the life span while controlling for these potentially confounding variables.

Within in-depth investigations into the nature of inconsistency in RT one question that has arisen is whether differences in inconsistency are observed across the entire RT distribution, or if the inconsistency effect is limited to a specific aspect of the distribution (i.e. the tail at the slow end of the distribution). The former implies some general process, while the latter might imply the effect of some specific variability-producing process that selectively affects the slow end of the RT distribution, such as attentional lapses.

To address this issue, some researchers have fit their data to an ex-Gaussian distribution (Leth-Steensen et al. 2000; West, 2001; West et al., 2002). The ex-Gaussian model conceptualizes an individual’s RT distribution as a combination of a Gaussian (i.e. normal) and an exponential distribution. The model allows for the estimation of quantitative measures of central tendency (mu) and spread (sigma) of the normal component, as well as a measure of the size of the exponential component (i.e. degree of
positive skew or size of the tail) (tau). This allows one to identify a specific type of inconsistency effect that is due to a relatively small number of very slow responses that might be obscured from observations of the parameters of the normal distribution, when considered alone, such as mean and standard deviation. That is, a small number of very slow responses could result in group differences in mean reaction time that might be erroneously interpreted as consistent mean differences in level of performance. Alternatively, such data could result in group differences in intra-individual standard deviation (ISD) that might be erroneously interpreted as greater deviation from the mean in both directions.

Leth-Steensen et al. (2000) demonstrated that boys with ADHD did not differ from normal controls on the measures of central tendency and general spread within the ex-Gaussian distribution, but did differ on the measure of size of the tail. It was concluded that the slow and more variable responding consistently observed in ADHD children is likely the result of a specific process that selectively affects the slow portion of the RT distribution such as attentional lapses (Leth-Steensen et al., 2000). Of note, this study included both younger (age 7) and older (ages 9-13) normal control groups providing for an examination of normal development. In contrast to the ADHD children, the RT distributions of the younger normals were characterised by high degrees of all three of the ex-Gaussian parameters suggesting that in addition to attentional lapses, the younger boys’ responding was characterised by more generalised processing difficulties (Leth-Steensen et al., 2000).

West et al. (2002) also used the ex-Gaussian model to examine age-related differences in inconsistency in adults. These effects were examined on a 1-back RT task,
a measure that includes a working memory load. Older adults were found to differ consistently from younger adults only on the tau component. As age-related differences in inconsistency were observed in the 1-back task and not on a simple choice RT task, West et al. (2002) concluded that the specific effect observed was the result of fluctuations in the efficiency of executive control processes in older participants.

Salthouse (1993) described another technique to examine this same issue that uses separate evaluations of the slow and fast ends of the RT distribution. Salthouse (1993) argued that if a specific variability-producing process, such as attentional blocks, was related to aging, (1) age-related differences should be observed in the slow end of the RT distribution and not the fast end, and (2) there should be independent age-related variance in the slow end of the RT distribution after controlling for the age-related effects of the fast end of the distribution. In an examination of mean level of response in both ends of the distribution, Salthouse (1993) did not find evidence for the attentional block hypothesis. However, in a modification of this technique whereby inconsistency was examined in each end of the RT distribution, Hultsch et al. (2002) did find support for the attentional block hypothesis. Throughout adulthood, the age-related effect of inconsistency was much more pronounced in the slow end rather than in the fast end of

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1 Although results consistent with the conditions set-out by Salthouse (1993) would provide a convincing argument that age-related differences in inconsistency are due to some mechanism that selectively affects the slow portion of the RT distribution, a number of other outcomes within the proposed analyses would prove informative with respect to the nature of any overall inconsistency effect observed. First, it would be similarly as convincing that the overall effect was due to some general process if a significant difference were found in the fast portion of the distribution and the slow effect was removed by first partialling out the effect from the fast end. However, one cannot rule-out the possibility of finding a statistically significant difference in both the fast and slow end of the distributions, and that either or both of these effects remain statistically significant with the opposite effect partialled. One must also consider that in addition to unique sources of variation, there may be shared variance between the slow and fast portions of the RT distribution. Thus, an analysis whereby one first examines group differences in inconsistency in the fast and slow portions of the RT distribution separately, followed by analyses of these same effects with the other partialled, could provide evidence for two unique sources of variance (i.e. present in the slow and not in the fast and vice versa) as well as shared variance (i.e. general to both portions of the distribution).
the RT distribution, and the age effect on inconsistency in the slow end of the distribution was attenuated only by a small degree when inconsistency in the fast end of the distribution was statistically controlled.

_Inconsistency in ADHD_

In addition to examinations of the normal development of inconsistency, a second focus of this dissertation is inconsistency as a marker of pathology, in particular, developmental psychopathology. The concept of moment-to-moment variability in performance (i.e. inconsistency) as a marker of neurological compromise has been evaluated in adults (e.g. Hultsch et al., 2000; Strauss et al., 2002). It is also not novel to those studying ADHD, a childhood disorder that is characterised by inattention, hyperactivity, and impulsivity (American Psychiatric Association, DSM-IV-TR, 2000). Although rarely the primary focus of research, the literature is replete with findings of differences between ADHD and various control groups on inconsistency in RT across a wide variety of task manipulations (e.g. Leth-Steensen, Elbaz, & Douglas, 2000; Steger et al., 2001; Hynd et al., 89; Scheres, Oosterlaan, & Sergeant, 2001; Kuntsi, Oosterlaan, & Stevenson, 2001; Rucklidge & Tannock, 2002).

The ubiquity of such findings has prompted researchers to begin to incorporate some type of accounting for inconsistency in theoretical models of ADHD. First, Castellanos and Tannock (2002) hypothesized that inconsistency arises from deficits in temporal processing which are thought to be a primary characteristic of ADHD that is more closely related to its causative factors than the behavioural symptoms which form the basis of its diagnosis. Indeed, according to a twin study conducted by Kuntsi and Stevenson (2001), there is a statistically significant relationship between genetic effects
of extreme hyperactivity and within-subject variability of reaction time. Second, Douglas (1999) characterises inconsistency as one of several pervasive regulatory problems that provides a context within which more specific regulatory deficits such as sustained effort, response preparation, adaptation to changing demands, and maintaining focus must be understood.

Although it may be conceptualized in different manners, inconsistency appears to be emerging as an important phenomenon with respect to the cognitive and neuropsychological performance of individuals with ADHD, and is causing researchers to consider change over short periods of time in their accounts of such performance. As previously indicated, Leth-Steensen et al. (2000) probed findings of group differences in mean and standard deviation by fitting reaction time data to the ex-Gaussian distribution. They found that boys with ADHD did not differ from normal controls on mu and sigma, but did differ on tau. This suggested that the group differences could be best accounted for by a higher level of inconsistency in the ADHD boys resulting from a relatively larger number of very slow responses. Leth-Steensen et al. (2000) concluded that the slow and more variable responding consistently observed in ADHD children is likely the result of a specific variability-producing process such as attentional lapses. Recently, Hervy et al. (2004) replicated the findings of the Leeth-Steensen et al. (2000) study in a large sample that was tested on the Connors Continuous Performance task. There was a statistically significant difference between ADHD and controls on tau in the absence of a difference in sigma. Interestingly, there was a mean difference in mu whereby ADHD children were faster than the matched controls (Hervy et al., 2004).
Examinations of inconsistency in ADHD have been limited to the childhood age range to the exclusion of adolescents. Moreover, none of the studies completed to date have addressed the methodological issues described earlier. This is likely because inconsistency has not been the primary focus of the research, and as such, researchers examining these issues have failed to control for the effects of time on task (i.e. practice and fatigue) as well as group differences in level of performance.

Study 1: Inconsistency in Reaction Time Across the Lifespan

Introduction

The purpose of the first study was to examine inconsistency in RT across the lifespan. Data from an existing study (Williams, Ponnesse, Schachar, Logan, & Tannock, 1999) of 275 participants ranging in age from 6 to 81 years were re-analysed for inconsistency. It was expected that inconsistency in RT would vary as a function of age after the effects of trial and age group in mean level of performance were removed from the data. More specifically, inconsistency was expected to follow a u-shaped curve across the age distribution such that childhood and older adulthood would be associated with higher levels of inconsistency than early adulthood.

A second aim of the study was to examine the nature of any age-related variance in inconsistency. Following the method used by Hultsch et al. (2002), separate evaluations of inconsistency in the fastest and slowest 25% of responses were evaluated. If the overall inconsistency effect were due to a specific age-related variability-producing process selectively affecting the slow portion of the RT distribution, it would be expected that age-related change in inconsistency would be present in the slow end of the RT distribution and absent in the fast end of the RT distribution. Furthermore, it would be
expected that partialling the variability in inconsistency in the fast end of the distribution would have little effect on the relationship between age and inconsistency in the slow end of the distribution. Alternatively, evidence for a general effect would be provided if age-related differences were observed in both ends of the RT distribution, and if variation in inconsistency in the fast end of the distribution could account for age-related variation in inconsistency in the slow end of the RT distribution.

Method

This study is based on data that were collected by researchers at the Hospital for Sick Children in Toronto, Canada. Data were collected over a two-week period in the summer of 1996 from volunteers visiting the Ontario Science Centre, a science museum located in Toronto. The participants, methods, and procedures have previously been described in detail (Williams et al. 1999). As such, only those aspects of the method pertinent to the current study are provided here.

Participants

Of a total of 284 non-related volunteers that participated in the Williams et al. (1999) study, 9 (3%) were screened out because of outlying data. Of these 275 participants, a further 2 were eliminated from the current study leaving a total sample of 273. One of the two was eliminated because of missing data, and the other was eliminated because no valid data points could be derived from the portion of the cognitive task used in the current study.

The participants ranged in age from 6 to 81 years. They were divided into seven age groups based on those used in the original study. As can be observed in Table 1,
these groupings were determined by a desire to construct age groups that reflected meaningful stages in the life cycle as well as the need to obtain groups of relatively equal sample sizes. One hundred thirty six participants were male, and 135 were female (gender data were missing for 2 participants); the gender distribution was roughly uniform across the age groups (See Table 1).

The study design was not adapted to special needs; thus, volunteers with vision, hearing, or motor function impairments and those who did not speak at least some English or French were not eligible to participate. In addition, although information on ethnicity was not collected, as the Ontario Science Centre attracts visitors from all over the world, a wide range of ethnic groups were likely represented in the sample.

**Measures**

RT data were derived from the stop signal procedure, a computer-generated cognitive measure of inhibitory control (Logan, 1994; Logan & Cowan, 1984). It involves two concurrent tasks: The “go” task, and the “stop” task. For the version of the task used in the Williams et al. (1999) study, the “go” task was a visual, two-choice reaction-time task. Participants were presented with a fixation point in the middle of the screen for 500 ms, followed by either an “X” or an “O” of 1000 ms duration. Participants were required to respond to the stimulus by pressing the corresponding button on a response box as quickly as possible. The “stop task” involved the demand that participants inhibit their response to the “go task” when a 1000 Hz tone was presented (the stop signal). Stop signals occurred on 20% of the trials.

The task consisted of 256 trials divided into eight 32-trial blocks. However, preceding the test blocks, two 32-trial practice blocks were given. The first practice
block was administered before the demand to stop was introduced. That is, participants were instructed to respond to the “go” stimuli, and to ignore the intermittent tones. The data analysed in the current study were derived from this first 32-trial practice block, which was not reported in the previous study.

Procedure

Data from questionnaires (demographic and personality) and the computer tasks were collected in two separate rooms that were located within a neuroscience exhibit of the museum. Before the participants completed the stop signal procedure, an informed consent procedure and questionnaires were first completed. The stop signal procedure was described to each participant by a researcher attending the terminal. A uniform set of instructions was read to each participant, and time was allowed for familiarization with the equipment and for any questions the participants had to be answered. A total of four participants could be tested at individual terminals at any given time.

Data Preparation

For each of the 273 participants, the raw data obtained from the stop signal procedure included 32 trials of RT data expressed in milliseconds. In order to eliminate error variance, outliers from the 8736 (32 trials * 273 participants) observations were identified and eliminated according to the following procedure. First, extremely fast data points were eliminated which might reflect accidental key presses. A lower bound level for legitimate responses was set at 150ms, and scores falling below this value were eliminated. Second, data points were eliminated if they fell above the upper bound limit which was defined as the mean plus 3 standard deviations within each age group. Values
above this cut point were expected to reflect errors such as momentary distraction of the
participant. Across the 32 trials for each participant, 3.2% of the data points were
eliminated. The proportion of observations that were identified as outliers was roughly
equivalent across the seven age groups.

Following the removal of outliers, values were imputed for the missing data
points via a regression procedure where estimates were based on the relationship of
responses across trials. This was done in order to avoid statistical problems associated
with missing data. Because eliminating outlying data points and imputing estimates for
the missing values are likely to decrease inconsistency, these procedures represent a
conservative approach to the examination of inconsistency.

Results

There were four main parts of the analysis. First, an initial mixed-model analysis
of variance ANOVA (age group by trial) was conducted on the raw choice reaction time
(CRT) data in order to determine the presence of trial and age group differences, and any
interactions between the two factors, on the raw latency scores. Second, the observed
effects on the raw CRT scores were removed from the data and individual ISD scores
were calculated for each individual across the 32 trials. Third, the primary inconsistency
analysis was conducted whereby ISD was regressed on age. Because the relationship
between age and ISD was not expected to be linear, quadratic, and cubic functions of age
were entered into the analysis. Finally, ISDs were calculated separately for the slowest
and fastest 25% of raw latency scores (i.e. slowest and fastest 8 trials). A series of
hierarchical regression analyses were conducted in order to determine if the age-related
differences in ISD could be accounted for solely by the slowest trials in the CRT distribution.

**Trial and Age Group Effects in Level of Performance**

Level of performance was not the primary outcome measure of interest in the current study. However, since we wished to control for any effects of age group and trial on the raw latency scores before inconsistency was examined, it was necessary to first confirm the presence of any such effects. In order to do so, a 7 (age group) by 32 (trial) mixed model ANOVA was conducted on CRT. Consistent with the expected result, there were statistically significant main effects for age group ($F(6, 266) = 63.24, p < .001, \eta^2 = .59$), and trial ($F(31, 8246) = 35.37, p < .001, \eta^2 = .12$). Although the effect size was small, there was also a statistically significant interaction between age group and trial ($F(186, 8246) = 1.64, p < .001, \eta^2 = .04$).

Follow-up one-way polynomial contrasts were conducted examining the effect of age group on mean CRT (i.e. collapsed across the 32 trials). The effect size was highest for the quadratic effect ($F(1, 266) = 307.92, p < .001, \eta^2 = .54$). The relationship was a U-shaped curve such that performance in the young adulthood group (age 18-29) was the fastest and the two slowest groups were the early childhood (age 6-8) and elderly groups (60-81). Means and standard deviations for the mean CRT for each of the seven age groups can be found in Table 1.

Follow-up tests were not interpreted between the 32 levels of trial. However, an examination of the means plot was suggestive of practice effects. These effects were characterised by rapid decrease in latency across the first 3 trials, followed by relative stability over the final 29 trials.
Purification of CRT and calculation of ISD

As previously indicated, the purpose of the study was to examine inconsistency (i.e. moment-to-moment within-person variability) in CRT across a wide age range. Because systematic effects, such as practice effects or age group differences in mean level of response could present a confound to an examination of inconsistency, it was important to remove these effects from the data. As such, the observed age group, trial, and age group by trial interaction effects were partialled from the 32 trials of raw CRT data from each of the 273 participants. This analysis produced residual latency scores that were uncontaminated by differences between age groups and differences across trials in mean level of performance. In order to permit comparison between the results of the current study and previous studies employing this method, the residual scores were standardized (using the mean and standard deviation for all data points) and then transformed into T scores. That is, the mean and standard deviation for all 8736 (32 trials * 273 participants) data points were 50, and 10, respectively.

Inconsistency

A graphic depiction of the residualized T scores provides a striking demonstration of inconsistency. For each of three age groups (6-8 year olds, 18-29 year olds, and 60-81 year olds), Figure 1 shows the residualized T scores plotted across 32 trials for each subject. It is interesting to note that inconsistency is apparent in the data despite the

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2 Our method of purifying the raw CRT data reflects our definition of inconsistency as intra-individual variability that is independent of systematic across-time effects such as practice and fatigue. Moreover, recognizing that age group differences in average individual standard deviations can be contaminated by age group differences in means, we include in our method the removal of age group effects. Thus, although age groups can still differ in their average inconsistency (refer to Figure 1), that difference cannot be due to a confound with systematic across-time effects, or age group differences in mean performance.
removal of age group differences and effects of trial in mean level of response. The early childhood group appears to have the most inconsistency, followed by the elderly, and then finally, the young adulthood group.

A more formal analysis of inconsistency was accomplished by examining the intraindividual standard deviation (ISD). This measure was defined as the standard deviation for each individual across the 32 residualized T scores of CRT. The mean and standard deviation for the ISDs for each group are shown in Table 1.

In order to examine the hypothesis that inconsistency would vary across age, a hierarchical multiple regression analysis was conducted. Age, expressed as a continuous variable, was entered as a predictor of ISD, followed by the quadratic, and cubic functions of age. A summary of the findings of the analysis may be found in Table 2. Although both the linear ($AR^2=.04$) and cubic ($AR^2=.13$) functions of age were statistically significant predictors of ISD, the effect size was highest for the quadratic ($AR^2=.29$) function of age. The relationship between age and ISD followed a U-shaped function: increases in age throughout childhood and adolescence to young adulthood were associated with smaller ISD, whereas increases in age throughout adulthood were associated with higher ISD (refer to Figure 2).

A series of analyses were conducted in order to examine the nature of the inconsistency found across the entire distribution of the RT data. Is the age-related

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3 For trend analysis involving continuous variables, components of lower order trends (e.g., linear) must be included in the regression equation in order to uniquely identify the effects of higher order trends (e.g., quadratic). However, in the presence of higher order trends, the lower order trend components no longer represent main effects. For example, when Age$^2$ is included in the equation, Age does not represent the main effect of the linear component (with the quadratic partialled). As such, for this and each subsequent polynomial regression analysis, statistics are reported only for the step where they provide information relevant to the analysis. That is, effect size is reported for the step in which the effect in question and all lower-order polynomials were included in the regression equation.
effect in inconsistency limited to the slow end of the RT distribution as opposed to the fast end? And if not, do similar processes contribute to the inconsistency found in both ends of the distribution? In order to answer these questions, ISDs (using residual CRT scores) were calculated separately for both tails of the RT distribution. Each individual's raw CRT distribution was sorted in ascending order, and the fastest 25% (8 trials) and slowest 25% (8 trials) of trials were selected and submitted to the residualisation process detailed above. The only difference being that the CRT scores were purified by age group only, not trial and age group by trial interactions. The CRT data were not purified by trial for this analysis as trial order was lost when the data were sorted in ascending order. This resulted in two ISDs for each participant reflecting inconsistency in the slow end of the CRT distribution (slow ISD), and the fast end of the CRT distribution (fast ISD).

The linear, quadratic, and cubic functions of age were entered in two hierarchical regression analyses, one for each of the fast and slow ISDs. All three functions of age were statistically significant predictors of the fast ISDs; however, the effect for the quadratic ($\Delta R^2 = .12$) function of age was stronger than both the linear ($\Delta R^2 = .05$) and cubic ($\Delta R^2 = .08$) functions. The strength of effect for slow ISD was also the highest for quadratic ($\Delta R^2 = .14$) function of age. However, while a statistically significant proportion of the variance in slow ISD was predicted by the cubic ($\Delta R^2 = .05$) function of age, this was not the case for the linear ($\Delta R^2 = .001$) function of age (Refer to Table 2).

Subsequently, another hierarchical regression analysis was conducted in order to determine if the age-related variance in slow ISD could be accounted for by fast ISD. As such, fast ISD and all three functions of age were entered as predictors of slow ISD in
separate steps. After the variability in slow ISD associated with fast ISD ($\Delta R^2 = .04$) was accounted for, both the quadratic ($\Delta R^2 = .10$) and cubic ($\Delta R^2 = .04$) functions of age accounted for statistically significant proportions of the variance in slow ISD. As can be observed, the quadratic function of age accounted for 4% less of the variability in slow ISD after the variability attributable to fast ISD had been accounted for. A summary of these results can be found in Table 2.

The regression analyses examining inconsistency in the two tails of the RT distribution were then repeated for two halves of the sample: Younger participants (age < 21, n = 135), and older participants (age >= 21, n = 138). For these analyses the cubic effect of age was not entered as only half of the age distribution was under consideration. Similar results were found in the younger participants as were found in the overall sample; that is, there was statistically significant age-related variation in both the slow and fast ends of the RT distribution, and inconsistency in the fast end of the distribution accounted for some variation in the slow end of the distribution. However, in the older portion of the sample, only a small proportion of the variance in fast ISD was accounted for by age. Moreover, the age-related variance in slow ISD was not reduced by first accounting for the variability in fast ISD for this age group. Refer to Tables 3 and 4 for summaries of these results.

Discussion

Nesselroade (1991a;1991b) conceived of a developmental fabric consisting of a dynamic weave of intraindividual change and intraindividual variability; however, little research has addressed issues of basic normal development in intraindividual variability. This paper is the first to examine inconsistency in RT across the life span while
controlling for the potential confounds of practice and fatigue, and age group differences in mean level of response. Consistent with the notion of individual differences in moment to moment change, inconsistency varied across age. The relationship between age and inconsistency was best characterised by a u-shaped curve: increases in age throughout childhood were associated with lower levels of inconsistency, whereas increases in age across adulthood were associated with higher levels of inconsistency. The effect size was large: approximately 29% of the variability in inconsistency was accounted for by the quadratic function of age. Moreover, this relationship was established independent of age-related differences in mean speed of response and independent of the effects of practice and fatigue.

It should be noted that a statistically significant cubic trend was also observed in the data. Although the effect was smaller than that for the quadratic effect, the cubic effect accounted for approximately 13% of the variability in inconsistency, an effect in no sense insignificant. This was likely the result of higher levels of diversity in the latter half of the age distribution. Although no formal tests of diversity were conducted, an inspection of the scatter plot (refer to Figure 2) reveals a larger spread of scores with increasing age beginning between ages 30 and 40. Greater diversity would allow a wider range of curves to be fit to the data. Although the overall trend appears to clearly be quadratic in nature, the observed cubic trend highlights the importance of individual differences in inconsistency, particularly in the latter half of the age distribution.

In an attempt to tie psychological functioning to concomitant neurobiological functioning, some researchers (Li & Lindenberger, 1999) have postulated a link between age-related changes in inconsistency and age-related changes in the activity of the
catecholaminergic system. More specifically, they ascribe to a neural-noise hypothesis whereby catecholamines play a key role in modulating the signal-to-noise ratio of neurons by enhancing their responsivity to other incoming signals. That is, age-associated changes in catecholaminergic activity is thought to be associated with alterations in the responsivity of neurons and thus increases or decreases in neural noise (Li & Lindenberger, 1999). At the behavioural level, increases in neural noise may manifest in higher levels of inconsistency. A more complete understanding of the mechanisms involved in inconsistency is likely to be informed by investigations spanning the behavioural and neurobiological levels of analysis.

Important information regarding the nature of the inconsistency was revealed by analyses conducted in the fast and slow tails of the RT distribution. At first glance, the results from the entire sample appeared to be inconsistent with previous research in adults suggesting that age-related differences might be explained by attentional lapses (i.e. age effects in inconsistency were driven primarily by the slow end of the RT distribution) (Hultsch et al., 2002). In the current investigation, comparable quadratic age effects were observed in inconsistency in both the slow and fast ends of the RT distribution. Moreover, the quadratic effect of age accounted for 29% less of the variability in inconsistency in the slow tail after the variability in the fast tail had been accounted for. This finding supports an interpretation that both general and specific variability-producing processes may be influencing age-related differences in inconsistency.

These analyses were then repeated in two halves of the age distribution in order to determine if the findings were consistent across childhood and adulthood. In the younger
half of the sample (age <21), similar findings were observed as in the entire sample: age accounted for a significant proportion of variance in inconsistency in both tails of the RT distribution, and partialling inconsistency in the fast tail resulted in a substantial reduction in the age-related variance in the slow tail (the linear effect of age was of primary significance since only half of the age distribution was under consideration). This, taken with the finding that there was significant age-related variance in inconsistency in the slow end of the tail that was unique from age-related variance in the fast tail indicated influences of both general and specific factors for the younger sample. This interpretation is consistent with previous research in children using ex-gaussian parameter estimates suggesting that the RT distribution of younger children is characterised by both increased general spread and larger tails (Leth-Steensen et al., 2000).

In contrast, in the older half of the sample (age >=21), age accounted for a statistically significant, albeit, much smaller proportion of the variance in inconsistency in the fast tail (4%) as compared to the slow tail (15%). Moreover, after removing variance in inconsistency in the slow end of the tail that was due to inconsistency in the fast end of the tail, there was no reduction in the age effect. These results are consistent with previous research indicating that age-related differences in inconsistency through adulthood may be related to a specific variability-producing process influencing the data in the slow end of the distribution only, such as attentional lapses (Hultsch et al., 2002).

This paper supports a growing body of literature demonstrating that in addition to level of performance, fluctuations in performance constitute an important psychological phenomenon. It should be noted that age related differences in inconsistency were
observed in a simple choice reaction time task, a basic measure of speed of information processing. As such, within-person moment-to-moment variability provides an important context within which much of the research literature in psychology should be evaluated and interpreted. Evidence has been presented that demonstrates that inconsistency varies across age throughout the lifespan independent of age-related differences in mean level of performance. Age-related change in inconsistency through childhood appears to be related to both general and specific processes, while inconsistency through adulthood appears to be largely due to specific factors influencing the slow end of the RT distribution, such as attentional lapses.

Study 2: Reaction Time Performance in Adolescents with ADHD: Evidence of Inconsistency in the Fast and Slow Portions of the RT Distribution

Introduction

The purpose of the second study was to examine inconsistency in adolescents with ADHD. First, we wished to determine if adolescents with ADHD exhibit higher levels of RT inconsistency than normal controls after controlling for the effects of practice and fatigue, and after controlling for group differences in speed of response. Moreover, we were also interested in whether this deficit was specific to ADHD or if it could also be found in those with reading difficulties (RD). RD are often co-morbid with ADHD, and there is a growing body of literature that suggests a potential common genetic link (Willcutt, Pennington, & DeFries, 2000; Willcutt et al., 2002) between the two. On the other hand, there is some limited evidence that inconsistency of response is characteristic of ADHD, but not of RD (Rucklidge & Tannock, 2000). However, studies
to date have not taken into account the measurement issues associated with repeated measurement as described above.

RT data of four separate groups (ADHD, RD, Combined ADHD+RD, and normal controls) from previous research conducted at Sick Children’s Hospital in Toronto were examined in the second study. It was expected that the ADHD groups would exhibit higher levels of inconsistency than both the normal control and RD groups after controlling for systematic across-time effects and group differences in level of performance.

A second purpose of the study was to investigate whether the high levels of inconsistency found in ADHD are due to a specific process such as attentional lapses (e.g. Leth-Steens et al., 2000), or to some process that is general across the RT distribution. It was expected that the ADHD groups would demonstrate more inconsistency in the slow end of the distribution than both the RD and the normal controls, that these group differences would not be present in the fast end of the distribution, and that the former group differences would be present after controlling for the effects of the fast portion of the distribution.

A final question focused on the relationship of inconsistency to cognitive functioning. We wished to investigate whether inconsistency in RT is correlated with level of performance on common measures of cognitive functioning. If inconsistency is predictive of age-related differences and is able to differentiate groups based on childhood psychopathology and neurological deterioration, then inconsistency may be a predictor of cognitive and neuropsychological functioning. Hultsch et al. (2002) demonstrated that inconsistency in RT was related to a variety of cognitive measures in
adults, but little is known about the relationship between inconsistency in RT and cognitive functioning in adolescence. The pattern of relationships between inconsistency and measures of cognitive functioning may provide additional insight into the nature of inconsistency.

Method

This study is based on data that were collected by researchers at the Hospital for Sick Children in Toronto, Canada. The participants, methods, and procedures have previously been described in detail (Rucklidge & Tannock, 2000). As such, only those aspects of the method pertinent to the current study are provided here.

Participants

Data from 99 adolescents divided into 4 groups (ADHD, RD, ADHD+RD, and controls) that participated in a previous study (Rucklidge & Tannock, 2000) were available. Participants were eliminated from the current study if their accuracy on the portion of the RT task examined (32 practice trials of a 2-choice RT task) did not reach 90%. This resulted in the elimination of 27 participants who were distributed relatively evenly across the 4 groups: 8, 5, 6, and 8 for the ADHD, RD, ADHD+RD, and controls, respectively. A total of 72 participants were included in the analyses (ADHD= 23, RD=7, ADHD+RD=13, control=29). There were 37 male and 35 female participants and they ranged in age from 12 to 17 years (mean= 14.9). See Table 5 for a breakdown of the demographic variables by group.
ADHD participants.

Adolescents in the ADHD groups were recruited from previous participants in research conducted at the hospital, new referrals to the hospital, and from ads placed in pediatric offices. Participants included in the ADHD groups had a confirmed current and childhood DSM-IV-TR (2000) diagnosis of ADHD. Diagnosis was based on standardized semi-structured clinical interviews conducted with both the adolescent and a parent, as well as standardized measures of ADHD symptomatology completed by parents, teachers, and adolescents. Specific information regarding the diagnostic protocol and algorithm used is described by Rucklidge and Tannock (2002).

Control participants.

Control participants were recruited by hospital staff and from connections within the community. Potential participants were excluded from the control group if they had a history or current complaints of hyperactivity, impulsivity, or inattention, if they met criteria for an Axis I disorder of any kind, or if they scored below the 25th percentile on standardized tests of achievement.

RD participants.

No specific recruiting of adolescents for the RD group was conducted: They were participants recruited from the other sources who were found to have reading difficulties on the screening measures used. Selection for the RD groups was based on a score that fell below the 25th percentile on one of four standardized reading and spelling tests administered. Classification of RD in this manner was justified on the basis of a rejection of the IQ-Achievement discrepancy definition as well as evidence that spelling scores are
accurate indicators of literacy and language-based skills (See Rucklidge & Tannock, 2000). Inclusion in the ADHD+RD group was based on qualifying for both the ADHD and RD groups on the criteria listed above.

The first language of all participants was English. Participants were not accepted for the study if their estimated IQ score, based on Vocabulary and block design subtests of the WISC-III (Wechsler, 1991) was below 80, or if they had uncorrected hearing or vision difficulties. All participants were free from any serious neurological disorders such as epilepsy or cerebral palsy.

Measures

Choice Reaction Time (CRT).

CRT data from which inconsistency measures were derived were obtained from the stop signal procedure, a computer-generated measure of inhibitory control (Logan, 1994; Logan & Cowan, 1984). The procedure involves two concurrent tasks: The "go" task, and the "stop" task. The version used in the current study is described by Williams, Ponnesse, Schachar, Logan, & Tannock (1999). The "go" task was a visual, two-choice reaction-time task. Participants were presented with a fixation point in the middle of the screen for 500 ms, followed by either an "X" or an "O" of 1000 ms duration. Participants were required to respond to the stimulus by pressing the corresponding button on a response box as quickly as possible. The "stop task" involved the demand that participants inhibit their response to the "go task" when a 1000 Hz tone was presented (the stop signal). Stop signals occurred on 20% of the trials.

The task consisted of 256 trials divided into eight 32-trial blocks. However, preceding the test blocks, two 32-trial practice blocks were given. The first practice
block was administered before the demand to stop was introduced. That is, participants were instructed to respond to the "go" stimuli, and to ignore the intermittent tones. The data analysed in the current study were derived from this first 32-trial practice block which was not reported in the previous study.

*Cognitive Measures.*

Measures of intellectual functioning were obtained from several sub-scales of the WISC-III (Wechsler, 1991), a standardized instrument that is commonly used in clinical settings. As noted previously, full-scale IQ was estimated by scores on the vocabulary and block design subscales. Freedom from distractibility (FDI) and processing speed (PSI) indices were also available.

The Stroop Color and Word Test (Golden, 1978) requires participants to quickly and accurately name color words, and to name the color of the ink for printed X’s, and discordant color words. The three successive 45 second trials provide for measures of speed under the three conditions as well as a measure of susceptibility to interference, controlling for color and word naming speed. This measure is conceptualised as a type of interference control in that it involves inhibition of a competing but not intended response (Nigg, 2000). That is, in the third condition, the participant must inhibit the tendency to name the color word in order to successfully name the color of the ink in which the word is printed. The type of inhibition examined by the Stroop task is thought to be part of the executive control system that permits goal-directed behaviour (Nigg, 2000).
Procedure

All data were collected within a research unit at the Hospital for Sick Children in Toronto, Canada. The interviews, screening measures, and outcome tasks took approximately 6 hours to complete. A registered psychologist conducted all clinical interviews and made diagnostic determinations. The cognitive and experimental measures were all given/collected and scored by research assistants who were blind to group identification. Those participants on short-acting stimulant medication were medication-free on testing. They were required to discontinue use of such medication 24 hours before their test day.

Data Preparation

A total of 32 trials of CRT data (resolved to the millisecond) were obtained from the stop signal procedure for each of the 72 participants (2304 observations). In efforts to reduce measurement error, outlying data points were identified and eliminated according to the following procedure. First, a lower bound level for legitimate responses was set at 150 ms, and scores falling below this value were eliminated. It was expected that such extremely fast data points might reflect errors such as accidental key presses. Second, data points were eliminated if they fell beyond the upper bound limit which was defined as three standard deviations above the mean of each group. Values above this cut point were thought to reflect errors such as momentary distraction of the participant. Across the 2304 observations, 3.3%, and 2.0% of the data points were eliminated as lower-bound, and upper-bound outliers, respectively. The proportion of observations that were
identified as outliers was roughly equivalent across the four groups.

Following the removal of outliers, values were imputed for the missing data points via a regression procedure where estimates were based on the relationship of responses across trials. Both procedures, eliminating outlying data points and imputing estimates for the missing values, were likely to decrease intraindividual variability, and thus provide a conservative examination of inconsistency.

Results

Group differences in sample characteristics and cognitive variables

A series of tests were conducted in order to provide further description of the sample with respect to demographic characteristics and the cognitive variables. Mean age was examined in a 2(ADHD) x 2(RD) factorial ANOVA. There was no statistically significant ADHD x RD interaction effect \(F(1, 68)=0.90, p=.35, \eta^2=.01\), nor were there statistically significant main effects for RD \(F(1, 68)=0.07, p=.80, \eta^2=.001\) or ADHD \(F(1, 68)=1.81, p=.18, \eta^2=.03\). Chi square test of association was used in order to examine differences in gender across the four groups. There was no statistically significant relationship between gender and the group variable \(\chi^2(3)=2.61, p=.46\).

Group differences were also examined for certain sub-tests and indices of the WISC-III and T-scores derived from the Stroop Test via 2(RD) x 2(ADHD) factorial ANOVAs. Only those effects that were statistically significant at alpha<.05 are reported below. There was a statistically significant main effect for RD on estimated full-scale IQ \(F(1, 68)=5.46, p=.022, \eta^2=.074\); the mean of those classified as RD \((M=99.12)\) was lower than those not classified as RD \((M=106.69)\). Similarly, there were statistically
significant main effects for RD on mean vocabulary score \( (F(1, 68)=11.14, p=.001, \eta^2=.141) \) and mean FDI \( (F(1,65)=21.83, p<.001, \eta^2=.251) \); Both mean vocabulary \( (M=8.84) \) and mean FDI \( (M=87.29) \) were lower for those with RD compared with mean vocabulary \( (M=11.03) \) and mean FDI \( (M=105.95) \) for those without. In contrast, there was no main effect for RD on PSI; however, there was a statistically significant main effect for ADHD \( (F(1, 64)=13.59, p<.001, \eta^2=.175) \) such that PSI was lower for those with ADHD \( (M=101.42) \) as compared to those that without \( (M=114.59) \). Individual cell means and standard deviations may be found in Table 5.

There were no statistically significant main effects nor was there an interaction in interference on the Stroop Test. However, there were main effects for RD on each of the three conditions: mean T-score for those with RD was lower than for those without on word naming \( (F(1, 68)=29.59, p<.001, \eta^2=.303) \) (RD \( M=39.76 \), no RD \( M=48.60 \)), color naming \( (F(1, 68)=8.77, p=.004, \eta^2=.116) \) (RD \( M=42.42 \), no RD \( M=48.01 \)), and color word naming \( (F(1, 68)=8.52, p=.005, \eta^2=.112) \) (RD \( M=48.64 \), no RD \( M=56.96 \)). There was no main effect for ADHD on word naming, however, those with ADHD were significantly lower for both color naming \( (F(1, 68)=8.91, p=.004, \eta^2=.114) \) (ADHD \( M=42.40 \), no ADHD \( M=48.03 \)) and color word naming \( (F(1, 68)=9.41, p=.003, \eta^2=.121) \) (ADHD \( M=48.46 \), no ADHD \( M=57.14 \)). Refer to Table 5 for individual cell means and standard deviations. The findings for the demographic variables and cognitive variables were substantially commensurate to those found for the full sample in the original study (Rucklidge & Tannock, 2000).
Level of Performance in CRT

Level of performance in CRT (i.e. speed) was not the primary outcome of interest. However, since we wished to control for any effects of group and trial on the raw latency scores before inconsistency was examined, analyses were conducted in order to confirm the presence of any such effects. Moreover, such an analysis provides a descriptive context within which group differences in inconsistency can be interpreted. The analysis involved a 2(RD) x 2(ADHD) x 32(trial) mixed model ANOVA on the raw latency scores.

There were statistically significant ADHD by Trial ($F(31, 2108)=1.71, p=0.009, \eta^2=.024$) and ADHD by RD ($F(1,68)=6.18, p=.015, \eta^2=.083$) interactions. The control group did not differ from the ADHD-only group, but the ADHD+RD group was slower than the RD only group. That is, the relationship between ADHD and CRT depended on RD such that there was no difference between those with ($M=361.40, SD=44.55$) and without ($M=368.86, SD=71.08$) ADHD in participants who did not have RD ($t(68)=.44, p=.66$), however, in those who did have RD, those diagnosed with ADHD ($M=414.75, SD=67.86$) were reliably slower ($t(68)=2.63, p=.011$) than those without ADHD ($M=340.45, SD=33.53$).

The ADHD by trial interaction was not followed up by individual t-tests across the 32 levels of trial. However, visual examination of the means plot was suggestive of an effect of practice (i.e. rapid increase in speed) over the first three trials, where those with ADHD demonstrated slower reaction times over a period at the beginning of the block (trials 1-8) and a period at the end of the block (trials 25-29).
Purification of CRT and calculation of ISD

As previously indicated, the purpose of the study was to investigate group differences in inconsistency (i.e. moment-to-moment within-person variability) in CRT. Our definition of inconsistency involves the examination of variation in performance that is independent of systematic effects across time such as practice and fatigue. As such, we remove these effects, as well as others (i.e. group differences in level of performance) that might confound an examination of inconsistency. These effects were removed statistically from the data before calculating intra-individual standard deviations (ISD), our measure of inconsistency.

The purification of the raw CRT data was accomplished by partialling out the observed group, and trial effects (and all interactions) from the 32 trials of raw CRT data from each of the 72 participants. This analysis produced residual latency scores that were uncontaminated by differences between groups and differences across trials in mean level of performance. In order to permit comparison between the results of the current study and previous studies employing this method, the residual scores were transformed into T-scores (using the mean and standard deviation for all data points). That is, the mean and standard deviation for all 2304 (32 trials * 72 participants) data points were 50, and 10, respectively.

Group Differences in Inconsistency

The latency scores that have been purified from the effects of trial and group (residual T-scores) are graphically depicted in Figure 3. Separate plots are provided for each group (ADHD, RD, ADHD+RD, Control) that illustrate each participant’s residualized T-score across all 32 trials. It can be observed from these graphs that
although the mean T-score does not differ between groups, nor does mean T-score differ across trials (i.e. there are no trends across time), within-person variation across trials is observable, and this variation appears to differ between groups.

ISD was recorded for each subject by calculating the standard deviation across the 32 trials of residualized T-scores. The primary inconsistency analysis consisted of examining group differences in ISD in a 2 (ADHD) x 2 (RD) factorial ANOVA. It was expected that both ADHD groups would score higher than the control and the RD only groups. Contrary to the expectations, there was a statistically significant ADHD x RD interaction ($F(1,68)=14.66, p<.001$, $\eta^2=.177$). The relationship between ADHD and ISD depended on RD such that there was no difference between the controls ($M=7.40$, $SD=2.80$) and the ADHD only group (i.e. when RD were not present) ($M=7.28$, $SD=2.13$) ($t(68)=0.16, p=.874$). However, for those with RD, ADHD adolescents ($M=10.96$, $SD=3.61$) exhibited statistically significantly more inconsistency than controls (i.e. RD only group) ($M=5.53$, $SD=0.90$) ($t(68)=4.36, p<.001$). See Figure 4 for a graphical depiction of mean ISD across the 4 conditions.

Further analyses were conducted on ISD in order to determine if the group differences observed in ISD were due to some specific factor present only in the slowest portion of the CRT distribution. In order to do so, ISDs were calculated for eight (i.e. 25%) trials taken from either end of the CRT distribution. These fastest and slowest 25% of trials were purified from systematic effects across time as well as from group differences in average speed of response as was done for the original ISD. A series of analyses were conducted in order to examine the hypothesis that group differences in ISD are due solely to effects present in the slow portion (or tail) of the CRT distribution.
First, two 2(ADHD) x 2(RD) factorial ANOVAs were conducted on ISDfast (ISD in the fastest 25% of trials) and ISDslow (ISD in the slowest 25% of trials). It was expected that group differences would be present in ISDslow, but not ISDfast. Second, a 2 (ADHD) x 2(RD) ANCOVA was conducted in order examine group differences in ISDslow controlling for the effects of ISDfast (i.e. inconsistency in the fast portion of the CRT distribution). It was expected that this analysis would not result in a reduction of the group effect observed when ISDslow was examined uncontrolled.

Consistent with the hypothesis, there was a statistically significant ADHD by RD interaction effect on ISDslow ($F(1,68)=15.27, p<.001$, $\eta^2=.183$). The relationship between ADHD and ISDslow was dependent on RD in a similar manner to that for the ISD for all trials: There was no difference between the Control and ADHD only groups ($t(68)=0.35, p=.728$), however, there was a statistically significant difference between the RD only group and the ADHD+RD group ($t(68)=4.34, p<.001$). See Table 5 for individual cell means and standard deviations. In contrast to the expected result for ISDfast, there was a statistically significant main effect for ADHD ($F(1,68)=7.05$, $p=.010$, $\eta^2=.094$). Those with ADHD ($M=5.08$) were statistically significantly higher for ISDfast than those without ADHD ($M=6.70$).

ANCOVA was deemed appropriate in order to examine group differences in ISDslow controlling for ISDfast because preliminary analyses demonstrated that there was no 3-way interaction (i.e. ISDfast by ADHD by RD) or two-way interactions (ISDfast by ADHD, ISDfast by RD) in the prediction of ISDslow (i.e., the ANCOVA assumption of homogeneity of slope was met). Consistent with the predicted result, controlling for ISDfast within the ANCOVA analysis examining group differences in
ISDslow did not result in a reduction of the observed ADHD by RD interaction effect for ISDslow when it was uncontrolled. In fact, the interaction effect in this instance \( (F(1,67)=15.00, p<.001, \eta^2=.183) \) was of identical magnitude.

Since there was a statistically significant main effect of ISDfast on ADHD, ANCOVA (controlling for ISDslow) was used in order to determine if this effect was unique to the fast portion of the RT distribution or if it was general to the fast and slow portions. If the effect were general, it would be expected that including ISDslow as a covariate would reduce or eliminate the observed main effect for ISDfast on ADHD. ANCOVA was again deemed appropriate in this instance as the assumptions of homogeneity of slope was met. Inconsistent with what would be expected if the effect was general across the RT distribution, there was a statistically significant main effect for ISDfast on ADHD after controlling for the effects of ISDslow \( (F(1,67)=6.43, p=.014, \eta^2=.088) \).

**Inconsistency and Cognitive Variables**

A series of hierarchical linear regression analyses were conducted in order to examine the relationship between the various cognitive variables and ISD. All available data from the 72 participants were used in separate analyses where ISD was entered as a predictor of each of the cognitive variables after the effects of ADHD, RD, and the ADHD by RD interaction effect had been accounted for. The grouping variables were controlled in this manner in order to rule out the possibility that the relationship between ISD and the cognitive variables was due to status on the grouping variables.

Hierarchical linear regression revealed several statistically significant relationships between ISD and the various cognitive variables. With respect to the
WISC-III sub-tests and indices examined, there were reliable relationships between ISD and block design \(F(1,67)=9.19, p=.003, \Delta R^2=.112\), FDI \(F(1,64)=4.65, p=.035, \Delta R^2=.046\), and PSI \(F(1,63)=6.81, p=.011, \Delta R^2=.067\). ISD was also a predictor of the color \(F(1,67)=12.85, p=.001, \Delta R^2=.116\) and color-word \(F(1,67)=7.68, p=.007, \Delta R^2=.076\) subtests of the Stroop. Refer to Table 6 for a summary of the hierarchical linear regression analyses.

**Discussion**

The primary purpose of the current study was to conduct an in-depth, well-controlled, examination of inconsistency in RT in a sample from the adolescent ADHD population. The results of the study demonstrated that there is indeed a relationship between ADHD and inconsistency in adolescents. However, inconsistent with our hypothesis, we found this relationship to be dependent, in part, on RD. That is, ADHD adolescents with RD were more inconsistent than those with RD only, but there was no difference between those with ADHD only and normal controls. The findings do not suggest that increased inconsistency in ADHD is an artefact of difficulties (i.e. RD) that are often co-morbid with the disorder, but rather, that heightened inconsistency, particularly in the slow portion of the RT distribution, may be peculiar to that portion of the ADHD sample who also have RD.

Analyses were also undertaken to examine this inconsistency more closely with respect to the RT distribution. Consistent with others (i.e. Leeth-Steensyen et al., 2000) who have examined the distributional properties of performance on RT tasks in ADHD, we found evidence for inconsistency in the slow portion of the RT distribution that was unrelated to performance in the fast portion of the RT distribution. This suggests that
ADHD responding in RT tasks is not characterised by inconsistency that is general across the RT distribution. That is, there appears to be some variability-producing mechanism that affects the slow portion of the RT distribution such as attentional lapses. Similar to inconsistency across all trials, we found this effect in adolescents to be dependent on RD such that the combined group appeared to be the most impaired with respect to inconsistency.

Although it was contrary to our hypothesis, it is perhaps not completely unsurprising that the relationship between ADHD and inconsistency (overall and in the slow portion of the RT distribution) relied on RD. Similar to our finding, several studies examining the co-morbidity between ADHD and RD have found the combined group to be the most impaired on cognitive tasks (Willcutt et al., 2001). In addition, as previously mentioned, there is some evidence of a genetic link between ADHD and RD. It could be that the presence of RD in an individual with ADHD represents a risk factor for increased compromise in cognitive performance.

Also inconsistent with expectations, we found evidence for inconsistency in the fast end of the RT distribution. This effect was not taken as evidence of a general variability-producing mechanism for three reasons. First, the observed effect in the slow end of the distribution was not reduced when inconsistency from the fast end of the distribution was controlled. Second, the effect in the fast end of the distribution was different in that it was unrelated to RD. That is, ADHD adolescents demonstrated higher levels of inconsistency in the fast end of the RT distribution regardless of their classification with respect to RD. Third, the main effect of ADHD held up within an ANCOVA analysis controlling for inconsistency in the slow portion of the RT
distribution. Thus, it appears that there is some mechanism that produces inconsistent responding in the fast end of the RT distribution in ADHD that is independent of that in the slow end of the distribution.

To our knowledge, this is the first paper that has demonstrated that ADHD responding may be characterised, in part, by inconsistent responding in the fast portion of the RT distribution. This could be because previous examinations of the distributional properties of ADHD performance have typically relied on fitting the data to the ex-gaussian distribution where the aim is to account for inconsistency in the slow portion of the RT distribution only. These results highlight the possibility that inconsistency of response may not be a unidimensional construct. Inconsistent responding may be the result of more than a single causal mechanism.

The methodological approach taken in the current study was strong for a number of reasons. To our knowledge, this is the first study examining inconsistency in ADHD to control for systematic across-time effects such as practice and fatigue, as well as group differences in mean level of performance. The group differences observed in inconsistency have been demonstrated to be independent of group differences in mean level of performance and of the effects of practice and fatigue. These controls are not trivial, particularly in ADHD populations where deterioration in performance across time differentiates these individuals from normal controls (Douglas, 1999). It is essential that future examinations of inconsistency in ADHD populations incorporate some kind of control for these factors.

It is also noteworthy that group differences were observed between ADHD and controls in a very basic and relatively short RT task. A two-choice RT task such as the
one used in the current study requires little load on working memory. Thus, it is unlikely that the inconsistency of response observed in the current study was an artefact of poor executive control of behaviour. This issue is also particularly relevant since some view response inconsistency to be related to deficient executive functioning (West, Murphy, Armilio, Craik, & Stuss, 2002), and executive functions play a key role in theoretical models of ADHD (e.g. Barkley, 1997). Moreover, it should also be recognised that reliable differences in inconsistency were observed in the first 32 trials of the RT task. That group differences in inconsistency are apparent almost immediately (i.e. within minutes of task onset) and do not require lengthy periods of time on-task suggests that the effect is likely to permeate studies utilising different task manipulations.

Yet another methodological strength of the current study is the inclusion of a relatively equal number of male and female participants in all groups. While some might suggest that this reduces the generalisability of the findings to the ADHD population since boys outnumber girls in the population by a wide margin, much of the research literature to date has been conducted on boys to the exclusion of girls. As such, this study is able to extend the finding of inconsistent responding in ADHD to those in the population who are female.

It was also demonstrated that inconsistency is related to a number of cognitive variables. As might be expected, inconsistency was not related to crystallized ability. It was, however, related to tasks that involved speeded information processing. Virtually all of the cognitive variables that were related to RT inconsistency in this study involved some element of timing. That is, these tasks required quick and efficient responses for successful performance.
This finding may lend support to the hypothesis of Castellanos and Tannock, (2002) who suggest that inconsistency may be a marker of impaired temporal processing. Future research should examine this hypothesis further. The model put forth by Castellanos and Tannock (2002) postulates links between performance inconsistency and dopaminergic and cerebellar dysfunction. Li & Lindenberger (1999) suggest a similar link indicating that age-related changes in inconsistency may be related to age-related change in catecholamine function. These papers provide examples of the kind of conceptualisation that will move research in inconsistency beyond the descriptive stage and into a more complete understanding of its neurobiological causative mechanisms.

It should be noted that the effects observed in the current study were not overwhelmingly large. The effect size of the group differences observed were in the range of $\eta^2 = .09$ to .18. The moderate effect sizes observed may be the result of the adolescent developmental stage where performance inconsistency is approaching its optimal level and there is not as much between-person variation in inconsistency (i.e. diversity) as in other developmental stages. However, it is not the perspective of the authors that inconsistency is the solitary unifying construct that purports to explain the complex behavioural phenomenon known as ADHD. Rather, we argue that an important piece of a larger puzzle may be better understood by allowing for a view that permits the individual to be observed from the perspective of change rather than assuming stability.

Future research into the cognitive deficits of ADHD should take group differences in inconsistency into account. As Douglas (1999) indicated, inconsistency likely represents a pervasive impairment. Task manipulations aimed at producing group differences in level of performance should be interpreted within this context. Not only is
increased inconsistency in ADHD characteristic of the slow portion of the RT
distribution, but it can also be found in the fast portion of the RT distribution. Future
research should investigate this latter issue in the childhood ADHD population. This
study also highlights the importance of the consideration of RD in evaluations of the
cognitive functioning in ADHD.

Observable group differences in inconsistency were confirmed in this adolescent
sample. ADHD adolescents appear to exhibit higher levels of response inconsistency
than controls. This relationship is dependent, in part, on RD such that increased
inconsistency in the slow aspect of the RT distribution appears to be limited to those
ADHD adolescents who also have co-morbid RD. In contrast, increased inconsistency in
the fast portion of the RT distribution appears to be generally higher in ADHD regardless
of level of RD. All of these relationships were found independent of systematic across-
time effects such as practice and fatigue as well as group differences in level of
performance.

Study 3: Inconsistency in children with ADHD

Introduction

The final study extends the investigation to younger children with ADHD.
Specifically, group differences in RT inconsistency between children with ADHD and
controls were examined. A sample of ADHD children (aged 6 to 12 years old) with and
without RD, obtained from outpatient referrals to The Hospital for Sick Children in
Toronto were compared with a group of control children of similar age. This study was
conducted in order to determine if children with ADHD exhibit higher levels of RT
inconsistency than normal controls after controlling for the effects of practice and fatigue, and after controlling for group differences in speed of response.

As previously indicated, a number of studies have demonstrated that children with ADHD appear to exhibit higher levels of RT inconsistency than control children (e.g. Leth-Steensen, Elbaz, & Douglas, 2000; Steger et al., 2001; Hynd et al., 89; Scheres, Oosterlaan, & Sergeant, 2001; Kuntsi, Oosterlaan, & Stevenson, 2001), and there is some limited evidence in adolescents that inconsistency of response is characteristic of ADHD, but not of RD (Rucklidge & Tannock, 2000). Correspondingly, it was expected that children with ADHD would exhibit higher levels of RT inconsistency than controls. Alternatively, if children with ADHD performed similarly to the adolescents examined in study 2, it would be expected that group differences between ADHD and control would be limited to the ADHD group with RD.

Previous research examining the ex-Gaussian distribution has suggested that group differences between those with ADHD and controls is likely due to heightened inconsistency in the tail of the slow portion of the RT distribution (Leth-Steensen et al., 2000; Hervey et al., 2004). The current study approached this issue by examining the fastest and slowest 25% of responses in a manner consistent with that suggested by Salthouse (1993). If heightened inconsistency in children with ADHD were due to some mechanism selectively affecting the slow portion of the RT distribution, we would expect to find group differences in RT inconsistency limited to the slow as opposed to fast portion of the RT distribution, and the group effect in the slow portion of the RT distribution to be unattenuated after partialling any effect of the fast portion of the RT distribution. Alternatively, if children with ADHD perform similarly to the adolescents
in study 2, it would be expected that there would be evidence of effects specific to both the fast and slow portions of the RT distribution.

The final study was also undertaken to examine the relationship between behavioural symptoms of ADHD (i.e. inattention, hyperactivity, and impulsivity) and inconsistency via an individual differences approach. While group differences have been observed between those with and without a diagnosis of ADHD, little research has examined the relationship between inconsistency and symptom severity. In the sole study to examine this issue to date, Hervey et al. (2004) found the number of ADHD symptoms to be related to overall inconsistency as well as tau (i.e. a measure of inconsistent responding resulting from relatively higher number of abnormally slow responses), however, these analyses were not broken down by symptom type. It is expected that a more thorough examination of ADHD symptomatology and inconsistency might provide further insight into the relationship between inconsistency and ADHD. Specifically, these analyses will examine the relationship between measures of overall inconsistency, and inconsistency in the fast and slow portions of the RT distribution, with measures of inattention, hyperactivity/impulsivity.

Method

Participants

The sample consisted of 158 children, aged 6 to 12 years old, who underwent testing at the Hospital for Sick Children in Toronto, Canada. Children in the ADHD groups (ADHD and ADHD +RD) were referred to the hospital from community physicians because of difficulties with behaviour, attention, and learning. Those in the control group were recruited via ads placed in the hospital newsletter. Exclusionary
criteria for the study included any evidence of neurological dysfunction, uncorrected sensory impairment, history of psychosis, or a full-scale intelligence quotient (WISC-III FSIQ) (Wechsler, 1991) of less than 70. Of those included in the study, four were eliminated because of invalid administrations of the primary outcome measure leaving data from a total of 154 participants for analysis. See Table 7 for further descriptive information broken down across the three groups.

ADHD Groups.

Participants included in the ADHD groups had a confirmed DSM-IV-TR (2000) diagnosis of ADHD. Diagnosis was based on standardized semi-structured clinical interviews conducted with the children’s parents (Parent Interview for Child Symptoms-IV [PICS-IV]) (Ickowicz et al., 2002) and school teachers (Teacher Telephone Interview-IV [TTI-IV]) (Tannock, Hum, Masellis, Humphries, & Schachar, 2002). Information from these interviews was submitted to a “6/4” algorithm (Refer to Bedard et al., 2003 for a description) which is a diagnostic algorithm based on the DSM-IV-TR (2000) criteria for ADHD. The algorithm is designed to ensure pervasiveness of symptom presentation across both home and classroom settings. Information from behavioural rating scales was also available in order to aid diagnosis. In each case, a team consisting of several clinicians convened in order to confirm the diagnostic decision.

The children in the ADHD group were sub-divided into two groups: ADHD only, and ADHD+RD. This classification was based on their norm-referenced scores on three measures of reading: Word Attack and Word Identification sub-scales of the Woodcock Reading Mastery Test- Revised (WRMT-R) (Woodcock, 1987) and the Reading sub-scale of the Wide Range Achievement Test- Third Edition (WRAT-3) (Wilkinson, 1993).
Consistent with others (e.g. Bedard et al., 2003), we used a standard such that those with at least one score more than 1.5 standard deviations below the mean, or at least two scores more than 1 standard deviation below the mean were classified as RD. Identification of those with RD in this manner is consistent with a rejection of the IQ discrepancy definition (Meyer, 2000; Fletcher et al., 2002). Please note that the terminology "reading difficulties" (RD) is adopted recognising that learning disorders are best defined by criteria (e.g. response to intervention) (Meyer, 200; Fletcher et al., 2002) that are outside the parameters of this study.

*Control Group.*

Control participants originated from the same geographical region as those in the ADHD groups. Participants were excluded from the group if they were found to meet the criteria for the ADHD group or if they met the classification requirement for RD as outlined above.

*Measures*

*Choice Reaction Time (CRT).*

CRT data from which inconsistency measures were derived were obtained from the stop signal procedure, a computer-generated measure of inhibitory control (Logan, 1994; Logan & Cowan, 1984). The procedure involves two concurrent tasks: The "go" task, and the "stop" task. The version used in the current study is described by Williams, Ponnesse, Schachar, Logan, & Tannock (1999). The "go" task was a visual, two-choice reaction-time task. Participants were presented with a fixation point in the middle of the screen for 500 ms, followed by either an "X" or an "O" of 1000 ms duration. Participants
were required to respond to the stimulus by pressing the corresponding button on a response box as quickly as possible. The "stop task" involved the demand that participants inhibit their response to the "go task" when a 1000 Hz tone was presented (the stop signal). Stop signals occurred on 20% of the trials.

The task consisted of 256 trials divided into eight 32-trial blocks. However, preceding the test blocks, two 32-trial practice blocks were given. The first practice block was administered before the demand to stop was introduced. That is, participants were instructed to respond to the "go" stimuli, and to ignore the intermittent tones. The data analysed in the current study were derived from this first 32-trial practice block which has not been examined in any previous study.

*Measures of Symptom Severity.*

The PICS-IV and TTI-IV are semi-structured clinical interviews that are used to form the basis of ADHD diagnosis as well as to screen for other childhood disorders. They have been developed and used at the Hospital for Sick Children in Toronto and current information on their psychometric properties may be found elsewhere (See Bedard et al., 2003). For each interview, clinicians rate the severity of each symptom on a four-point scale and the symptom is regarded as "impairing" if it exceeds the threshold of two. In the current study two measures of symptom severity are provided for each interview according to the two domains of ADHD symptoms in the *DSM-IV-TR* (2002): Inattentive, and hyperactive/impulsive. They are defined as the sum of the number of symptoms that reached the impairment criteria. In addition, two measures are provided based on information combined across the two interviews. That is, a symptom was counted within a domain if it reached criteria on either the PICS-IV or the TTI-IV.
Procedure

All data were collected by a team of researchers and clinicians at the Hospital for Sick Children in Toronto, Canada between July 1996 and June 1998. Parents of the children provided informed consent and each child provided verbal assent to the procedures of the study. The children were required to attend the hospital for one day during which CRT was collected and the psychometric assessment was conducted. The PICS-IV and TTI-IV interviews were conducted by trained clinicians on staff at the hospital. All children were free of stimulant medication on their testing day.

Data Preparation

CRT data (resolved to the millisecond) were obtained from the stop signal procedure for each of the 154 participants. Of the total possible observations (154*32=4928 observations), 175 (3.6%) were missing because either no response was made, the participant responded before the stimulus was presented, or more than one response was made during the given trial. Furthermore, in an effort to reduce measurement error, outlying data points were identified and eliminated according to the following procedure. First, a lower bound level for legitimate responses was set at 150 ms, and scores falling below this value were eliminated. It was expected that such extremely fast data points might reflect errors such as accidental key presses. Second, data points were eliminated if they fell beyond the upper bound limit which was defined as three standard deviations above the mean of each group. Values above this cut point were thought to reflect errors such as distraction of the participant. Across the 4753 observations, 35 (0.7%), and 83 (1.7%) of the data points were eliminated as lower-
bound, and upper-bound outliers, respectively. The proportion of observations that were identified as outliers was roughly equivalent across the three groups.

Following the removal of outliers, values were imputed for all missing data points via a regression procedure where estimates were based on the relationship of responses across trials. Both procedures, eliminating outlying data points and imputing estimates for the missing values, were likely to decrease intraindividual variability, and thus provide a conservative examination of inconsistency.

Results

Group differences in sample characteristics

A series of tests were conducted in order to provide further description of the sample with respect to demographic characteristics. Mean age was examined in a one-way ANOVA across the three groups. Age varied among the three groups ($F(2, 151)=5.71, p=0.004, \eta^2=0.07$). Follow-up t-tests demonstrated that the ADHD+RD group was statistically significantly younger than both the control ($t(151)=3.17, p=0.002$) and ADHD only ($t(151)=2.65, p=0.009$) groups. Refer to Table 7 for group means and standard deviations. Chi square test of association was used in order to examine differences in gender across the three groups. The distribution of gender differed across the three groups ($\chi^2(2)=14.11, p=0.001$); The ratio of male to female participants was 1:1, 5.6:1, and 3:1 in the control, ADHD only, and ADHD+RD groups, respectively.

Group differences were also examined via a one-way ANOVA for WISC-III FSIQ. There was statistically significant variation among the groups with respect to FSIQ ($F(2, 149)=25.79, p<0.001, \eta^2=0.26$). Follow-up t-tests revealed that FSIQ was
higher in the controls as compared to both the ADHD ($t(149)=3.81, p<.001$) and ADHD+RD ($t(149)=7.06, p<.001$) groups, and higher in the ADHD group as compared to the ADHD+RD group ($t(149)=4.79, p<.001$). Individual cell means and standard deviations may be found in Table 7.

**Level of Performance in CRT**

Level of performance in CRT (i.e. speed) was not the primary outcome of interest. However, since we wished to control for any effects of group and trial on the raw latency scores before inconsistency was examined, analyses were conducted in order to confirm the presence of any such effects. Moreover, such an analysis provides a descriptive context within which group differences in inconsistency can be interpreted. The analysis involved a 3(Groups) x 32(Trial) mixed model ANOVA on the raw latency scores.

The group by trial interaction effect was not statistically significant. However, there was a statistically significant main effect of group ($F(2, 151)=6.44, p=.002$) such that the ADHD+RD group was slower than both the ADHD only ($t(151)=2.50, p=.014$) and control ($t(151)=3.51, p=.001$) groups. Group means for individual’s mean (across-trial) choice reaction time (MCRT) can be found in Table 7. In addition, there was a statistically significant main effect for trial ($F(31, 4681)=12.24, p<.001$). Follow-up t-tests were not examined, however, visual inspection of the means plot suggested mean performance on the first trial was slow relative to the rest of the trials.

**Purification of CRT and calculation of ISD**

As previously indicated, the purpose of the study was to investigate group differences in inconsistency (i.e. moment-to-moment within-person variability) in CRT.
Our definition of inconsistency involves the examination of variation in performance that is independent of systematic effects across-time such as practice and fatigue. As such, we remove these effects, as well as others (i.e. group differences in level of performance) that might confound an examination of inconsistency. These effects were removed statistically from the data before calculating intra-individual standard deviations (ISD), our measure of inconsistency.

The purification of the raw CRT data was accomplished by partialling out the observed group, and trial effects (and all interactions) from the 32 trials of raw CRT data from each of the 154 participants. This analysis produced residual latency scores that were uncontaminated by differences between groups and differences across trials in mean level of performance. In order to permit comparison between the results of the current study and previous studies employing this method, the residual scores were transformed into T-scores (using the mean and standard deviation for all data points). That is, the mean and standard deviation for all 4928 (32 trials * 154 participants) data points were 50, and 10, respectively.

*Group Differences in ISD*

Line graphs overlaying each individual’s residualized latency scores (T-Scores) across the 32 trials provides a striking demonstration of inconsistency. Figure 5 depicts such plots separately for each group (ADHD, ADHD+RD, & Control). Note that as a result of having been purified from the effects of trial and group it can be observed from examination of these graphs that the mean latency score does not differ between groups, nor does mean latency score differ across trials (i.e. there are no trends across time).
However, within-person variation across trials (i.e. inconsistency) is observable, and this variation appears to differ between groups.

ISD was recorded for each subject by calculating the standard deviation across the 32 trials of residualized T-scores. The primary inconsistency analysis consisted of examining group differences in ISD in a one-way ANOVA. Because the groups differed with respect to age, and because of the strong relationship between age and inconsistency (Refer to study 1), age was included as a covariate within this analysis. Age was deemed an appropriate covariate as there was no group by age interaction in the prediction of ISD. Consistent with predictions, there was statistically significant variation in ISD between groups ($F(2,150)=7.70, p=.001$, $\eta^2=.09$). Follow-up t-tests were performed using Tukey’s LSD procedure. Consistent with expectations, ISD was higher in the ADHD+RD ($M=9.00$, $SE=0.36$) group as compared with the controls ($M=6.81$, $SE=0.43$) ($t(150)=3.84, p<.001$). Moreover, consistent with expectations based on previous research in children with ADHD, ISD was higher in the ADHD only ($M=8.36$, $SE=0.24$) group as compared to controls ($M=6.81$, $SE=0.43$) ($t(150)=3.16, p=.002$). However, inconsistent with that found in adolescents, there was no statistically significant difference between the ADHD only ($M=8.36$, $SE=0.24$) and ADHD+RD ($M=9.00$, $SE=0.36$) groups ($t(150)=1.46, p=.148$). Figure 6 provides a graphical depiction of the adjusted group means.

Further analyses were conducted on ISD in order to determine if the group differences observed in ISD were due to some specific factor present only in the slowest portion of the CRT distribution. In order to do so, ISDs were calculated for eight (i.e.

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$^4$ Separate analyses were undertaken using gender and full-scale IQ as covariates. Within these analyses, the pattern of findings remained consistent with that reported above.
25%) trials taken from either end of the CRT distribution. These fastest and slowest 25% of trials were purified from systematic effects across time as well as from group differences in average speed of response as was done for the original ISD. A series of analyses were conducted in order to examine the hypothesis that group differences in ISD are due solely to effects present in the slow portion (or tail) of the CRT distribution. First, two one-way ANCOVAs (controlling for age) were conducted on ISDfast (ISD in the fastest 25% of trials) and ISDslow (ISD in the slowest 25% of trials). If the overall ISD effect were due to a mechanism that selectively affected the slow portion of the CRT distribution, we would expect that group differences would be present in ISDslow, but not ISDfast and that the group differences in ISD slow would be present despite controlling for the effects of ISDfast in a subsequent analysis. Accordingly, a one way ANCOVA was subsequently conducted in order examine group differences in ISDslow controlling for the effects of ISDfast (i.e. inconsistency in the fast portion of the CRT distribution) and age.

Inconsistent with the predicted result, statistically significant group differences were not observed in either ISDslow ($F(2,150)=2.82$, $p=.063$, $\eta^2=.04$) or ISDfast ($F(2,150)=0.45$, $p=.642$, $\eta^2=.01$) in the initial ANCOVAs. The subsequent ANCOVA did not reduce the effect size for ISDslow, but the group differences were nonetheless not statistically significant ($F(2,149)=2.79$, $p=.065$, $\eta^2=.04$).

Inconsistency and Symptom Severity

A series of analyses were undertaken in order to examine the relationship between inconsistency and severity of symptoms of ADHD. First, zero-order correlations were calculated between measures of inconsistency, symptom severity measures, and age
(Refer to Table 8). Of primary interest was the relationship between ISD and the measures of symptom severity, which, with the exception of inattentive symptoms on the PICS-IV ($r=.148, p>.05$), were statistically significant. The magnitude of the correlations ranged from .181 to .335 and were in the expected direction (i.e. higher inconsistency associated with higher symptom severity). Positive correlations were also found between ISDslow and the symptom severity measures, but they were lower in magnitude and limited to hyperactive/impulsive symptoms on the PICS-IV ($r=.178, p<.05$), and combined ($r=.185, p<.05$), and inattentive symptoms on the TTI-IV ($r=.182, p<.05$). In contrast, no statistically significant correlations were found between ISDfast and the measures of symptom severity. However, there was a strong negative relationship between ISDfast and age ($r=-.409, p<.01$), (i.e. younger age associated with higher inconsistency). Age was related to ISD ($r=-.323, p<.01$), but not ISDslow ($r=-.038, p>.05$).

Hierarchical linear regression was used in a subsequent analysis in order to determine if symptom severity would predict ISD after accounting for the variability associated with age. Therefore, age was entered on an initial step, followed by combined (from both interviews) symptoms of inattention and hyperactivity/impulsivity which were entered together. The final model accounted for a statistically significant proportion of the variance in ISD ($F(3, 150)=11.07, p<.001, R^2=.181$). Moreover, the addition of symptom severity added a statistically significant proportion of variability accounted for in ISD over and above that accounted for by age alone ($AF(2, 150)=7.05, p=.001, \Delta R^2=.077$).
Hierarchical linear regression was also examined to determine if the relationship between ISDslow and symptoms of hyperactivity/impulsivity combined across the two interviews would remain statistically significant while controlling for age and ISDfast. The change in variance explained by hyperactive/impulsive symptoms over and above age and ISDfast was statistically significant ($\Delta F(1,150)=5.06$, $p=.026$, $\Delta R^2=.033$).

Discussion

The purpose of this study was to examine the relationship between inconsistency and ADHD in children while controlling for the effects of mean speed of response and time on task. The first set of analyses examined group differences in inconsistency. As expected, there were significant differences among the groups with respect to moment-to-moment fluctuations in performance on the CRT task. The pattern of the findings was similar to other studies examining this issue in children (e.g. Leth-Steensø, Elbaz, & Douglas, 2000; Steger et al., 2001; Hynd et al., 89; Scheres, Oosterlaan, & Sergeant, 2001; Kuntsi, Oosterlaan, & Stevenson, 2001). That is, children with ADHD exhibited higher levels of inconsistency than normal controls. Inconsistent with what might be expected based on findings in adolescents with ADHD in study 2, the ADHD+RD group was not more inconsistent than the ADHD only group.

A series of analyses were also undertaken to determine how inconsistency is manifested throughout the CRT distribution. Some suggest that inconsistent responding in ADHD can be accounted for by inconsistency in the slow portion of the RT distribution only which might reflect some specific mechanism such as attentional blocks. However, in the adolescent sample examined in study 2, evidence was found for group differences in both the fast and slow portions of the CRT distribution. The current study
was unable to confirm the presence of reliable group differences in either the fast or slow portion of the CRT distribution in this sample of children. However, as the effect size was higher when group differences were examined in the slow ($\eta^2 = .04$) portion of the distribution rather than the fast ($\eta^2 = .01$) part of the distribution, the results are most consistent with the attentional lapse hypothesis. Unfortunately, there was not enough power (.55) to detect an effect as small as that observed in even the slow portion of the distribution. It is noteworthy, however, that in a subsequent analysis whereby the relationship between inconsistency in the slow portion of the distribution and ADHD was examined while controlling for inconsistency in the fast portion of the distribution, the effect size was not attenuated.

The relationship between inconsistency and symptoms of ADHD were also examined within an individual differences approach. Symptoms of ADHD across both domains (inattentive & hyperactive/impulsive) were available from ratings of interviews with teachers, parents, and combined across interviews with teachers and parents. Inconsistency was associated with virtually every measure (barring inattentive symptoms on parent interviews) such that higher inconsistency was related to the presence of a higher number of ADHD symptoms. Further evidence of this relationship was provided in a subsequent hierarchical regression analysis whereby symptoms from both domains combined from both interviews added significant variance in the prediction of inconsistency after the variance associated with age had been taken into account. This provides further evidence of the relationship between symptoms of ADHD and inconsistency independent of the effect of age.
Since the relationships between inconsistency in the tails of the distribution and symptoms of ADHD were also examined, the individual differences analyses also speak to the distributional issues outlined above. There were statistically significant relationships between inconsistency in the slow portion of the distribution and various measures of ADHD symptoms: hyperactive/impulsive symptoms on both parent interviews and combined across interviews, and inattentive symptoms on the teacher interview. In a follow-up regression analysis, the relationship between inconsistency in the slow portion of the distribution and hyperactive/impulsive symptoms combined across both interviews was not attenuated when inconsistency in the fast portion of the distribution was controlled. Although the effect size was modest ($AR^2=.033$), this finding lends support to the hypothesis that there may be some mechanism that is selectively affecting the slow portion of the CRT distribution.

In contrast, there were no statistically significant relationships between inconsistency in the fast portion of the distribution and any of the measures of symptom severity. In fact, in this sample consisting predominantly of children with ADHD, inconsistency in the fast portion of the distribution was related to age. Thus, although inconsistency in the fast portion of the distribution was not related to ADHD symptoms, there was some evidence that it was associated with different variables in this sample. It is also interesting to note that inconsistency in the slow portion of the distribution was not related to age. That is, there appears to be something unique about inconsistency happening in each end of the distribution.

Some view response inconsistency to be related to deficient executive functioning, particularly when group differences are observed in the slow portion of the
RT distribution (West, Murphy, Armilio, Craik, & Stuss, 2002). Since executive functions play a key role in theoretical models of ADHD (e.g. Barkley, 1997), this issue is particularly relevant when examining the ADHD population. On one hand, there was evidence of support for the attentional lapse hypothesis when differences in inconsistency were examined via the individual differences approach. However, the effect sizes were not large. Moreover, group differences were observed between ADHD and controls in a very basic and relatively short RT task. A two-choice RT task such as the one used in the current study requires little load on working memory, and reliable differences in inconsistency were observed in the first 32 trials of the RT task suggesting that task persistence was likely not a contributing factor. Thus, strong conclusions regarding deficient executive function as a source of response inconsistency cannot be made. In addition, there may be multiple sources of variation in response inconsistency.

There was no evidence to suggest that inconsistency was related to either type of ADHD symptoms to the exclusion of the others. That is, there were relationships between the measures of inconsistency across both inattentive and hyperactive/impulsive symptoms. However, based on the pattern of correlations, there is some evidence that inconsistency was more strongly related to hyperactive/impulsive symptoms on parent interviews, and inattentive symptoms on teacher interviews. It could be that impairment related to inconsistency manifests differentially across the two settings, however, any such conclusion could only be considered tentative based solely on these results.

The current study did not provide evidence that inconsistency was related to ADHD differentially, depending on the presence of RD as was suggested by the results of study 2. Several potential explanations could provide insight as to the divergence
between the results of the two studies. First, for several reasons, some researchers (e.g. Fletcher et al., 2002) suggest that cut-points are unreliable when used to classify individuals into learning disability categories. Thus, the differences between the studies could be due to error in classification. Alternatively, one could consider the developmental trajectories of those identified as RD. It would not be surprising that of this group of children, several might be exposed to and respond to treatment offered for their difficulties. Thus, those identified with RD in adolescence might represent only a sub-set of those identified as children, namely, those resistant to remediation. This explanation corresponds with the pattern of results across the two studies whereby the ADHD+RD group was found to be more severe in the adolescent sample, but not the child. Furthermore, a specific limitation of the current study was that those in the RD group were younger even when compared to the ADHD only and controls within this study (i.e. There were no ADHD+RD children in the sample 11 years or older). The interaction between the developmental trajectory of the presentation of RD and that of the symptoms of ADHD further complicate the matter. Many of such issues could be ameliorated by the examination of longitudinal data.

The methodological approach taken in the current study was strong for a number of reasons. To our knowledge, this is the first study to provide evidence of heightened inconsistency in children with ADHD while controlling for systematic across-time effects such as practice and fatigue, as well as group differences in mean level of performance. The group differences observed in inconsistency have been demonstrated to be independent of group differences in mean level of performance and of the effects of practice and fatigue. These controls are not trivial, particularly in ADHD children where
deterioration in performance across time differentiates these individuals from normal controls (Douglas, 1999). It is essential that future examinations of inconsistency in ADHD populations incorporate some kind of control for these factors.

The current study confirms the relationship between inconsistency and childhood ADHD. Children with ADHD exhibit higher levels of moment-to-moment fluctuations in performance as compared to controls. Limited evidence was also provided to suggest that symptoms of ADHD are uniquely related to inconsistency in the slow portion of the RT distribution (as compared to the fast portion of the RT distribution) indicating that inconsistency may be influenced by some mechanism specific to that portion of the distribution such as attentional lapses. The study certainly highlights the importance of the consideration of moment-to-moment fluctuations in performance in ADHD children for future research as well as in the development of theoretical models of ADHD.

General Discussion

Through these three investigations, we intended to broaden the scope of our understanding of RT performance in a number of populations. While most accounts of such performance are limited to examinations of mean level of performance (i.e. speed), following Nesselroade’s (1991a) conception of intraindividual variability, we sought to describe the ebb and flow of performance that may be observed over very brief time intervals. That is, we examined inconsistency of response, or the moment-to-moment fluctuations that can be observed in RT performance. We did so by applying a method and approach that has been developed by researchers of the MIND project (Hultsch, Strauss, Hunter and others) that is designed to provide a measure of inconsistency while controlling for systematic across-time effects (e.g. practice and fatigue) and group
differences in mean level of response. While this aspect of performance (i.e. inconsistency) is typically regarded as error variance, this thesis demonstrates that there is systematic variation in the ebb and flow of RT performance. In other words, it was demonstrated that there is meaning in what is typically disregarded as imprecision of measurement.

This thesis was the first work to examine life span developmental differences in inconsistency in the general population while controlling for systematic across-time effects and age group differences in mean level of performance. Variation in inconsistency was observed across ages from children as young as 6 years to those aged 81 years. In general, this performance may be characterised as following a U-shaped developmental curve with optimal performance observed around 20 years of age. Differences in inconsistency between those of differing ages in the healthy population provide a context for an examination of inconsistency in developmental psychopathology. More specifically, group differences were observed in both children and adolescents between those with ADHD and healthy controls. ADHD children were significantly more inconsistent that healthy controls. Moreover, this was also generally the case for adolescents with ADHD, however, within the adolescent sample, the effect depended on RD such that the effect was only present within ADHD participants who also had RD. In addition to group differences, when examined via an individual differences approach, inconsistency was related to the behavioural symptoms (inattention, hyperactivity/impulsivity) that form the basis of the diagnosis of ADHD: those children with more symptoms of inattention and hyperactivity/impulsivity were more inconsistent than those with fewer symptoms.
In order to provide a more thorough description of inconsistency as well as to test the hypothesis of whether heightened inconsistency may be due to a specific variability-producing mechanism that selectively affects the slow portion of the RT distribution, a set of analyses were conducted within each of the samples in order to follow-up on the overall inconsistency effects observed. As expected, evidence for a specific effect selectively affecting the slow portion of the distribution was found across all three samples. However, in addition, there was also evidence for effects that were general to both the fast and slow portions of the distribution (e.g. in the normal population in those age 6 to 20 years), as well as evidence for an effect selectively affecting the fast portion of the distribution (e.g. in adolescents with and without ADHD). While it would be appealing from an explanatory perspective to employ a unitary account of inconsistency effects, there is evidence that there may be multiple sources of variation in inconsistency. That is, the inconsistency effects observed in this thesis cannot be accounted for solely by a single variability-producing mechanism that is at work in the slow portion of the RT distribution.

It is not surprising that inconsistency is not a unitary phenomenon. After all, inconsistency in and of itself is not a deficit in human performance. Inconsistency is a description or a summary of performance. As a measure of performance, inconsistency may be sensitive to several different types of deficits. Otherwise put, performance may be inconsistent for several different reasons. The analysis of differing aspects of the RT distribution is one method of examining differing sources of moment-to-moment variation. However, one might find that different tasks, or task manipulations within the same task are also sensitive to differing sources of variability in inconsistency. As
research continues to consider moment-to-moment fluctuations in performance, interpretation of such performance must consider that within our measures of inconsistency are potentially multiple sources of variation.

It is somewhat unsatisfying that this thesis remains at the descriptive level with respect to its account of inconsistency in performance. It is, however, beyond the scope of this work to address the "why" questions (e.g. Why are young children and older adults more inconsistent than young adults?). This fact certainly doesn't undermine the importance of the work. It is a necessary first step to provide a description of such effects. Moreover, the explanation of the effects will likely involve a cross-disciplinary approach including the behavioural, neurochemical and neuroanatomical levels of analysis. As previously noted, some of this work has already begun. For example, one may point to Li & Lindenberger's (1999) work in the examination of age-related change in catacholamine function in the normal population, or the theory of Castellanos and Tannock (2002) which postulates links between performance inconsistency and dopaminergic and cerebellar dysfunction in children with ADHD. Currently, the questions of "Why" remain unanswered and definitively beyond the scope of this work.

As previously indicated, the purpose of this dissertation was to identify and examine sources of systematic variation in inconsistency in performance. This task was met largely with success. Evidence was provided of age-related differences across the life span in the general population, and group differences between healthy controls and those with ADHD, and individual differences in inconsistency related to behavioural symptoms of ADHD. Because large systematic sources of variation are observed in inconsistency, the fluctuations in moment-to-moment RT performance cannot be
regarded as measurement error. That is, inconsistency appears to be important aspect of performance. As such, descriptions of RT performance are incomplete without some accounting for inconsistency. Level of performance must be considered either within the context of the ebb and flow of performance, or hand and hand with an account of inconsistency.
References


Tables
Table 1.

Descriptive statistics for demographic and outcome variables across age groups of study 1.

<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>Description</th>
<th>N</th>
<th>% Female</th>
<th>CRT M</th>
<th>CRT SD</th>
<th>ISD M</th>
<th>ISD SD</th>
<th>Fast ISD M</th>
<th>Fast ISD SD</th>
<th>Slow ISD M</th>
<th>Slow ISD SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6-8</td>
<td>Early childhood</td>
<td>29</td>
<td>59</td>
<td>565.77</td>
<td>91.13</td>
<td>14.10</td>
<td>3.78</td>
<td>8.07</td>
<td>3.52</td>
<td>10.04</td>
<td>4.74</td>
</tr>
<tr>
<td>2</td>
<td>9-12</td>
<td>Mid childhood</td>
<td>39</td>
<td>51</td>
<td>408.45</td>
<td>83.76</td>
<td>8.49</td>
<td>2.31</td>
<td>5.94</td>
<td>2.97</td>
<td>6.22</td>
<td>2.89</td>
</tr>
<tr>
<td>3</td>
<td>13-17</td>
<td>Adolescence</td>
<td>50</td>
<td>64</td>
<td>338.67</td>
<td>53.84</td>
<td>6.68</td>
<td>1.78</td>
<td>4.49</td>
<td>1.74</td>
<td>4.92</td>
<td>2.16</td>
</tr>
<tr>
<td>4</td>
<td>18-29</td>
<td>Young adulthood</td>
<td>47</td>
<td>40</td>
<td>320.10</td>
<td>35.58</td>
<td>6.04</td>
<td>0.97</td>
<td>3.95</td>
<td>1.87</td>
<td>4.65</td>
<td>1.77</td>
</tr>
<tr>
<td>5</td>
<td>30-44</td>
<td>Mid adulthood</td>
<td>55</td>
<td>46</td>
<td>345.98</td>
<td>40.90</td>
<td>6.45</td>
<td>1.43</td>
<td>3.95</td>
<td>1.77</td>
<td>5.43</td>
<td>1.99</td>
</tr>
<tr>
<td>6</td>
<td>45-59</td>
<td>Older adulthood</td>
<td>28</td>
<td>46</td>
<td>396.80</td>
<td>64.31</td>
<td>6.98</td>
<td>2.08</td>
<td>4.34</td>
<td>2.40</td>
<td>5.83</td>
<td>2.76</td>
</tr>
<tr>
<td>7</td>
<td>60-81</td>
<td>Elderly</td>
<td>25</td>
<td>40</td>
<td>448.79</td>
<td>71.86</td>
<td>8.58</td>
<td>2.58</td>
<td>4.94</td>
<td>1.87</td>
<td>8.16</td>
<td>4.50</td>
</tr>
<tr>
<td>Total</td>
<td>6-81</td>
<td></td>
<td>273</td>
<td>50</td>
<td>387.08</td>
<td>95.64</td>
<td>7.77</td>
<td>3.15</td>
<td>4.90</td>
<td>2.60</td>
<td>6.10</td>
<td>3.31</td>
</tr>
</tbody>
</table>

Note. ISD = intraindividual standard deviation; Fast ISD = ISD within the fastest 25% of trials; Slow ISD = ISD within the slowest 25% of trials.
Table 2.

*Summary of hierarchical multiple regression analyses of inconsistency and age (N=273)*

*from study 1.*

<table>
<thead>
<tr>
<th>Analysis</th>
<th>y</th>
<th>x Entered</th>
<th>R</th>
<th>$\Delta R^2$</th>
<th>$\Delta F$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ISD</td>
<td>Age</td>
<td>0.21</td>
<td>0.04</td>
<td>$F(1,271)= 12.44, p&lt; .001$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age$^2$</td>
<td>0.58</td>
<td>0.29</td>
<td>$F(1,270)= 117.31, p&lt; .001$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age$^3$</td>
<td>0.68</td>
<td>0.13</td>
<td>$F(1,269)= 63.15, p&lt; .001$</td>
</tr>
<tr>
<td>2</td>
<td>Fast ISD</td>
<td>Age</td>
<td>0.22</td>
<td>0.05</td>
<td>$F(1,271)= 13.90, p&lt; .001$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age$^2$</td>
<td>0.41</td>
<td>0.12</td>
<td>$F(1,270)= 38.57, p&lt; .001$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age$^3$</td>
<td>0.49</td>
<td>0.08</td>
<td>$F(1,269)= 27.34, p&lt; .001$</td>
</tr>
<tr>
<td>3</td>
<td>Slow ISD</td>
<td>Age</td>
<td>0.03</td>
<td>0</td>
<td>$F(1,271)= .19, p = .67$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age$^2$</td>
<td>0.37</td>
<td>0.14</td>
<td>$F(1,270)= 42.22, p&lt; .001$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age$^3$</td>
<td>0.43</td>
<td>0.05</td>
<td>$F(1,269)= 16.40, p&lt; .001$</td>
</tr>
<tr>
<td>4</td>
<td>Slow ISD</td>
<td>Fast ISD</td>
<td>0.2</td>
<td>0.04</td>
<td>$F(1,271)= 11.52, p = .001$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age</td>
<td>0.22</td>
<td>0.01</td>
<td>$F(1,270)= 1.50, p = .22$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age$^2$</td>
<td>0.38</td>
<td>0.1</td>
<td>$F(1,269)= 30.68, p&lt; .001$</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age$^3$</td>
<td>0.43</td>
<td>0.04</td>
<td>$F(1,268)= 13.92, p&lt; .001$</td>
</tr>
</tbody>
</table>

*Note.* Effect size and tests of statistical significance are provided for the step in which the effect in question and all lower-order polynomials were included in the regression equation. ISD = intraindividual standard deviation; Fast ISD = ISD within the fastest 25% of trials; Slow ISD = ISD within the slowest 25% of trials.
Table 3.

*Summary of hierarchical multiple regression analyses of inconsistency and age in the younger half (age <21) of the sample (n=135) from study 1.*

<table>
<thead>
<tr>
<th>Analysis</th>
<th>y</th>
<th>x Entered</th>
<th>R</th>
<th>ΔR²</th>
<th>ΔF</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Fast ISD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.51</td>
<td>0.26</td>
<td></td>
<td>F(1, 133) = 45.99, <em>p</em> &lt; .001</td>
</tr>
<tr>
<td></td>
<td>Age²</td>
<td>0.55</td>
<td>0.04</td>
<td></td>
<td>F(1, 132) = 8.11, <em>p</em> = .005</td>
</tr>
<tr>
<td>2</td>
<td>Slow ISD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.49</td>
<td>0.24</td>
<td></td>
<td>F(1, 133) = 42.54, <em>p</em> &lt; .001</td>
</tr>
<tr>
<td></td>
<td>Age²</td>
<td>0.52</td>
<td>0.03</td>
<td></td>
<td>F(1, 132) = 5.28, <em>p</em> = .023</td>
</tr>
<tr>
<td>3</td>
<td>Slow ISD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fast ISD</td>
<td>0.31</td>
<td>0.1</td>
<td></td>
<td>F(1, 133) = 14.51, <em>p</em> &lt; .001</td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.5</td>
<td>0.15</td>
<td></td>
<td>F(1, 132) = 26.24, <em>p</em> &lt; .001</td>
</tr>
<tr>
<td></td>
<td>Age²</td>
<td>0.52</td>
<td>0.03</td>
<td></td>
<td>F(1, 131) = 4.46, <em>p</em> = .037</td>
</tr>
</tbody>
</table>

*Note.* Effect size and tests of statistical significance are provided for the step in which the effect in question and all lower-order polynomials were included in the regression equation. ISD = intraindividual standard deviation; Fast ISD = ISD within the fastest 25% of trials; Slow ISD = ISD within the slowest 25% of trials.
Table 4.

Summary of hierarchical multiple regression analyses of inconsistency and age in the older half (age $\geq 21$) of the sample (n=138) from study 1.

<table>
<thead>
<tr>
<th>Analysis</th>
<th>y Entered</th>
<th>x Entered</th>
<th>$R$</th>
<th>$\Delta R^2$</th>
<th>$\Delta F$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Fast ISD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.2</td>
<td>0.04</td>
<td>$F(1, 136)= 5.65, p = .019$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age$^2$</td>
<td>0.2</td>
<td>0</td>
<td>$F(1, 135)= .00, p = .980$</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Slow ISD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.39</td>
<td>0.15</td>
<td>$F(1, 136)= 24.81, p &lt; .001$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age$^2$</td>
<td>0.4</td>
<td>0.01</td>
<td>$F(1, 135)= 1.19, p = .227$</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Slow ISD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fast ISD</td>
<td>0.03</td>
<td>0</td>
<td>$F(1, 136)= .16, p = .688$</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.41</td>
<td>0.17</td>
<td>$F(1, 135)= 26.98, p &lt; .001$</td>
<td></td>
</tr>
</tbody>
</table>

Note. Effect size and tests of statistical significance are provided for the step in which the effect in question and all lower-order polynomials were included in the regression equation. ISD = intraindividual standard deviation; Fast ISD = ISD within the fastest 25% of trials; Slow ISD = ISD within the slowest 25% of trials.
Table 5.

*Means and standard deviations of demographic, cognitive, and outcome variables across four groups of study 2.*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n=29)</th>
<th>ADHD (n=23)</th>
<th>RD (n=7)</th>
<th>ADHD+RD (n=13)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Age</td>
<td>15.01</td>
<td>1.13</td>
<td>14.88</td>
<td>1.26</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male (n)</td>
<td>13</td>
<td></td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Female (n)</td>
<td>16</td>
<td></td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>WISC-III</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Estimated FSIQ</td>
<td>110.03</td>
<td>13.33</td>
<td>103.35</td>
<td>9.45</td>
</tr>
<tr>
<td>Vocabulary</td>
<td>11.31</td>
<td>2.69</td>
<td>10.74</td>
<td>2.26</td>
</tr>
<tr>
<td>Block Design</td>
<td>12.10</td>
<td>3.33</td>
<td>10.43</td>
<td>1.62</td>
</tr>
<tr>
<td>FDI</td>
<td>109.22</td>
<td>14.20</td>
<td>102.68</td>
<td>15.96</td>
</tr>
<tr>
<td>PSI</td>
<td>119.19</td>
<td>12.52</td>
<td>101.00</td>
<td>13.61</td>
</tr>
<tr>
<td>Mean CRT</td>
<td>368.86</td>
<td>71.08</td>
<td>361.40</td>
<td>44.55</td>
</tr>
<tr>
<td>ISD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All Trials</td>
<td>7.40</td>
<td>2.80</td>
<td>7.28</td>
<td>2.13</td>
</tr>
<tr>
<td>Fastest 25%</td>
<td>5.19</td>
<td>2.32</td>
<td>5.93</td>
<td>1.71</td>
</tr>
<tr>
<td>Slowest 25%</td>
<td>6.35</td>
<td>2.77</td>
<td>6.08</td>
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<tr>
<td>Stroop</td>
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<tr>
<td>Word (T)</td>
<td>50.26</td>
<td>4.90</td>
<td>46.93</td>
<td>7.05</td>
</tr>
<tr>
<td>Color (T)</td>
<td>51.07</td>
<td>7.29</td>
<td>44.96</td>
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</tr>
<tr>
<td>Color-Word (T)</td>
<td>60.00</td>
<td>11.20</td>
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</tr>
<tr>
<td>Interference (T)</td>
<td>57.69</td>
<td>10.35</td>
<td>57.61</td>
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</table>

Note. FDI= freedom from distractibility index, PSI=processing speed index, CRT=choice reaction time, and ISD= intraindividual standard deviation.
Table 6.

Summaries of the final step of hierarchical linear regressions (study 2) demonstrating the increment in the prediction of various cognitive variables by ISD over and above the grouping variables (ADHD, RD) and their interaction (ADHD by RD).

<table>
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<tr>
<th>Analysis</th>
<th>Y</th>
<th>R</th>
<th>R²</th>
<th>Δ R²</th>
<th>ΔF</th>
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<td>WISC-III</td>
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</tr>
<tr>
<td>1</td>
<td>Estimated FSIQ</td>
<td>.408</td>
<td>.167</td>
<td>.026</td>
<td>(F(1,67)=2.11, p=.151)</td>
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<tr>
<td>2</td>
<td>Vocabulary</td>
<td>.430</td>
<td>.184</td>
<td>.001</td>
<td>(F(1,67)=0.11, p=.744)</td>
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<tr>
<td>3</td>
<td>Block Design</td>
<td>.428</td>
<td>.184</td>
<td>.112</td>
<td>(F(1,67)=9.19, p=.003)</td>
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<tr>
<td>4</td>
<td>FDI</td>
<td>.602</td>
<td>.362</td>
<td>.046</td>
<td>(F(1,64)=4.65, p=.035)</td>
</tr>
<tr>
<td>5</td>
<td>PSI</td>
<td>.614</td>
<td>.377</td>
<td>.067</td>
<td>(F(1,63)=6.81, p=.011)</td>
</tr>
<tr>
<td>Stroop</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Word (T)</td>
<td>.643</td>
<td>.413</td>
<td>.034</td>
<td>(F(1,67)=3.86, p=.054)</td>
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<tr>
<td>7</td>
<td>Color (T)</td>
<td>.628</td>
<td>.394</td>
<td>.116</td>
<td>(F(1,67)=12.85, p=.001)</td>
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<tr>
<td>8</td>
<td>Color-Word (T)</td>
<td>.581</td>
<td>.338</td>
<td>.076</td>
<td>(F(1,67)=7.68, p=.007)</td>
</tr>
<tr>
<td>9</td>
<td>Interference (T)</td>
<td>.308</td>
<td>.095</td>
<td>.031</td>
<td>(F(1,67)=2.31, p=.133)</td>
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</table>

Note. FDI= freedom from distractibility index, PSI=processing speed index
Table 7.

Descriptive statistics displayed for the entire sample and across the three groups of study 3.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (n=28)</th>
<th>ADHD (n=86)</th>
<th>ADHD+RD(n=40)</th>
<th>Total (N=154)</th>
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<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
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<td>Gender</td>
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<td>Male (n)</td>
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<tr>
<td>Female (n)</td>
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<td>13</td>
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<tr>
<td>WISC-III</td>
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</tr>
<tr>
<td>FSIQ</td>
<td>114.15</td>
<td>13.93</td>
<td>103.18</td>
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<td>VIQ</td>
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<td>PIQ</td>
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<td>15.37</td>
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<td>Parent-based symptoms</td>
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<tr>
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<td>2.06</td>
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<td>Hyperactive/Impulsive</td>
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<td>5.87</td>
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<td>Teacher-based symptoms</td>
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<tr>
<td>Inattentive</td>
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<td>5.83</td>
<td>1.83</td>
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<tr>
<td>Hyperactive/Impulsive</td>
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<td>1.42</td>
<td>4.20</td>
<td>2.49</td>
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<td>Combined symptoms</td>
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<tr>
<td>Inattentive</td>
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<td>1.44</td>
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<tr>
<td>Hyperactive/Impulsive</td>
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<td>2.32</td>
<td>7.19</td>
<td>1.81</td>
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<tr>
<td>Mean CRT</td>
<td>463.37</td>
<td>52.81</td>
<td>497.71</td>
<td>89.67</td>
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<td>ISD</td>
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<tr>
<td>All Trials</td>
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<td>1.56</td>
<td>8.31</td>
<td>1.98</td>
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<td>Fastest 25%</td>
<td>4.48</td>
<td>1.91</td>
<td>5.11</td>
<td>2.36</td>
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<tr>
<td>Slowest 25%</td>
<td>5.49</td>
<td>2.38</td>
<td>6.66</td>
<td>2.58</td>
</tr>
</tbody>
</table>

Note. VIQ= verbal intelligence quotient, PIQ=Performance intelligence quotient, CRT=choice reaction time, and ISD= intraindividual standard deviation.
Table 8.

Zero-order correlations between measures of inconsistency (ISD, ISD fast, & ISD slow), symptom severity (Parent-based, Teacher-based, & Combined), and age in the entire sample (N=154) of study 3.

<table>
<thead>
<tr>
<th></th>
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<th>2</th>
<th>3</th>
<th>4</th>
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<td>ISD fast</td>
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<tr>
<td>ISD slow</td>
<td>.655**</td>
<td>.032</td>
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<tr>
<td>Age</td>
<td>-.323**</td>
<td>-.409**</td>
<td>-.038</td>
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<td>.448**</td>
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<td>TTI Inattentive</td>
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<tr>
<td>TTI-Hyperactive/Impulsive</td>
<td>.181*</td>
<td>.137</td>
<td>.095</td>
<td>-.251**</td>
<td>.194*</td>
<td>.384**</td>
<td>.392**</td>
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<tr>
<td>Combined-Inattentive</td>
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<td>.115</td>
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<td>.511**</td>
<td>.837**</td>
<td>.388**</td>
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<tr>
<td>Combined-Hyperactive/Impulsive</td>
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<td>.118</td>
<td>.185*</td>
<td>-.235**</td>
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<td>.860**</td>
<td>.476**</td>
<td>.682**</td>
<td>.599**</td>
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</table>

Note. * p<.05  **p<0.1
Figures
Figure 1. Line graphs for 3 age groups of study 1 depicting choice reaction time (CRT) (residual T-Score) for each participant across 32 trials.
Figure 2. Scatter plot of inconsistency (intraindividual standard deviation (ISD) of residualized CRT) across the life span (study 1).
Figure 3. Line graphs for 4 groups of study 2 depicting choice reaction time (CRT) (residual T-Score) for each participant across 32 trials.
Figure 4. Group means (bar) and 2 standard deviations (error bars) of intraindividual standard deviation (ISD) of residualized CRT across four groups of study 2.
Figure 5. Line graphs for 3 groups of study 3 depicting choice reaction time (CRT) (residual T-Score) for each participant across 32 trials.
Figure 6. Group means of intraindividual standard deviation (ISD) of residualized CRT across three groups of study 3.