Motor timing deficits in children with Attention-Deficit/Hyperactivity disorder

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ABSTRACT

Children with Attention-Deficit/Hyperactivity Disorder (ADHD) are thought to have fundamental deficits in the allocation of attention for information processing. Furthermore, it is believed that these children possess a fundamental difficulty in motoric timing, an assertion that has been explored recently in adults and children. In the present study we extend this recent work by fully exploring the classic Wing and Kristofferson (1973) analysis of timing with typically developing children ($n = 24$) and children with ADHD ($n = 27$). We provide clear evidence that not only do children with ADHD have an overall timing deficit, they also time less consistently when using a similar strategy to typically developing children. The use of the Wing and Kristofferson approach to timing, we argue, will result in the discovery of robust ADHD-related timing differences across a variety of situations.

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1. Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is characterized by a persistent pattern of developmentally inappropriate levels of inattention, hyperactivity, and impulsivity (American Psychiatric Association, 2000). The high rates of heritability for ADHD suggest a genetic contribution, leading to investigations of cognitive endophenotypes in ADHD (Castellanos & Tannock, 2002; DiMaio, Grizenko, & Joober, 2003; Faraone & Doyle, 2001). However, the search for an elementary,

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behaviorally identifiable marker of ADHD that is not part of the symptomatology used in the DSM-IV definition has been elusive.

Past attempts to identify cognitive endophenotypes have almost exclusively focused on dysfunctions in the prefrontal cortex, namely executive functioning. In explicating these dysfunctions, however, both past theoretical and empirical work (for a review see Barkley (1997), and recent work by Rommelse and colleagues (Rommelse et al., 2008), suggest an endophenotypic component in ADHD related to time estimation and production. Individuals with ADHD and their non-affected siblings exhibited motor timing deficits compared to participants from families with no formally diagnosed or suspected ADHD behaviors or symptoms. Other studies, however, have failed to show differences in time estimation when comparing children with and without ADHD (see Toplak, Dockstader, & Tannock (2006) for a review).

Luman et al. (2009) examined timing variance of children with ADHD as well as children with ADHD and Oppositional Defiant Disorder (ODD) in a 1000 millisecond (ms) timed interval tapping task. The notion that ADHD is primarily a difficulty in response inhibition (Barkley, 1997) was supported by the observation that children with ADHD and children with ADHD + ODD underestimated the 1000 ms interval compared to typically developing children. Furthermore, children with ADHD exhibited a much larger timing variance than typically developing children.

Valera et al. (2010) utilized a timed tapping task and demonstrated that along with increased timing variability of adults with ADHD compared to adults without ADHD, neuro-anatomical areas of the central nervous system such as the cerebellum and basal ganglia, known to be motor timing areas, showed less activity for adults with ADHD compared to adults without ADHD. This result provides initial evidence that a tapping timing task can be used to capture fundamental neurological differences in ADHD.

Valera et al. (2010) and Luman et al. (2009) employed the most widely used and useful analytical model of time-keeping by Wing and Kristofferson (1973). However, in both studies, there was not a detailed analysis of what might be called Wing and Kristofferson behavior. For example, Luman et al. (2009) did not compute the classic motor and clock variances. Furthermore, Valera et al. did not report whether participants obeyed the fundamental assumptions of the Wing and Kristofferson model. Thus, in the current study, we examined timing in children with ADHD within the timing framework of Wing and Kristofferson. Furthermore, we fully explored how child participants with and without ADHD produce temporal intervals in a tapping task when the interval time series obeys the Wing and Kristofferson assumptions, compared to not obeying these assumptions.

In the Wing and Kristofferson (1973) model, it is assumed that timing is open-loop; participants are not basing the production of the next interval upon evaluating the duration of the previous interval(s). Wing and Kristofferson show how the variance of the time series can be decomposed into two additive components, the variance attributed to a central time-keeping process (clock), and the variance attributed to an implementation process (motor variance). Motor variance is computed from the covariance of adjacent intervals, termed the lag one covariance. The model computations require that the lag one covariance be negative. Furthermore, the lag one autocorrelation must be bounded between 0.0 and −.05. In other words, a long interval is followed by a short interval and vice versa, and the covariance cannot be greater than half of the total variance. Once the motor variance is calculated, the clock variance can be estimated by subtracting twice the implementation variance from the total variance.

One caveat is in order. The interval time series might drift from the prescribed rate. This "drift" increases the total variance and of course reduces the negativity of the lag one covariance. Thus, a time series is first detrended, on a trial by trial basis, to remove this unwanted source of variance and then the total detrended variance is partitioned into clock and implementation (motor) components (Keele, Pokorny, Corcos, & Ivry, 1985; Wing & Kristofferson, 1973).

If a time series of intervals is not consistent with the Wing and Kristofferson (1973) model, then time keeping might not be attributable to an open-loop central clock-like timing process (see Zelaznik, Spencer, & Ivry, 2008). In the present study we examined timing behavior across groups when the Wing and Kristofferson model was obeyed and not obeyed, respectively. By examining how timing precision differs for children with ADHD compared to children without ADHD in these conditions, we are able to examine various sources of timing precision.

Finally, timing variance is composed of other sources outside of the Wing and Kristofferson (1973) model. People can change strategies across trials, as well as within trials. Small alterations in behavior (a sneeze or a yawn) can produce large changes in timing variance. Because in the present work we are interested in clock-like timing, we want to have a maximum likelihood of capturing the variability due to the inherent nature of an unadorned clock-like timing process. Thus, we also report on the best eight trials in terms of timing precision. It is possible that children without ADHD are not better time-keepers than children with ADHD once these unwanted sources of variability have been removed. Zelaznik and colleagues have used this technique for over a decade, with great success, to examine timing precision (see Zelaznik et al., 2008).

Thus, we now re-examine the Valera et al. (2010) and Luman et al. (2009) work relative to the tenets of the Wing and Kristofferson (1973) model. Valera et al. found that in a timed tapping task, at a 500 ms goal interval, adults with ADHD exhibited a greater clock variance but not a greater motor variance compared to adult participants without ADHD. This result was interpreted as supporting a central time keeping deficit associated with ADHD, but not a motor output deficit. Luman et al. discussed the Wing and Kristofferson model, but did not conduct the classic and expected analyses. Instead they reported what appears to be trial to trial variability, not an interval time series variance.

Although the recent work of Valera et al. (2010) and Luman et al. (2009) supports the idea of a timing deficit in people with ADHD, the overall research literature offers conflicting messages. Timing in the millisecond range has been investigated in children and adolescents with ADHD with visual synchronized tapping (Rubia, Overmeyer et al., 1999; Rubia, Taylor, Taylor, & Sergeant, 1999; Rubia et al., 2001), auditory synchronized tapping (Pitcher, Piek, & Barrett, 2002), simultaneous visual and auditory synchronized tapping (Ben-Pazi, Gross-Tsur, Bergman, & Shalev, 2003), visual synchronized tapping followed by a continuation phase (Toplak & Tannock, 2005), and auditory synchronized tapping followed by a continuation phase (Tiffin-Richards, Hasselhorn, Richards, Banaschewski, & Rothenberger, 2004; Toplak & Tannock, 2005) (see Toplak et al., 2006, for a review). Because the current study focused on the continuation phase of tapping, it is of interest that, to our knowledge, only four of the above studies (Luman et al., 2009; Tiffin-Richards et al., 2004; Toplak & Tannock, 2005; Valera et al., 2010) included a continuation phase. One of these studies compared 10- to 13-year-old children with or without an ADHD diagnosis and reported no differences in timing (average inter-response interval or inter-response interval variability) in the continuation phase (Tiffin-Richards et al., 2004). The second study reported a significantly greater coefficient of variation in the continuation phase of a visually-defined 1000-ms interval for 13- to 18-year-old children with an ADHD diagnosis (Toplak & Tannock, 2005). Notably, neither of these studies employed the Wing and Kristofferson (1973) analysis.

Valera et al. (2010) conducted the Wing and Kristofferson (1973) analysis on the continuation portion of the time series (the Wing and Kristofferson decomposition should not be conducted on the synchronization portion of the trial). Given the importance of the Valera study in examining timekeeping in a principled and theoretical fashion, we present a comparable behavioral study that examines children with ADHD and typically developing children. In pursuing this aim, we extend Valera et al. and Luman et al. (2009) by reporting on key details of the Wing and Kristofferson analysis. Accordingly, we provide the first detailed Wing and Kristofferson analysis of timing in tapping for children with and without ADHD. Furthermore, we examined timing variability as a function of the child performing their best, or not. By providing detailed analyses of timing, we hope to be able to begin to delineate a behavioral marker of ADHD that is not part of the diagnostic symptomology.

2. Method

2.1. Participants

Children ($N = 51$) aged 7–12 years were recruited through an ongoing, unrelated study ($n = 39$) and through a local elementary school ($n = 12$). Recruitment was designed to gain a representative sample through the use of school settings, primary medical care settings, mental health practitioners, and self-referrals solicited through advertisements and word of mouth. Participants with ADHD ($n = 27$) for the current study met criteria either for ADHD, Combined Type (ADHD-C) or ADHD, Predominantly

Hyperactive/Impulsive Type (ADHD-HI) following the assessment procedures discussed below[1]. Children with ADHD had a mean age of 9.50 (*SD* = 1.06) years. The majority were male (77.8%) and Caucasian (88.9%).

Children recruited for the ADHD group received a comprehensive ADHD assessment following guidelines established by the American Academy of Pediatrics (2000) and practice parameters outlined by the American Academy of Child and Adolescent Psychiatry (Pliszka, Bernet, Bukstein, & Walter, 2007). Specifically, information was gathered through the use of parent and teacher symptom-based and empirically-derived rating scales, a comprehensive structured diagnostic interview, and a semi-structured clinical interview (regarding developmental, social, academic, and family functioning). Child-based measures included: (1) cognitive ability and achievement tests; (2) self-report measures of self-perceptions, anxiety symptoms, and depressive symptoms; and (3) an informal clinical interview regarding family, school, and peer functioning. Licensed psychologists or trained graduate-level research assistants supervised by licensed clinical psychologists administered the clinical assessment. Diagnosis of ADHD was agreed upon by two independent, doctoral-level (Ph.D.) clinicians.

Children not meeting criteria for ADHD (*n* = 24) were recruited either using similar recruitment sources as children with ADHD (*n* = 12) or through a streamlined school-based recruitment (*n* = 12). Participants without ADHD were recruited to be of similar age (*M* = 9.82 years; *SD* = 1.02), gender (75.0% male), and race (83.3% Caucasian) as children with ADHD. No differences in age, $F(1, 49) = 1.16$, $p > .05$; gender, $\chi^2(1) = 0.05$, $p > .05$; or race, $\chi^2(1) = 0.33$, $p > .05$, existed across groups. Parent and teacher ratings of ADHD symptoms were obtained for all children not diagnosed with ADHD, regardless of recruitment strategy employed, documenting that symptom criteria were not met for an ADHD diagnosis.

Children with a Brief Intellectual Ability standard score below 80 on the Woodcock-Johnson III Tests of Cognitive Abilities (Woodcock, McGrew, & Mather, 2001) or a previous diagnosis of any pervasive developmental disorder were excluded from the study. Twelve children without ADHD who were recruited through a local school did not receive IQ testing. All were from a mainstream classroom and no learning difficulties were reported by their classroom teacher; hence they were included as study participants.

Children typically receiving stimulant medications did not take medication on the day of testing. If needed, medication was administered following completion of research tasks.

2.2. Timing task

Participants pressed the space bar of a USB keyboard in synchrony with a metronome (10 ms beep duration, 1600 Hz frequency, and a 490 ms beep onset asynchrony) for 10 synchronized intervals (11 beeps), followed by enough time to continue at that 500 ms period for about 40 intervals. Participants completed 15 trials of this task. There were no practice trials, but we did not use the first two trials of tapping in any analysis.

2.3. Apparatus

The apparatus consisted of a Dell GX270 computer, a standard Dell keyboard, a 17 inch (diagonal measurement) flat panel LCD monitor, and a Harman/Kardon loudspeaker. Matlab running the Psychology Toolbox controlled the metronome and computed interval duration to the nearest millisecond.

2.4. Procedure

All recruitment, informed parental consent (and child assent), and experimental procedures were reviewed and approved by all appropriate institutional review boards. Data collection occurred at a

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[1] Predominantly Inattentive Type (ADHD-PI) were excluded because of the controversy in the literature regarding whether ADHD-PI constitutes a separate or related disorder, given its unique etiology, core deficits, associated features, and co-morbidity profile (Milich, Balentine, & Lynam, 2001). In light of our detailed analysis, also we were concerned that these children would have a difficult time meeting the Wing and Kristofferson (1973) model assumptions.

mid-sized, public Northeastern U.S. university or, for some children without ADHD, at their school in the area of that Northeastern University. After procuring informed parental consent and child assent, participants completed the timing task individually with a research assistant present in a quiet, well-lit room in order to minimize distractions. Following verbal instructions provided by the research assistant reviewing the goals of the timing task, the child was seated in front of a keyboard placing the index finger of the dominant hand on the space bar. A warning tone was presented and then one second later the metronome engaged. As instructed, the child attempted to entrain her or his tapping with the metronome, attempting to press the space bar coincident with the beep. Following 11 beeps, the metronome disengaged and the participant attempted to continue producing tapping intervals at the 500 ms prescribed rate, consistently and as accurately as possible. The trial ended with a 2400 Hz, 100 ms duration beep. There was approximately a 20 s intertrial interval.

3. Results

For each trial we excluded the first two intervals and the last interval of continuation from all analyses. Furthermore, for the Wing and Kristofferson (1973) analyses, the time series of the remaining contiguous intervals was linearly detrended to remove the contribution of drift in interval duration to timing variance (Zelaznik et al., 2008). We then computed the variance of the detrended time series and computed the coefficient of variation, defined as the ratio of the standard deviation of the time series to the average interval produced, converted to a percentage. We also computed the variance for the raw time series. The difference in variance between the raw and detrended time series is one way to determine the level of drift in the time series, and to compare the drift across different groups of research participants.

Timing behavior was examined for all trials. In a separate analysis only the eight lowest coefficient of variation trials were analyzed. The latter procedure has been used by Zelaznik and colleagues (Robertson et al., 1999; Zelaznik, Spencer, & Ivry, 2002) in order to examine timing with minimal variance contributed by changing strategies within a trial, and changing strategies across trials.

3.1. Descriptive data

3.1.1. All trials

In Fig. 1 we present the average interval duration (top panel) and the coefficient of variation (bottom panel) for each group. There was a tendency for the children without ADHD to produce longer intervals than children with ADHD (488 versus 469 ms), $t(49) = 1.94$, $p = .06$, and a large difference (about 7%) in coefficient of variation. The children without ADHD were more precise (lower coefficient of variation) than children with ADHD, $t(49) = 6.45$, $p < .0001$

3.1.2. Best eight trials

In Fig. 2 we present the same two dependent variables using the eight trials with the lowest coefficient of variation data. The 22 ms difference in average cycle duration (children without ADHD exhibited longer duration than children with ADHD) is significant, $t(49) = 2.11$, $p < .05$. A smaller difference in the coefficient of variation (children without ADHD were 3% more precise than children with ADHD) is observed compared to the all trials analysis, but still is statistically significant, $t(49) = 3.92$, $p < .001$

3.1.3. Time series analyses

We examined the structure of the time series in two ways. First, we compared the difference between the variance of the raw time series and the detrended time series. If there was no temporal drift, then the variance of the raw time series and the detrended time series are equal (i.e., difference is zero). The difference for the children without ADHD was 210 ms^2 and for the children with ADHD was 558 ms^2, $t(49) = 3.57$, $p < .001$. Therefore, not only did the children with ADHD exhibit a larger detrended variance (recall the coefficient of variation results), they also exhibited a greater propensity to drift in the time series. Thus, children with ADHD exhibited two timing deficiencies. They were
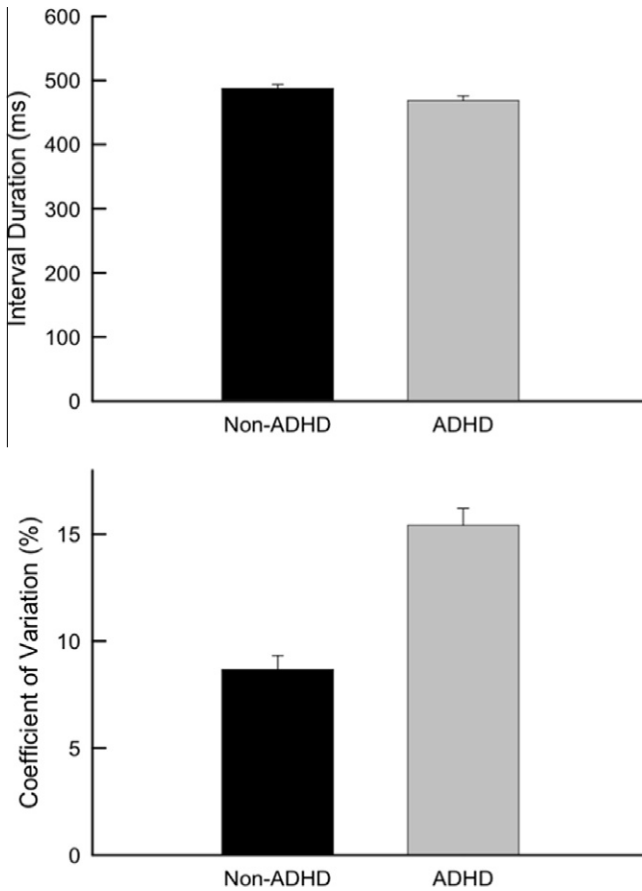
Fig. 1. Average tapping interval duration (top panel) and coefficient of variation (bottom panel) by group for all trials. The bars are two standard errors of the mean.

inherently more variable, and they had a greater propensity to drift away from the prescribed rate compared to the children without ADHD.

Second, the variance in the time series was partitioned according to the Wing and Kristofferson (1973) model of timing, using all 15 trials. By determining how well the time series conforms to the Wing and Kristofferson assumption, we can make inferences about the integrity of clock-like timing (see Robertson et al., 1999; Zelaznik et al., 2008). Seventy percent of trials performed by children without ADHD exhibited a negative lag one covariance. For children with ADHD, only 51% of trials exhibited a negative lag one covariance, $t(49) = 4.03$, $p < .001$. Thus, children with ADHD did not conform as well to the archetypal timing model as children without ADHD.

We recalculated coefficient of variation scores, only including trials with a negative lag one covariance, and the two groups of participants remained different. Children without ADHD exhibited a coefficient of variation of 8.2%, and children with ADHD produced a coefficient of variation of 13.8%, $t(49) = 4.60$, $p < .0001$. We calculated the Wing and Kristofferson (1973) variance decomposition on these trials. We found that children with ADHD exhibited a greater clock variance than children without ADHD (5471 versus 1533 ms^2), $t(49) = 2.68$, $p = .01$. Furthermore, motor variances were different for the two groups. Children with ADHD exhibited a motor variance of 1207 ms^2 and children without ADHD exhibited a motor variance of 447 ms^2, $t(49) = 3.04$, $p < .01$. This latter result differs from that obtained from the adult participants in Valera et al. (2010).
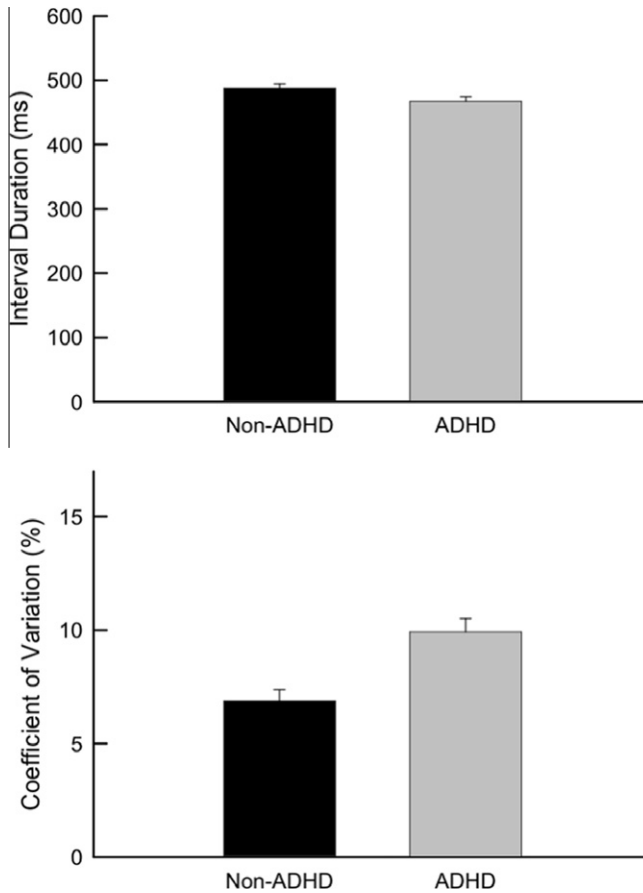
Fig. 2. Average tapping interval duration (top panel) and coefficient of variation (bottom panel) by group for best 8 trials. The bars are two standard errors of the mean.

In Fig. 3 we present the analysis of the lag covariance for lags 1 through 5. If participants are timing with an open-loop strategy the lag covariance values for lags greater than one will be equivalent to zero. A negative lag covariance value at lags greater than one is evidence that feedback is being utilized to adjust interval durations, and thus the lag one covariance value does not represent implementation variance. We are not interested in lag differences between groups, nor whether there is a lag effect. We are interested in whether a lag value significantly differs from zero and, thus, is negative. Thus, at each lag for each group we tested the hypothesis that the value of the lag was zero. Because we are conducting 10 t-tests, we set significance to .01 to minimize type I errors. For the children with ADHD, lag one was significantly below zero $t(26) = -5.3$, $p < .001$, as was lag three, $t(26) = -3.0$, $p < .01$. For the children without ADHD, only the first lag value was significantly below zero, $t(23) = -6.5$, $p < .001$. Thus, children with ADHD did not use solely an open-loop timing strategy.

4. Discussion

The basic finding of the present work is straightforward and important. On a non-complicated timing task, purportedly capturing the basic integrity of clock-like timing processes, children with ADHD
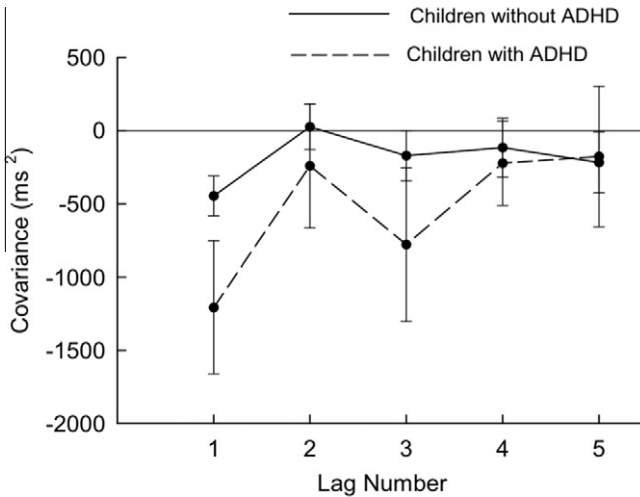
Fig. 3. Lag covariances for lags one through five by group. The bars are two standard errors of the mean.

were temporally less precise than their typically developing counterparts. Furthermore, this difference was exhibited regardless of whether all trials or the most temporally precise trials were examined.

Examining timing under best (perhaps even optimal) performance is thought to be a window into the basic operation of movement timing procedures (Zelaznik et al., 2008). The variance of a relatively short time series is sensitive to an interval that is too long or too short. Instead of trying to determine which trials and/or intervals are outliers, we compared timing performance based upon different criteria. The analysis of timing for the best trials compared to all trials lead us to infer that children with ADHD exhibit a fundamental timing decrement (best trials), but also potentially a strategic one (all trials), respectively.

We also see that children with ADHD drift off the mean tapping rate to a greater extent compared to children without ADHD. The difference between the total variance and the detrended variance was twice as large in the children with ADHD compared to the children without ADHD. We infer that children with ADHD exhibit a deficit in executive control necessary for monitoring the quality of their own performance and making adjustments to remain on the target pace. Such a conclusion is consistent with prior theoretical arguments (e.g., Barkley, 1997) that the executive functioning deficits associated with ADHD contribute to impaired self-regulation.

Additional support for the notion that children with ADHD have a deficit in inhibitory processes is seen in the mean rate data. Overall, children with ADHD produced shorter intervals than children without ADHD. Not being able to inhibit keypressing may have produced these shorter time intervals.

We also observed that the detrended time series for children with ADHD did not adhere to the assumptions of the Wing and Kristofferson (1973) timing decomposition model compared to children without ADHD. The crucial assumption is that timing control is open loop. However, children with ADHD seem less able to adopt the open-loop strategy. Open-loop control is thought not to be attention demanding (Zelaznik, Shapiro, & McClosky, 1981). If children with ADHD have "deficits" in attention capacity, they would be expected to tend toward more open-loop control. We show this is not the case. Less of their trials fit the Wing and Kristofferson timing model, and those trials exhibiting negative lag one covariance also exhibit negative lag three covariance, implicating closed-loop control of timing.

We speculate that the motor timing difficulties of our participants with ADHD are due to a difficulty with inhibiting executive control and other attention demanding processes. This leads to interesting questions for future research. For example, if attention is occupied with a secondary executive task along with the tapping timing task, will children with ADHD then perform the tapping task in an

open-loop fashion and exhibit stronger Wing and Kristofferson (1973) behavior? The results are consistent with the Barkley (1997) hypothesis that lack of inhibition is core to ADHD. Children with ADHD have difficulty inhibiting their response to feedback, and these processes thus produce increased temporal variability.

Valera et al. (2010) showed that only the clock variance was greater in adults with ADHD compared to adults without ADHD. The motor variance component was equivalent between these two populations. In our study, albeit with children, we showed that there were motor and clock differences between children with ADHD and their typically developing counterparts. There are three possible explanations for the discrepancy between our study and Valera et al. First, it is not clear whether Valera et al. only examined trials in which the time series exhibited a negative lag one covariance. If trials with positive and negative lag one covariance were included, the motor variances would tend to be underestimated, increasing the likelihood of not finding a difference between participant populations. Second, many studies have documented that developmental coordination disorder (DCD) is expressed in a significant proportion of the ADHD population (see Rasmussen & Gillberg, 2000). We did not screen for DCD, nor did Valera et al. inquire whether their adult subjects were diagnosed with DCD during childhood. Thus, it is possible that differences in prevalence of DCD could explain our differences. Finally, Valera et al. did not report on lag covariances for lags greater than one. Recall that children with ADHD showed a negative lag three covariance. This signifies feedback control, and of course is coupled with an increase in the negative lag one covariance, also due to feedback control (this latter suggestion rests on the result that for children with ADHD, the lag 3 covariance was significantly less than zero, i.e., was negative). Perhaps adults adapt over time and let open loop processes become more dominant in the performance of simple repetitive skills such as tapping. Obviously, the motor variance differences as a function of ADHD in children require replication prior to exploring the efficacy of explanations.

One likely source of increased variability in timed-interval tapping in the children with ADHD is the cerebellum. The cerebellum has been shown to play a critical role in precise timing required by the timed-interval tapping task (Ivry, Keele, & Diener, 1988; Spencer, Zelaznik, Diedrichsen, & Ivry, 2003). Recent studies have documented cerebellar abnormalities in children and adolescents with ADHD. For example, these individuals have a smaller cerebellum (Castellanos et al., 1996; Durston et al., 2004; Mackie et al., 2007; Mostofsky, Reiss, Lockhart, & Denckla, 1998; Valera, Faraone, Murray, & Seidman, 2007) and lower density of white matter (Ashtari et al., 2005). Furthermore, Durston et al. (2007) showed children and adolescents with ADHD to have abnormal cerebellar activation to the timing of stimulus events in a go/no-go task. Also, Valera et al. (2010) documented, with fMRI, cerebellar differences between adult participants with ADHD and without ADHD.

Eyeblink classical conditioning is another task that has been used in timing research and that is dependent upon the cerebellum (Christian & Thompson, 2003). Timed-interval tapping and eyeblink conditioning share the need for precise timing and appear to rely on a common mechanism for representing temporal information (Green, Ivry, & Woodruff-Pak, 1999), called event timing (Zelaznik et al., 2008). Children with ADHD exhibit abnormal timing of learned eyeblinks (Coffin, Baroody, Schneider, & O'Neill, 2005; Frings et al., 2010). Furthermore, Green and colleagues have demonstrated that two different rat models of ADHD (spontaneously hypertensive rats [SHRs] and Wistar-Kyoto hyperactive [WKHA] rats) exhibit abnormal timing of conditioned eyeblinks in eyeblink conditioning (Chess & Green, 2008; Thanellou, Schachinger, & Green, 2009).

The fact that two tasks that index cerebellar function (timed-interval tapping; eyeblink conditioning) reveal imprecise event timing in children with ADHD, and the fact that rodent models of ADHD show abnormal timing in eyeblink conditioning suggests a role for the cerebellum in ADHD. Poorer event timing in children with ADHD may be caused by abnormal cerebellar processing and may play a role in some of the impulsive behaviors these children exhibit.

In summary, we have shown that a detailed analysis of timed behavior, motivated by a classic model of timing (Wing & Kristofferson, 1973), can document and unravel different types of timing processes in children with ADHD. Future research that is designed to clarify such processes will meaningfully extend the knowledge base on ADHD.

Acknowledgments

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