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Relation Between Outcomes on a Continuous Performance Test and ADHD Symptoms Over Time

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ADHD is among the most frequently diagnosed disorders of childhood and is defined by developmentally inappropriate levels of inattention, hyperactivity, and impulsivity (American Psychiatric Association, 2000). Research strongly suggests children with ADHD have significant neuropsychological dysfunction (Barkley, 1997; Faraone & Biederman, 1998) that persists into adulthood (Hervey, Epstein, & Curry, 2004). Children with ADHD exhibit a wide range of performance deficits across a range of neuropsychological domains including response inhibition, working memory, planning, sense of time, sustained attention, and verbal learning (Barkley, Grodzinsky, & DuPaul, 1992; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Nigg, Blaskey, Huang-Pollock, & Rappley, 2002; Nigg, Butler, Huang-Pollock, & Henderson, 2002; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005; Seidman, 2006; Seidman, Benedict et al., 1995; Seidman, Biederman, Faraone, Weber, & Ouellette, 1997; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Despite the considerable evidence documenting the presence of neuropsychological deficits in individuals with ADHD, questions still exist regarding the persistence and course of these deficits over time. The current study begins to address this gap in the literature by examining the trajectory of specific neuropsychological deficits as measured by outcomes on a continuous performance test (CPT).

Understanding the developmental course of neuropsychological functioning in individuals with ADHD has descriptive as well as theoretical implications. Descriptively, ADHD-related neuropsychological deficits have been documented across the lifespan, albeit using primarily cross-sectional samples (Barkley et al., 1992; Seidman, Biederman et al., 1995; Seidman et al., 1997; Seidman, Biederman, Weber, Hatch, & Faraone, 1998). Although research has documented improvement in neuropsychological performance through adolescence into young adulthood (Biederman, Petty, Fried, Doyle, Spencer, Seidman et al., 2007; Fischer, Barkley, Smallish, & Fletcher, 2005), little is known regarding the effects of such neuropsychological improvements in terms of the presence or magnitude of deficits especially in relation to developmentally-related changes in core ADHD symptomatology.

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Theoretically, investigators have hypothesized that the developmental course of ADHD-related neuropsychological deficits may provide insights into which neuropsychological deficits are core to the disorder and which are epiphenomenal (Carr, Nigg, & Henderson, 2006; Halperin & Schulz, 2006). Some have suggested that attenuation in neuropsychological deficits, specifically when this attenuation parallels reductions in ADHD symptomatology, may be indicative of epiphenomenal rather than core ADHD deficits (Carr et al., 2006; Halperin, Trampush, Miller, Marks, & Newcorn, 2008). Recently, Halperin and Schulz (2006) proposed that developmentally-related attenuation in executive functioning deficits among patients with ADHD is likely caused by prefrontal cortex maturation which promotes compensatory cognitive and neural mechanisms in patients. In support, they cite the parallel developmental course of prefrontal cortex during late childhood and adolescence and the typical attenuation in ADHD symptomatology during this same span (Biederman, Mick, & Faraone, 2000; Hill & Schoener, 1996). Halperin and Schulz (2006) further argue that core ADHD deficits are more likely linked to non-cortical structures (e.g., striatum, cerebellum); hence, neuropsychological measures that assess function in these structures should remain relatively constant throughout the lifespan irrespective of ADHD symptom status.

In order to examine the developmental course of ADHD symptoms and neuropsychological deficits, longitudinal studies including children with and without ADHD with repeated assessment of neuropsychological function are necessary. However, few such longitudinal studies with ADHD samples exist (Biederman et al., 2008; Drechsler, Brandeis, Foldenyi, Imhof, & Steinhausen, 2005; Fischer et al., 2005; Halperin et al., 2008; Hinshaw, Carte, Fan, Jassy, & Owens, 2007). Of the existing longitudinal studies, most have collected neuropsychological data with groups of children with and without ADHD using a follow-up assessment in late adolescence or young adulthood. The typical lag between assessments ranges from 5 to 9 years. In summary, these studies demonstrated that 1) children with ADHD have poorer neuropsychological performance across a range of measures compared to normal controls; 2) neuropsychological performance improves from childhood to adolescence/young adulthood among both ADHD and normal control samples; and 3) despite time-related improvements in neuropsychological performance, patients diagnosed with ADHD in childhood continue to demonstrate poorer neuropsychological performance in adolescence/young adulthood than normal controls (Drechsler et al., 2005; Fischer et al., 2005; Halperin et al., 2008; Hinshaw et al., 2007). Results also suggested that individuals meeting criteria for a diagnosis of ADHD at both time points had the poorest levels of neuropsychological performance (Biederman et al., 2007; Hinshaw et al., 2007). One criticism of these studies is the lengthy lag between assessment points.

One study (Drechsler et al., 2005) using a shorter time lag between neuropsychological assessments examined a small group (n=28) of children with ADHD aged 8–13 and a group of age-matched normal controls (n=25) yearly over 2 years. The only neuropsychological outcome on which the ADHD group performed more poorly than the normal controls was variability in reaction time. However, this between-group effect was present only at the Time 1 and Time 2 assessments. At the third assessment, between-group differences were no longer present with both groups improving from the Time 2 to Time 3 assessment. There was an interesting interaction effect indicating that improvement was more pronounced for children with ADHD than was demonstrated for normal controls. Their results suggested that neuropsychological development appeared to be non-linear and possibly differentially non-linear across ADHD and normal control groups. Indeed, data exist for both normal control and ADHD samples demonstrating that brain development progresses at different rates especially in the teenage years (Barnea-Goraly et al., 2005; Krain & Castellanos, 2006) and that these rates of brain development may be different in ADHD samples versus normal controls (Castellanos et al., 2002; Shaw et al., 2007). These results suggest that research

examining shorter intervals within critical developmental periods (e.g., early adolescence) is necessary to clarify the developmental course of neuropsychological functioning.

In addition to examining changes in neuropsychological performance over targeted developmental periods, it is equally relevant to examine how these developmentally-related neuropsychological changes correspond to changes in ADHD symptoms. As noted earlier, the relationship between neuropsychological performance and behavior has implications for determining core versus epiphenomenal neuropsychological deficits (Carr et al., 2006; Halperin et al., 2008). In regards to developmental changes in ADHD symptoms, despite high rates of documented persistence (Biederman et al., 2006), studies have shown that the actual presentation of ADHD symptoms tends to change over time (Barkley, Fischer, Edelbrock, & Smallish, 1990; Biederman et al., 1996; Hart, Lahey, Loeber, Applegate, & Frick, 1995). Significant declines (i.e., improvement) over time in hyperactive/impulsive symptoms have been shown with more stable trends in inattentive symptoms over the same period (Fischer et al., 2005; Hart et al., 1995). However, despite these documented decreases of hyperactivity and impulsivity with age, children with ADHD continue to exhibit non-normative levels of ADHD symptomatology (Barkley et al., 1990; Biederman et al., 1998; Molina et al., 2009).

A hypothesized relation between developmental trends in neuropsychological performance and trends in ADHD symptom change over time is not well-established. All research to date attempting to address this issue has examined ADHD symptoms dichotomously (i.e., either the patient meets ADHD criteria or not) and unitarily (i.e., examining ADHD symptom domains collectively). For example, Halperin and colleagues (Halperin et al., 2008) found that only individuals with a persistent diagnosis of ADHD continued to have difficulties with effortful executive processes over time; whereas, children failing to meet criteria for ADHD were no longer different from controls. The use of diagnostic stability may fail to accurately account for the heterotypic continuity in the developmental course of ADHD symptoms that is commonly observed in patients with ADHD (Biederman et al., 2000; Hart et al., 1995; Hinshaw, Owens, Sami, & Fargeon, 2006; Larsson, Lichtenstein, & Larsson, 2006; Larsson, Larsson, & Lichtenstein, 2004). That is, there seems to be significant attenuation in hyperactivity/impulsivity symptoms and maintenance of inattention symptoms from childhood to adolescence.

While the majority of findings suggest that ADHD symptoms and neuropsychological dysfunction are correlated (Nigg, 2005; Seidman, 2006), questions remain unanswered. For example, are observed improvements in neuropsychological functioning selectively related to hyperactivity/impulsivity, as would be expected given the observed improvements in this symptom domain, or are these improvements in neuropsychological functioning also related to inattentive symptoms? Also, are the relations between neuropsychological and behavioral improvements specific to particular neuropsychological outcomes? According to the hypothesis offered by Halperin and Schulz (2006), neuropsychological deficits on task outcomes measuring effortful processing (e.g., commission errors on a go/no-go task) should attenuate with maturation paralleling attenuation in ADHD symptomatology. On the other hand, neuropsychological deficits on outcomes measuring less conscious control (e.g., reaction time variability) should largely persist over time remaining unrelated to ADHD symptom presentation.

The purpose of the present study is threefold. First, we examined the trajectory of neuropsychological deficits, as measured by outcomes on a CPT task, in children with and without ADHD over a 1 year period. Second, we examined the trajectory of ADHD symptoms in each domain over the same period. Third, as no research has examined directly the relations among developmental changes in neuropsychological functioning and ADHD

symptomatology, we examined the correspondence between developmental changes in performance on a CPT and in ADHD symptomatology over the same period.

Children in the Multimodal Treatment Study of Children with ADHD (MTA) sample completed the Conners' CPT (i.e., a Go/No-Go task) twice with an approximate 1 year lag. Results from the first test administration demonstrated that children with ADHD responded more slowly, more variably, and with more errors when compared to a matched control group (Hervey et al., 2006). Consistent with previous longitudinal studies, albeit over a shorter course of time, we predict that children with ADHD as well as a matched normative comparison group will exhibit improved neuropsychological performance as evidenced by decreased reaction time, reaction time variability and errors of commission and omission with development (Fischer et al., 2005). As raw scores on the CPT task (e.g., errors, reaction time) are the primary dependent variable, age-related improvements across both groups are expected. A similar pattern would be observed if raw scores were used on achievement tests. The majority of previous research has not shown rates of improvement to be different across ADHD and normal control groups thus only a main effect, not an interaction effect, is predicted. Also consistent with previous research, we expect to confirm that hyperactive and impulsive symptoms will improve with development while inattention symptoms will remain stable. Finally, in respect to correspondences between neuropsychological function, we predict correlations between CPT outcomes and ADHD symptoms at each time point (Nigg, 2005; Seidman, 2006). Further, consistent with Halperin and Schulz's hypothesis (2006) we expect that changes in CPT outcomes measuring effortful control processes (i.e., omission and commission errors) will be related to ADHD symptom domain trajectories while other CPT outcomes measuring processes with little conscious control (i.e., reaction time variability) will demonstrate no relation to ADHD symptom trajectories.

Methods

Participants

The MTA is a large, randomized clinical trial (n = 579) jointly conducted by six independent research teams and the National Institute of Mental Health (NIMH). Subjects were children aged 7.0–9.9 who met full diagnostic criteria for ADHD, Combined type, using parent and teacher rating scales, and the Diagnostic Interview Schedule for Children Version IV (DISC-P). Exclusion criteria included children with a score below 80 on the WISC-III Verbal IQ, Performance IQ, or Full Scale IQ and/or on the Scales of Independent Behavior (Hinshaw et al., 1997). These children comprised the ADHD sample for the present study. Greater detail regarding the rationale, design, and results of the MTA study may be found elsewhere (Arnold et al., 1997; MTA Cooperative Group, 1999).

The original purpose of the study was to compare four treatment strategies for childhood ADHD over 14 months of treatment. Following the 14 month assessment, randomized treatments were terminated and families were able to seek any form of treatment for their child. At the 24 and 36-month assessments, all children were thoroughly assessed again using a comprehensive battery of measures. The retention rate of these children at 24-months (n=534) and 36-months (n=486) was 92% and 84%, respectively. The CPT was collected on 375 patients at the 24-month assessment point and 413 patients at the 36-month assessment point. We assessed whether those patients who completed the CPT differed from those who did not on ADHD behavioral ratings (i.e., parent and teacher SNAP ADHD Total Symptom Scores [TSS]). Children with no CPT data at 24-months had higher teacher TSS than children with CPT data ($p<.05$). There were no differences on parent ratings at 24-months nor were there differences between these groups on either parent or teacher ratings at 36-months (all $ps>.05$).

At the 24-month time point, a local, normative comparison group (LNCG, n = 289) was recruited to be of similar age and demographic characteristics as the children within the ADHD group. The recruitment strategy for the LNCG children was designed to reflect the local populations from which the ADHD sample was drawn. The same schools, grades, and sex proportions as were in the ADHD sample were targeted during LNCG recruitment. Exclusion criteria were parallel to that of the ADHD sample. These children were recruited at the 24-month time point and re-assessed at the 36-month time point.

For the purposes of the present study, LNCG children meeting criteria for any subtype of ADHD (n = 34) were removed from all analyses to avoid confounding groups. At 24 months, parent and teacher behavioral ratings were collected for 254 LNCG children and for 239 children completed behavioral ratings at 36 months; 220 children completed the CPT at the 24-month assessment point and 212 children completed the CPT at the 36-month assessment point. No differences between children who did and did not complete the CPT were found on parent or teacher behavioral ratings at 24 or 36 months.

Demographic comparisons between ADHD and LNCG children failed to show any significant differences regarding age, $F(1,787) = 1.12$, $p > .05$., sex, $\chi^2 (1,787) = 2.29$, $p > .05$, or ethnicity, $\chi^2 (1, 787) = .733$, $p > .05$. Children in the ADHD group had lower IQ estimates than children in the LNCG, $F (1,787) = 36.53$, $p < .001$. Demographic data for both groups is presented in Table 1.

Measures

Conners' Continuous Performance Test (CPT) (Conners, 1994)—The CPT was completed on an IBM-compatible desktop computer in a quiet setting to minimize distractions. Three hundred and sixty (360) total letters appeared on the computer screen, one at a time, each for approximately 250 milliseconds. The 360 trials were presented in the standard format of 18 blocks of 20 trials each. The blocks differed only in the interstimulus intervals (ISI) between letter presentations, and lasted 1-, 2-, or 4-seconds. Interstimulus intervals were randomized between blocks so that all three ISI conditions would occur every three blocks. Transition from one block to the next was unannounced and occurred without delay. Children were instructed to press the spacebar when any letter except the letter "X" appeared on the screen. The percentage of trials when letters other than "X" appeared was 90% across all ISI blocks. Reaction time was measured from the point at which any letter other than "X" appeared on the screen until the spacebar was depressed. This is considered a Go trial. No-Go trials occurred when an "X" was presented. Two types of errors were recorded. Errors of omission occurred when the participant failed to respond to a target stimulus. Errors of commission occurred when the participant responded to a non-target stimulus (i.e., "X"). The total Conners' CPT task takes approximately 14 minutes to complete. Summary measures used for this study included mean Go reaction time (RT), RT standard deviation (RTSD), percent errors of omission, and percent errors of commission

Swanson, Nolan, and Pelham Rating Scale (SNAP-IV) (Swanson, 1992)—The SNAP-IV is a parent and teacher rating scale comprised of 39 items derived from the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) criteria for ADHD and ODD. The 18 ADHD items (nine inattention, six hyperactive, and three impulsive symptoms) from the *DSM-IV* (American Psychiatric Association, 1994) were utilized in the current study. All items are rated on a 4-point Likert style scale indicating the severity of symptoms over the past four weeks. The SNAP-IV was completed by parents and teachers at each of the assessment points. For children in elementary school, only one set of teacher ratings was collected, primarily from the homeroom teacher. Children in middle school received up to three sets of teacher ratings. A composite score was calculated by averaging across teachers

to equate with ratings conducted in elementary school. To remain consistent with previous research examining symptom trajectories, the 18 ADHD items were examined by separating symptoms into subscales of inattention, hyperactivity, and impulsivity. Mean subscale scores were computed across each rater for each symptom domain. Although psychometric properties have not been examined for the SNAP-IV, similar DSM-IV checklists have exhibited adequate reliability and validity (Wolraich et al., 2003). Coefficient alphas ranged from .86 to .95 across all subscales and raters in the present sample.

Services Use in Children and Adolescents Parent Interview (SCAPI); Service Barriers and Attitudes (Jensen et al., 2004)—The SCAPI is a structured interview administered to parents capturing child and adolescent services use across mental health, primary care, school, and community settings. It was obtained every 6 months including the 24 and 36 month time points, either by phone or during face to face assessments. Medication use was queried at each time point. For this study, the percent of days in the interval between the last assessment and the current assessment that any stimulant medication was taken was used as an indicator of ADHD medication usage for analyses examining ADHD symptomatology. For analyses examining CPT tasks, the child's medication use on the day of testing was included. Test-retest reliability using an 18 day between test interval for reporting medication use on the SCAPI was excellent (kappa = .97) (Hoagwood et al., 2004).

Procedures

Informed consent was obtained for all participating families using procedures approved by local Institutional Review Boards at each site. Children with ADHD and their parents completed informed consent during the baseline visit of the MTA study. The LNCG participants and their parents were consented at the 24-month follow-up for the MTA study. Children in both groups were administered the CPT as part of a more comprehensive assessment lasting approximately 5 hours at the 24 month assessment. The CPT was the second measure of a fixed battery administered to the children. At 24 months, participants were taking the CPT for the first time. All children completed a parallel assessment at the 36-month time point.

Statistical Analyses

ANOVA and chi-square tests were conducted examining group differences across a range of demographic variables. To test for the effects of time on neuropsychological performance and behavior in the LNCG and ADHD groups, mixed effects models were conducted using SAS PROC MIXED. Group status (ADHD vs. LNCG) and time (24 and 36 months) were examined as between- and within-subjects variables, respectively. Neuropsychological outcomes included the four CPT outcome variables (i.e., mean hit RT, RT standard deviation, percent omission errors, percent commission errors). Behavioral outcomes included the three ADHD symptom domains as measured by the parent and teacher SNAP-IV scale (i.e., inattention, hyperactivity, impulsivity). In order to include both parent and teacher ratings in our models, a rater variable was included in the statistical models. The dichotomous rater variable indicated whether ratings were completed by parents or teachers.

Initial mixed effects models included main effects for Group, Time, and a Group X Time interaction term. When interaction effects were not significant, the model was re-run without the interaction term. Given documented effects of medication on CPT performance in this sample (Epstein et al., 2006), for all analyses involving CPT outcomes a dichotomous variable indicating whether the child took ADHD medication on the day of testing was included as a covariate. For analyses involving ratings of ADHD symptomatology, the percent of days any stimulant medication was taken between the previous and current

SCAPI assessment was included as a covariate. Though there were group differences on IQ, we did not include IQ as a covariate in statistical modeling since ADHD-related IQ deficits are likely related to neuropsychological deficits and removing variance associated with IQ would likely attenuate between-group differences on our neuropsychological performance outcomes.

To examine correspondences between neuropsychological outcomes on a CPT task and ADHD symptom domains, Pearson correlations were computed for all CPT outcomes and the three ADHD symptom domains at 24-months and 36-months separately. To examine correspondence between *changes* in CPT outcomes and behavioral outcomes *over time*, mixed effect models were conducted examining the relationship between change (36-months minus 24-months) on the CPT variables (i.e., mean hit RT, RT standard deviation, percent omission errors, percent commission errors) and change on the behavioral variables (i.e., inattention, hyperactivity, and impulsivity). Change on both medication variables (i.e., medication on day of testing and percent days on medication since last assessment) was included as covariates in the analyses. Further, a rater variable was included as a repeated measure to allow for both parent and teacher ratings to be included in the model. Beta coefficients were standardized by dividing their standard deviation by the standard deviation of the respective outcome variable. Standardized beta coefficients were examined as a measure of the variance explained by the predictor considered in the outcome variable. Statistically significant estimates between CPT variable change and behavioral variable change would indicate covariation in neuropsychological and behavioral course. Power analyses indicated the study was sufficiently powered and able to detect correlation effects of .14 or greater.

Results

Mixed effect models for CPT outcomes revealed no significant Group X Time interaction effects. A constrained model omitting the interaction terms revealed significant main effects for medication and time across all four CPT outcome variables (Table 2). Significant main effects of Group were observed for RT standard deviation, errors of omission, and errors of commission. Children in the ADHD group demonstrated significantly greater variability in responding and committed significantly more errors of omission and commission than LNCG children. No group differences were observed for mean RT. Across all four CPT variables there were significant main effects of Time indicating overall improvement with time (i.e., decreasing mean RT, RT standard deviation, errors of omission, and errors of commission). Effects for medication indicated significantly better performance for children receiving medication on the day of testing.

To assess whether children in the ADHD group who no longer met ADHD diagnostic criteria were affecting the outcomes, we re-ran the constrained models including only children meeting diagnostic criteria at either time point. The pattern of effects was the same as in the original model. Also, to assess whether the presence of comorbid disruptive behavior disorders might be contributing to the observed pattern of findings (i.e., Drechsler et al., 2005), we re-ran the constrained models controlling for comorbid diagnoses of ODD or CD by including ODD/CD status as a covariate in the model. Again, the pattern of findings remained the same.

A parallel set of mixed effect models for parent and teacher ratings of inattention, hyperactivity, and impulsivity on the SNAP again revealed no Group X Time interaction effects. A constrained model omitting the interaction terms revealed significant main effects for Group across all three ADHD symptom domains. As expected, children in the ADHD group had higher ADHD symptom ratings than LNCG children. There were significant main

effects of Time for both Hyperactivity and Impulsivity symptoms with both domains declining over time. No main effect of Time was noted for ratings of Inattention (Table 3). No effects for medication were found.

A series of correlational analyses were examined between ratings of ADHD symptomatology and CPT outcomes. Correlations were similar at each time point. No significant correlations were found in relation to mean reaction time at either time point. RT standard deviation was significantly correlated with all three symptom domains across raters at both 24 and 36 months (Tables 4 and 5). Likewise, commission errors were significantly associated with teacher ratings of inattention, hyperactivity, and impulsivity at 24 and 36 months as well as parent rated impulsivity at 24 months. Note that while these correlations reached statistical significance, the magnitude of the correlations were relatively small.

A final set of mixed effect models were conducted to examine correspondence between time-related changes in CPT outcomes and ADHD symptomatology controlling for medication and rater effects over time. No significant effects were found indicating that the observed changes in neuropsychological and behavioral performance over time were unrelated (Table 6). Additional models including an Age × Time × Group interaction were conducted to assess whether age of the children had a moderating effect on the Time × Group interaction. The three-way interaction was not significant across models (all ps > .05). Additional models were conducted utilizing the ADHD group including only children receiving a persistent diagnosis of ADHD in the ADHD group. In these models, again no significant relations were found between CPT outcomes and ADHD symptomatology over time (all ps > .05). Finally, to examine whether medication effects among children in the ADHD group were affecting the observed pattern of results, models were run with children receiving medication and with those not receiving medication at 24 or 36 months. No significant relations between any of the CPT and behavioral variables were found across models using either of these restricted samples (all ps > .05).

Discussion

The primary aims of the current study were to examine the trajectory of neuropsychological functioning and ADHD symptomatology in children with and without a history of ADHD over a 1 year period as well as to examine correspondence between changes among different aspects of CPT performance and ADHD symptom domains over the same period. As hypothesized, both children with ADHD and normal controls demonstrated significant improvements in CPT performance over a 1 year period. However, despite improvement over time, children with ADHD continued to be significantly more variable in responding and committed significantly more errors of omission and commission than children without ADHD. Analyses also indicated that group differences in commission errors over time The presence and magnitude of neuropsychological deficits observed in children with ADHD, although improved, remained relatively constant across the 1 year time period. In addition, the absence of any Group X Time interaction suggests that rates of neuropsychological development were similar across ADHD and normal controls.

Consistent with our hypothesis and previous literature (Biederman et al., 2000; Fischer, Barkley, Fletcher, & Smallish, 1993; Hart et al., 1995; Langberg et al., 2008), children with ADHD exhibited significantly greater rates of ADHD symptomatology than comparison children despite parent and teacher rated hyperactive/impulsive symptomatology decreasing over time across groups. Symptoms of inattention were relatively stable over time across groups. The absence of a Group X Time interaction for any ADHD symptom domain suggests that ADHD symptoms demonstrate similar trajectories among children with ADHD and normal controls.

Finally, we found significant, though relatively small correlations, between specific CPT outcomes and ADHD symptom domains at the 24- and 36-month assessment points. Namely, reaction time variability was correlated with all three ADHD symptom domains consistent with previous research (e.g., Epstein et al., 2003). However, when we examined change in ADHD symptom domains and change in neuropsychological performance over time, there were no observed relations. This finding may have theoretical implications. Namely, Halperin and Schulz (Halperin & Schulz, 2006) recently proposed a neurodevelopmental model of ADHD suggesting that ADHD is a result of sub-cortical neurological dysfunction that persists across development. Development of the frontal cortex, however, serves a mediational role through helping patients with ADHD to compensate for cognitive and behavioral deficits caused by the sub-cortical dysfunction. Thus, observed improvements in ADHD symptomatology over time are hypothesized to be the result of prefrontal cortex development. According to this model, improvement in frontally-mediated neuropsychological functioning should parallel ADHD symptom attenuation in children with ADHD (Halperin & Schulz, 2006). In the context of the present study, this model would predict that CPT outcomes linked to frontal lobe functionality should decrease over time concomitantly with ADHD symptoms; whereas, CPT outcomes linked to sub-cortical functioning should remain relatively constant and unrelated to ADHD symptomatology. Halperin and Schulz (2006) suggest errors on an inhibition task as an outcome reflecting frontally-mediated effortful processing and reaction time variability as a measure of less conscious control likely mediated by sub-cortical processing. Accordingly, ADHD-related deficits on commission errors should have decreased over time while ADHD-related deficits on RT variability should maintain. Our results did reveal a large main effect for Group on RT variability with no Group X Time interaction suggesting overall discrepancies in RT variability across ADHD and matched normal comparison children. This is consistent with Halperin and Schulz's (2006) hypotheses. However, this same pattern of results also was observed for commission errors where their model would predict that this discrepancy would decrease over time (i.e., significant Group X Time interaction effect).

Similar investigations examining the associations between neuropsychological performance and ADHD over longer periods utilizing dichotomous indicators of ADHD have resulted in mixed findings. For example, using the identical CPT task, Hinshaw and colleagues (Hinshaw et al., 2007) found that commission errors were not related to ADHD diagnostic status over a 5 year period. That is, persisters and remitters performed comparably on this outcome at follow-up. Conversely, Halperin and colleagues (Halperin et al., 2008) found that children with a "persistent" diagnosis of ADHD were reliably differentiated from controls on commission errors on an identical pairs CPT task while ADHD remitters were not distinguishable from control children on this outcome. In other words, a measure of effortful processing, commission errors, was related to the trajectory of ADHD symptomatology consistent with Halperin and Shultz's (2006) predictions.

In summary, our findings do not appear to support the hypothesized linkages between changes in frontally-mediated EF functioning and ADHD symptomatology. There are a couple of alternative explanations regarding this lack of support for our study hypotheses. First, it may be due to our study design, as despite a relatively large sample, our study was limited to two time points with a 1 year lag in children ranging from 9–14 years of age. Thus, we captured a relatively narrow range of development. Power to detect associations between developmental neuropsychological and symptom trajectories may be dependent upon following children over a longer period of time. Development of the frontal cortex, especially related to executive functioning processes, is among the last of the brain regions to fully develop with typical maturation occurring in late adolescence into young adulthood (Giedd et al., 1996; Gogtay et al., 2004). Hence, our study may have missed the period of

brain and cognitive development when the type of changes proposed by Halperin and Schulz (2006) occur. For example, past research examining a broader range of development (e.g., 10 years) found differences in effortful control process between individuals with persistent versus remittent ADHD symptomatology (Halperin et al., 2008); whereas, studies examining shorter periods have failed to find similar effects (Hinshaw et al., 2007). However, failure to find effects was unlikely due to lack of power in our study. Power analyses indicated that was sufficiently powered to detect significant effects. Second, our analyses of neuropsychological functioning were limited to outcomes on a go/no-go CPT task. Although similar go/no-go tasks have been utilized in numerous studies examining ADHD neuropsychological functioning, they provide limited information related to executive functioning by focusing primarily on response inhibition. A broader battery of measures examining other areas of executive functioning (e.g., working memory, planning, set maintenance and set shifting, etc.) may have provided a different set of relations between EF and ADHD symptoms.

Other limitations to our study must also be considered. As both groups improved similarly over time on a go/no-go task, it is possible practice effects may have contributed to these findings as opposed to purely developmental effects as interpreted. Should practice effects solely drive observed improvement, we would not expect to find correlations among CPT outcomes and ADHD symptomatology (which are developmentally driven) over time. However, past research has found practice effects limited to mean reaction time on a similar task in unreferred children (Halperin, Sharma, Greenblatt, & Schwartz, 1991). Another study limitation is that children with a diagnosis of ADHD, Predominantly Inattentive Type were not included in the MTA study. Given controversies in the literature regarding etiology, core deficits, associated features, and comorbid functioning of children diagnosed with ADHD-I (Milich, Balentine, & Lynam, 2001), future research should examine subtype differences in relation to developmental trajectories.

This study provides insights into the longitudinal course of ADHD-related neuropsychological deficits in relation to corresponding developmental trajectories of ADHD symptomatology. Our findings indicate that despite improvements in neuropsychological performance and ADHD-related behavior, children with ADHD continue to demonstrate impaired functioning relative to control children. More importantly, this was the first study to examine directly the relation among the developmental trajectories of neuropsychological functioning and ADHD symptom domains. Clinically, the persistence of these deficits highlights the need for continued interventions through childhood and into adolescence for children with ADHD. As our findings provide support regarding the persistence of both ADHD symptomatology and deficits in neuropsychological functioning during this developmental period, interventions should seek to understand and account for the impact these deficits are likely to have in relation to impairments experienced by children and adolescents with ADHD. Importantly, this period is associated with the transition into middle school which research has associated with greater academic impairment for children with ADHD (Langberg et al., 2008). Our findings also highlight the need for research examining not only the longitudinal course of neuropsychological functioning or ADHD symptomatology independently, but proposed relations among their developmental trajectories. We suggest that future studies include more frequent assessments over a longer period of time to fully map the likely non-linear developmental trajectories. Further, it would be beneficial to include longitudinal measures of brain morphology and/or brain functioning in order to examine the role of ADHD neural development on cognitive and behavioral trajectories.

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References

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: Author; 2000. text rev

Arnold L, Abikoff H, Cantwell D, Conners C, Elliott G, Greenhill L, et al. National Institute of Mental Health Collaborative Multimodal Treatment Study of Children with ADHD. Design challenges and choices. Archives of General Psychiatry. 1997; 54:865–870. [PubMed: 9294378]

Barkley RA. Behavorial inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. Psychological Bulletin. 1997; 121:65–94. [PubMed: 9000892]

Barkley RA, Fischer M, Edelbrock CS, Smallish L. The adolescent outcome of hyperactive children diagnosed by research criteria: I. An 8-year prospective follow-up study. Journal of the American Academy of Child and Adolescent Psychiatry. 1990; 29:546–557. [PubMed: 2387789]

Barkley RA, Grodzinsky G, DuPaul GJ. Frontal lobe functions in attention deficit disorder with and without hyperactivity: A review and research report. Journal of Abnormal Child Psychology. 1992; 20:163–188. [PubMed: 1593025]

Barnea-Goraly N, Menon V, Eckert M, Tamm L, Bammer R, Karchemskiy A, et al. White matter development during childhood and adolescence: a cross-sectional diffusion tensor imaging study. Cerebral Cortex. 2005; 15:1848–1854. [PubMed: 15758200]

Biederman J, Faraone S, Milberger S, Curtis S, Chen L, Marrs A, et al. Predictors of persistence and remission of ADHD into adolescence: results from a four-year prospective follow-up study. Journal of the American Academy of Child and Adolescent Psychiatry. 1996; 35:343–351. [PubMed: 8714323]

Biederman J, Faraone S, Taylor A, Sienna M, Williamson S, Fine C. Diagnostic continuity between child and adolescent ADHD: findings from a longitudinal clinical sample. Journal of the American Academy of Child and Adolescent Psychiatry. 1998; 37:305–313. [PubMed: 9519636]

Biederman J, Mick E, Faraone SV. Age-dependent decline of symptoms of attention deficit hyperactivity disorder: Impact of remission definition and symptom type. American Journal of Psychiatry. 2000; 157:816–818. [PubMed: 10784477]

Biederman J, Monuteaux MC, Mick E, Spencer T, Wilens TE, Silva JM, et al. Young adult outcome of attention deficit hyperactivity disorder: a controlled 10-year follow-up study. Psychological Medicine. 2006; 36:167–179. [PubMed: 16420713]

Biederman J, Petty C, Doyle AE, Spencer T, Henderson CS, Marion B, et al. Stability of executive function deficits in girls with ADHD: a prospective longitudinal followup study into adolescence. Developmental Neuropsychology. 2008; 33:44–61. [PubMed: 18443969]

Biederman J, Petty CR, Fried R, Doyle A, Spencer T, Seidman LJ, et al. Stability of executive function deficits into young adult years: a prospective longitudinal follow-up study of grown up males with ADHD. Acta Psychiatrica Scandinavica. 2007; 116:129–136. [PubMed: 17650275]

Carr LA, Nigg JT, Henderson JM. Attentional versus motor inhibition in adults with attention-deficit/hyperactivity disorder. Neuropsychology. 2006; 20:430–441. [PubMed: 16846261]

Castellanos FX, Lee PL, Sharp W, Jeffries NO, Greenstein DK, Clasen LS, et al. Developmental brain trajectories of brain volume abnormalities in children and adolescents with ADHD. Journal of the American Medical Association. 2002; 288:1740–1748. [PubMed: 12365958]

Conners, CK. The Conners Continuous Performance Test. Toronto, Canada: Multi- Health Systems, Inc; 1994.

Drechsler R, Brandeis D, Foldenyi M, Imhof K, Steinhausen HC. The course of neuropsychological functions in children with attention deficit hyperactivity disorder from late childhood to early adolescence. Journal of Child Psychology and Psychiatry and Allied Disciplines. 2005; 46:824–836.

Epstein JN, Conners CK, Hervey AS, Tonev ST, Arnold LE, Abikoff HB, et al. Assessing medication effects in the MTA study using neuropsychological outcomes. Journal of Child Psychology and Psychiatry and Allied Disciplines. 2006; 47(5):446–456.

Epstein JN, Erkanli A, Conners CK, Klaric J, Costello JE, Angold A. Relations between continuous performance test performance measures and ADHD behaviors. Journal of Abnormal Child Psychology. 2003; 31:543–554. [PubMed: 14561061]

Faraone SV, Biederman J. Neurobiology of attention-deficit hyperactivity disorder. Biological Psychiatry. 1998; 44:951–958. [PubMed: 9821559]

Fischer M, Barkley RA, Fletcher K, Smallish L. The adolescent outcome of hyperactive chldren diagnosed by research criteria: V. Predictors of outcome. Journal of the American Academy of Child and Adolescent Psychiatry. 1993; 32:324–332. [PubMed: 8444761]

Fischer M, Barkley RA, Smallish L, Fletcher K. Executive functioning in hyperactive children as young adults: attention, inhibition, response perseveration, and the impact of comorbidity. Developmental Neuropsychology. 2005a; 27:107–133. [PubMed: 15737944]

Giedd JN, Snell JW, Lange N, Rajapakse JC, Casey BJ, Kaysen D, et al. Quantitative magnetic resonance imaging of human brain development: ages 4–18. Cerebral Cortex. 1996; 6:551–560. [PubMed: 8670681]

Gogtay N, Giedd JN, Lusk L, Hayashi KM, Greensteing D, Vaituzis AC, et al. Dynamic mapping of human cortical development during childhood through early adulthood. Proceedings of the National Academy of Science. 2004; 101:8174–8179.

Halperin J, Schulz K. Revisiting the role of the prefrontal cortex in the pathophysiology of attention-deficit/hyperactivity disorder. Psychological Bulletin. 2006; 132:560–581. [PubMed: 16822167]

Halperin JM, Sharma V, Greenblatt E, Schwartz ST. Assessment of the Continuous Performance Test: Reliability and validity in a nonreferred sample. Psychological Assessment. 1991; 3:603–608.

Halperin JM, Trampush JW, Miller CJ, Marks DJ, Newcorn JH. Neuropsychological outcome in adolescents/young adults with childhood ADHD: profiles of persisters, remitters and controls. Journal of Child Psychology and Psychiatry and Allied Disciplines. 2008; 49:958–966.

Hart EL, Lahey BB, Loeber R, Applegate B, Frick PJ. Developmental changes in attention–deficit hyperactivity disorder in boys: A four–year longitudinal study. Journal of Abnormal Child Psychology. 1995; 23:729–750. [PubMed: 8609310]

Hervey AS, Epstein JN, Curry JF. The neuropsychology of adults with Attention Deficit Hyperactivity Disorder: A meta-analytic review. Neuropsychology. 2004; 18:485–503. [PubMed: 15291727]

Hervey AS, Epstein JN, Curry JF, Tonev S, Arnold LE, Conners CK, et al. Reaction time distribution analysis of neuropsychological performance in an ADHD sample. Child Neuropsychology. 2006; 12:125–140. [PubMed: 16754533]

Hill J, Schoener E. Age-dependent decline of attention deficit hyperactivity disorder. American Journal of Psychiatry. 1996; 153:1143–1146. [PubMed: 8780416]

Hinshaw S, Carte ET, Fan C, Jassy JS, Owens EB. Neuropsychological functioning of girls with attention-deficit/hyperactivity disorder followed prospectively into adolescence: evidence for continuing deficits? Neuropsychology. 2007; 21:263–273. [PubMed: 17402826]

Hinshaw S, March J, Abikoff H, Arnold L, Cantwell D, Conners C, et al. Comprehensive assessment of childhood attention-deficit hyperactivity disorder in the context of a multisite, multimodal clinical trial. Journal of Attention Disorders. 1997; 1:217–234.

Hinshaw S, Owens EB, Sami N, Fargeon S. Prospective follow-up of girls with attention-deficit/hyperactivity disorder into adolescence: Evidence for continuing cross-domain impairment. Journal of Consulting and Clinical Psychology. 2006; 74:489–499. [PubMed: 16822106]

Hoagwood KE, Jensen PS, Arnold LE, Roper M, Severe J, Odbert C, et al. Reliability of the services for children and adolescents-parent interview. Journal of the American Academy of Child and Adolescent Psychiatry. 2004; 43:1345–1354. [PubMed: 15502593]

Jensen PS, Hoagwood K, Roper M, Arnold LE, Odbert C, Crowe M, et al. The services for children and adolescents-parent interview: development and performance characteristics. Journal of the American Academy of Child and Adolescent Psychiatry. 2004; 43:1334–1344. [PubMed: 15502592]

Krain AL, Castellanos FX. Brain development and ADHD. Clinical Psychology Review. 2006; 26:433–444. [PubMed: 16480802]

Langberg JM, Epstein JN, Altaye M, Molina B, Arnold LE, Vitiello B. The transition to middle school is associated with changes in the developmental trajectory of ADHD symptomatology in young adolescents with ADHD. Journal of Clinical Child and Adolescent Psychology. 2008; 37:651–663. [PubMed: 18645755]

Larsson H, Lichtenstein P, Larsson J. Genetic Contributions to the Development of ADHD subtypes from childhood to adolescence. Journal of the American Academy of Child and Adolescent Psychiatry. 2006; 45:973–981. [PubMed: 16865040]

Larsson J, Larsson H, Lichtenstein P. Genetic and environmental contributions to stability and change of ADHD symptoms between 8 and 13 years of age: A longitudinal twin study. Journal of the American Academy of Child and Adolescent Psychiatry. 2004; 43:1267–1275. [PubMed: 15381894]

Martinussen R, Hayden J, Hogg-Johnson S, Tannock R. A meta-analysis of working memory impairments in children with attention-deficit/hyperactivity disorder. Journal of the American Academy of Child and Adolescent Psychiatry. 2005; 44:377–384. [PubMed: 15782085]

Milich R, Balentine AC, Lynam DR. ADHD combined type and ADHD predominantly inattentive type are distinct and unrelated disorders. Clinical Psychology-Science and Practice. 2001; 8(4): 463–488.

Molina B, Hinshaw S, Swanson J, Arnold L, Vitiello B, Jensen P, et al. The MTA at 8 years: prospective follow-up of children treated for combined-type ADHD in a multisite study. Journal of the American Academy of Child and Adolescent Psychiatry. 2009; 48:484–500. [PubMed: 19318991]

MTA Cooperative Group. A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. The MTA Cooperative Group. Multimodal Treatment Study of Children with ADHD. Arcives of General Psychiatry. 1999; 56:1073–1086.

Nigg JT. Neuropsychologic theory and findings in attention-deficit/hyperactivity disorder: The state of the field and salient challenges for the coming decade. Biological Psychiatry. 2005; 57:1424–1435. [PubMed: 15950017]

Nigg JT, Blaskey LG, Huang-Pollock CL, Rappley MD. Neuropsychological executive functions and DSM-IV ADHD subtypes. Journal of the American Academy of Child and Adolescent Psychiatry. 2002; 41:59–66. [PubMed: 11800208]

Nigg JT, Butler KM, Huang-Pollock CL, Henderson JM. Inhibitory processes in adults with persistent childhood onset ADHD. Journal of Consulting and Clinical Psychology. 2002; 70:153–157. [PubMed: 11860041]

Nigg JT, Willcutt EG, Doyle AE, Sonuga-Barke EJ. Causal heterogeneity in attention-deficit/hyperactivity disorder: do we need neuropsychologically impaired subtypes? Biological Psychiatry. 2005; 57:1224–1230. [PubMed: 15949992]

Seidman LJ. Neuropsychological functioning in people with ADHD across the lifespan. Clinical Psychology Review. 2006; 26:466–485. [PubMed: 16473440]

Seidman LJ, Benedict KB, Biederman J, Bernstein JH, Seiverd K, Milberger S, et al. Performance of children with ADHD on the Rey-Osterrieth complex figure: a pilot neuropsychological study. Journal of Child Psychology and Psychiatry and Allied Disciplines. 1995; 36:1459–1473.

Seidman LJ, Biederman J, Faraone SV, Milberger S, Norman D, Seiverd K, et al. Effects of family history and comorbidity on the neuropsychological performance of children with ADHD: preliminary findings. Journal of the American Academy of Child and Adolescent Psychiatry. 1995; 34:1015–1024. [PubMed: 7665440]

Seidman LJ, Biederman J, Faraone SV, Weber W, Ouellette C. Toward defining a neuropsychology of attention deficit-hyperactivity disorder: Performance of children and adolescents from a large clinically referred sample. Journal of Consulting and Clinical Psychology. 1997; 65:150–160. [PubMed: 9103744]

Seidman LJ, Biederman J, Weber W, Hatch M, Faraone SV. Neuropsychological functioning in adults with Attention-Deficit Hyperactivity Disorder. Biological Psychiatry. 1998; 44:260–268. [PubMed: 9715357]

Shaw P, Eckstrand K, Sharp W, Blumenthal J, Lerch JP, Greenstein D, et al. Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. Proceedings of the National Academy of Sciences of the United States of America. 2007; 104:19649–19654. [PubMed: 18024590]

Swanson, JM. School Based Assessments and Interventions for ADD Students. Irvine, CA: K.C; 1992.

Willcutt EG, Doyle AE, Nigg JT, Faraone SV, Pennington BF. Validity of the executive function theory of Attention-Deficit/Hyperactivity Disorder: A meta-analytic review. Biological Psychiatry. 2005; 57:1336–1346. [PubMed: 15950006]

Wolraich ML, Lambert W, Doffing MA, Bickman L, Simmons T, Worley K. Psychometric properties of the Vanderbilt ADHD diagnostic parent rating scale in a referred population. Journal of Pediatric Psychology. 2003; 28(8):559–567. [PubMed: 14602846]

Table 1

Demographic Characteristics for the ADHD and Control Groups

	ADHD 24 months	LNCG 24 months	ADHD 36 months	LNCG 36 months
Age (mean, *sd*)	10.4	10.5	11.5	11.6
	.85	*1.08*	*1.18*	*.92*
IQ (mean, *sd*)	101.01[1]	108.41		
	14.61	*19.19*		
Ethnicity (percentage Caucasian)	60.8	62.8		
Sex (percentage male)	80.3	81.0		
Primary Reporter (percentage mothers)	86.4	94.8		
Family Income (percentage earning < 40,0000)	35.3	22.7		
Percentage of each ADHD subtype				
No ADHD Diagnosis	50.6	100.0	47.5	100.0
Inattentive	18.4	0.0	22.1	0.0
Hyperactive/Impulsive	6.7	0.0	7.1	0.0
Combined	23.9	0.0	23.0	0.0
Percentage with specified psychological disorder				
Any Anxiety Disorder	14.2	11.1	14.8	8.1
Any Elimination Disorder	7.3	4.5	6.0	2.9
Tic	4.8	1.0	4.6	1.1
Any Mood Disorder	2.3	.7	2.1	.7
Any Eating Disorder	.4	.3	.6	.4
Oppositional Defiant Disorder/Conduct Disorder	22.1	4.8	25.4	2.9

[1] Cognitive testing was completed at baseline for the ADHD group.

Note: LNCG = Local, Normative Comparison Group; Any Anxiety Disorder includes: Simple Phobia, Social Phobia, Agoraphobia, Panic Disorder, Overanxious Disorder, Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, and Separation Anxiety Disorder; Any Mood Disorder includes: Major Depression, Dysthmia, Mania, and Hypomania; Any Elimination Disorder includes: Enuresis (primary and secondary) and Encopresis.

Table 2

Performance on CPT Measures by Group at Baseline and Follow-up

	ADHD		LNCG		ES for Time	ES for Group	ES for Group		Test of Fixed Effects for Medication	Test of Fixed Effects for Group	Test of Fixed Effects for Time
	Time 1 (n=375)	Time 2 (n=413)	Time 1 (n=220)	Time 2 (n=212)		Time 1	Time 2	df	*F*	*F*	*F*
	M (SD)	*M (SD)*	*M (SD)*	*M (SD)*							
Mean Reaction Time	440.04 (102.84)	416.17 (78.97)	428.40 (130.93)	405.98 (81.15)	.20	.10	.15	703	15.57***	2.75	23.62***
Reaction Time Standard Deviation	282.01 (143.12)	265.69 (122.51)	211.07 (123.75	180.79 (91.76)	.16	.60	.73	702	80.55***	75.42***	14.15***
Omission Errors	39.87 (50.61)	33.58 (43.93)	25.69 (47.36)	20.50 (50.75)	.09	.28	.30	703	27.54***	15.85***	5.27*
Commission Errors	30.42 (51.66)	25.39 (44.40)	25.24 (47.34)	20.19 (50.74)	.32	.23	.21	703	8.65**	7.47**	55.88***

Note: ES = Effect size; LNCG = Local, Normative Comparison Group

* $p \leq .05$;

** $p \leq .01$;

*** $p \leq .001$

Table 3

Parent and Teacher Mean Ratings by Subscale of Inattention, Hyperactivity, and Impulsivity Symptoms on SNAP-IV by Group at Baseline and Follow-up

	ADHD		LNCG		ES for Time	ES for Group	ES for Group		Test of Fixed Effects for Medication	Test of Fixed Effects for Group	Test of Fixed Effects for Time
	Time 1 (n=534)	Time 2 (n=486)	Time 1 (n=254)	Time 2 (n=239)		Time 1	Time 2	df	*F*	*F*	*F*
	M (SD)	*M (SD)*	*M (SD)*	*M (SD)*							
Inattention					.002	1.59	1.60	779	1.67	444.56***	.01
Parent	1.30 (.70)	1.36 (.72)	.46 (.46)	.39 (.40)							
Teacher	1.57 (.84)	1.51 (.80)	.69 (.69)	.65 (.70)							
Hyperactivity					.15	1.30	1.33	779	1.92	324.87***	20.46***
Parent	.97 (.72)	.89 (.71)	.24 (.33)	.16 (.30)							
Teacher	1.05 (.80)	.91 (.75)	.38 (.53)	.33 (.52)							
Impulsivity					.10	1.14	1.19	778	.10	273.99***	10.22**
Parent	1.21 (.81)	1.18 (.81)	.37 (.46)	.25 (.40)							
Teacher	1.16 (.89)	1.04 (.85)	.44 (.66)	.33 (.56)							

Note: ES = Effect size (Cohen's *d*); LNCG = Local, Normative Comparison Group

* p ≤ .05;

** p ≤ .01;

*** p ≤ .001

Table 4

Correlations between Mean Parent and Teacher Ratings of Inattention, Hyperactivity, and Impulsivity on the SNAP-IV and Outcomes on CPT Task at 24 months.

	Mean Reaction Time	Reaction Time Standard Deviation	Omission Errors	Commission Errors
Parent				
Inattention	−.047	.086*	.034	.034
Hyperactivity	.011	.132**	.094*	.002
Impulsivity	−.015	.099*	.122**	.009
Teacher				
Inattention	−.007	.206**	.044	.093*
Hyperactivity	−.041	.192**	.039	.089*
Impulsivity	−.027	.157**	.037	.108*

* p ≤ .05

** p ≤ .01

*** p ≤ .001

Table 5

Correlations between Mean Parent and Teacher Ratings of Inattention, Hyperactivity, and Impulsivity on the SNAP-IV and Outcomes on CPT Task at 36 months.

	Mean Reaction Time	Reaction Time Standard Deviation	Omission Errors	Commission Errors
Parent				
Inattention	−.032	.123**	.048	.096*
Hyperactivity	.022	.174**	.061	.097*
Impulsivity	.004	.141**	.084*	.061
Teacher				
Inattention	.002	.229**	.130**	.200**
Hyperactivity	.005	.222**	.136**	.128**
Impulsivity	−.008	.175**	.087*	.102*

* p ≤ .05

** p ≤ .01

*** p ≤ .001

Table 6

Mixed effect model standardized beta coefficients examining correspondence between time-related changes in neuropsychological performance and parent and teacher- rated ADHD symptomatology

	Inattention	Hyperactivity	Impulsivity
Mean Reaction Time	−.054	−.009	−.044
Reaction Time Standard Deviation	−.036	.006	−.006
Omission Errors	−.047	−.002	−.027
Commission Errors	−.042	−.101	.003

Note: All coefficients (F values) are non-significant (p >.05).