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# Neuropsychological outcome in adolescents/young adults with childhood ADHD: profiles of persisters, remitters and controls

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**Background:** This study examined neuropsychological functioning in a longitudinal sample of adolescents/young adults with attention deficit/hyperactivity disorder (ADHD) and controls as a function of the persistence of ADHD. We hypothesized that measures of executive processes would parallel adolescent clinical status, with ADHD-persisters, but not remitters, differing significantly from controls. In contrast, persisters and remitters were hypothesized to perform similarly, and different from controls, on tasks requiring less effortful processing. **Methods:** Ninety-eight participants diagnosed with ADHD in childhood were reevaluated approximately 10 years later. Eighty-five never-ADHD controls similar in age, IQ, and sex distribution served as a comparison group. Participants were administered a psychiatric interview and neuropsychological test battery. **Results:** Those with childhood ADHD demonstrated broad neuropsychological deficits relative to controls. When the group with childhood ADHD was subdivided based on adolescent ADHD status, compared to controls, both persisters and remitters showed deficient perceptual sensitivity and response variability, and increased ankle movements recorded by a solid-state actigraph. Only persisters differed from controls on several measures of more effortful executive processes. **Conclusions:** Findings provide preliminary support to the hypothesis that ADHD is associated with early-appearing and enduring subcortical dysfunction, while recovery over the course of development is associated with improvements in executive control functions. **Keywords:** Activity level, ADD/ADHD, adolescence, executive function, longitudinal studies, neuropsychology.

The behavioral phenotype of attention-deficit/hyperactivity disorder (ADHD) changes in characteristic ways, with a tendency for symptom improvement and at least partial recovery throughout development. Studies of children with ADHD followed into adolescence and adulthood indicate that 20–50% improve with regard to ADHD symptoms and no longer meet criteria for the disorder (Barkley, Fischer, Edelbrock, & Smallish, 1990; Biederman, Mick, & Faraone, 2000; Hill & Schoener, 1996). In addition, the nature of the symptoms changes over development; hyperactivity-impulsivity is most apparent during the younger ages, whereas inattention and related dysfunction are most prominent during later developmental stages (Hart, Lahey, Loeber, Applegate, & Frick, 1995).

Consistent with the behavioral heterogeneity in ADHD is the wide range of neurocognitive heterogeneity (Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005b). Despite the common conceptualization of ADHD as a disorder of executive functions (EFs) subserved by frontal-subcortical neural systems (Barkley, 1997), recent meta-analyses (Frazier, Demaree, & Youngstrom, 2004; van Mourik, Oosterlaan, & Sergeant, 2005; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005) indicate an array of executive and non-executive function deficits in children with ADHD. Furthermore, increased variability in reaction time (RT) is rapidly emerging as one of the most consistent deficits in children with ADHD (Castellanos & Tannock, 2002; Russell et al., 2006). Additional presumably 'non-executive' parameters have been shown to differentiate ADHD from non-ADHD children, including signal detectability ($d'$) and response bias ($\ln\beta$) variables from continuous performance tests (CPTs; Losier, McGrath, & Klein, 1996). Kuntsi and colleagues (2001) reported that RT variability discriminated ADHD children from controls better than measures of inhibition and working memory, and Epstein and colleagues (2003) reported variability in $d'$, $\ln\beta$, and RT to be strongly associated across multiple ADHD symptom domains. As such, it has been hypothesized that the EF deficits frequently observed in children with ADHD may be due to deficiencies in largely subcortical, regulatory systems, rather than cortical EF circuitry per se (Douglas, 1999; Rommelse et al., 2007; Sergeant, Oosterlaan, & van der Meere, 1999; Sonuga-Barke & Castellanos, 2007).

A smaller literature has examined neuropsychological deficits in adults with ADHD (Nigg et al., 2005a; Seidman, 2006) with findings generally

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indicating impairments similar to those in children. A recent meta-analysis (Hervey, Epstein, & Curry, 2004) reported that, compared to controls, adults with ADHD performed worse across multiple neuropsychological domains. Consistent impairments on various CPT paradigms were reported, whereas more traditional EF tests such as the Stroop, Wisconsin Card Sorting Test (WCST), and Trail-Making Test only differentiated the groups moderately well, if at all (Hervey et al., 2004; Nigg et al., 2005a; Seidman, 2006).

While it is important to determine which neurocognitive deficits are present in adults with persistent ADHD, it is equally important to understand the degree to which improvement in specific neurocognitive functions is associated with recovery. Carr, Nigg and Henderson (2006) reasoned that core deficits of ADHD should persist in adults who no longer meet criteria for the disorder, but more epiphenomenal characteristics should parallel symptom recovery. They compared adults with ADHD, adults with retrospectively assessed childhood histories of ADHD but partial recovery, and controls using an antisaccade task which evaluated distinct forms of inhibition. Directional errors on the task fit the pattern of epiphenomenal symptoms in that the ADHD group, but not the partially remitted group, differed from controls. In contrast, anticipatory errors behaved more like a core deficit; those with childhood ADHD differed from controls irrespective of adult status. This approach provides insight into the developmental trajectory of neurocognitive functioning in ADHD and the dissociation of potentially causal versus secondary deficits. Yet, retrospective recall of childhood ADHD symptoms and impairment is often inaccurate (Mannuzza, Klein, Klein, Bessler, & Shrout, 2002). As such, these kinds of questions are best addressed in a longitudinal sample.

Drechsler et al. (2005) examined measures of alertness and inhibitory control in controls and children with ADHD (mean age = 11.0 at baseline) three times over 2.6 years. By the final assessment, there were no group differences on their measures. However, these findings are difficult to interpret because 61% of their ADHD group no longer met criteria for the disorder at the final follow-up assessment and data were not examined relative to persistence/remittance.

Other longitudinal studies of children with ADHD followed into adolescence and beyond have generally found that neuropsychological dysfunction is prominent throughout development (Fischer, Barkley, Edelbrock, & Smallish, 1990). Seidman and colleagues (1997) reported deficits on the Stroop, WCST, and an auditory CPT among high school and college-age participants diagnosed with ADHD in childhood. However, they did not examine whether task performance varied as a function of ADHD persistence. Fischer and colleagues (2005) examined neuropsychological outcomes of childhood ADHD relative to the presence or absence of ADHD at early adult follow-up. Those with persistent ADHD made significantly more omission and commission errors on a CPT than controls, while those with ADHD in childhood, but *not* adulthood (i.e., remitters), did not differ from either group on these measures. Persisters, remitters, and controls earned similar amounts on a card task designed to measure inhibitory control, although both ADHD groups performed the task slower than controls.

To explain developmental changes associated with ADHD across the lifespan, Halperin and Schulz (2006) posited distinct neurocognitive mechanisms for the etiology of and recovery from ADHD. They hypothesized that ADHD is caused by non-cortical neural dysfunction that is present early in ontogeny, remains relatively static throughout life, and is not associated with the reduction of symptoms typically seen over development. Rather, variations in the diminution of symptoms with increasing age are accounted for by the degree to which prefrontally mediated EFs, which emerge throughout childhood and adolescence, can compensate for more primary and enduring subcortical deficits. According to this model, performance of adults who had ADHD in childhood, on measures which reflect executive control, should closely parallel their present symptom severity. In contrast, measures of more automatic or less consciously controlled processes should be linked to the presence of ADHD in childhood, irrespective of later clinical status.

This study examined neuropsychological functioning in a longitudinal sample of adolescents/young adults who were diagnosed with ADHD in childhood as compared to a never-ADHD control group. We hypothesized that those with childhood ADHD would perform more poorly than controls on a diverse neuropsychological test battery. Further, we examined the degree to which performance on measures requiring high levels of executive processing would parallel adolescent clinical status, such that ADHD-persisters, but not ADHD-remitters would differ from controls. In contrast, persisters and remitters were hypothesized to perform more similarly, and different from controls, on measures that reflect behaviors that are unlikely to be under executive control.

## Methods

### *Participants*

Ninety-eight adolescents/young adults who were evaluated in a research protocol during childhood (Halperin et al., 1997) participated in a follow-up evaluation on average 9.30 (*SD* = 1.65) years later. They were drawn from a group of 169 youth who were recruited between 1990 and 1997. Of these, 18 refused participation, one was deceased, five were incarcerated, and 47 were lost to follow-up. Those who were and were not followed did not differ significantly in age at initial evaluation, race/

**Table 1** Baseline characteristics of ADHD groups followed and not followed

| | Followed ($n$ = 98) | | Lost to follow-up ($n$ = 71) | |
|---|---|---|---|---|
| *Measure* | Mean | *SD* | Mean | *SD* |
| Age (years) | 9.09 | 1.30 | 8.87 | 1.29 |
| WISC-R/WISC-III FSIQ | 93.96* | 14.27 | 89.31* | 14.95 |
| Child Behavior Checklist | | | | |
| Attention Problems | 72.15 | 10.05 | 73.20 | 8.30 |
| Delinquency | 67.99 | 9.65 | 65.88 | 8.27 |
| Aggression | 72.12 | 13.94 | 68.73 | 12.64 |
| IOWA Conners' Scale | | | | |
| Inattention/Overactivity | 11.23 | 3.20 | 10.73 | 3.04 |
| Aggression/Defiance | 8.16 | 4.73 | 8.16 | 4.52 |

*$p$ = .048.

ethnicity, sex, childhood comorbidity, socioeconomic status (SES), or ADHD behavior ratings at baseline.

In childhood (ages 7–11 years), participants were evaluated using parent report on the Diagnostic Interview Schedule for Children (DISC), version 2.1 or 2.3, depending upon when they were recruited (Fisher et al., 1993; Shaffer et al., 1996). Parent and teacher reports using the Child Behavior Checklist (Achenbach, 1991) and IOWA Conners Rating Scale (Loney & Milich, 1982), respectively, were also obtained. Childhood assessments determined using DSM-III-R were re-coded to be consistent with DSM-IV; it is likely that most, if not all, participants met criteria for ADHD-Combined Type (ADHD-C). Table 1 shows childhood characteristics of the ADHD sample divided into those who were and were not followed.

In addition, 85 never-ADHD controls were recruited during adolescence/young adulthood via advertisements in neighborhoods that matched the ADHD sample by zip code. Controls resembled probands on most important demographic variables including age, gender, ethnicity, SES, and IQ ($P$ > .05; see Table 2). Like the original ADHD sample, prospective controls were excluded if they had any chronic medical/neurological condition or psychosis, or were non-English-speaking.

The sample was predominately male (87.8%) and racially/ethnically diverse (26.0% African-American, 23.8% Caucasian, 35.4% Hispanic, and 14.4% mixed or other ancestry). Ages ranged from 16 to 21 years.[1] SES, estimated using the socioeconomic prestige scale (Nakao & Treas, 1994), was 42.57 (*SD* = 17.34). The sample comprised individuals with a broad range of scores on this measure (20–96), with a modal score of 20 ($n$ = 32, 17.7%), representing, on average, a low to lower-middle status group, with a fairly large number at the poverty level.

All procedures were approved by the Institutional Review Boards of the participating institutions. Written informed consent was obtained from participants above 18 years old and the parents of those under 18 years; assent was obtained from youth under 18 years old.

[1] Three participants in the ADHD group were 15.55, 25.50, and 26.29 years-old at follow-up.

### *Clinical assessment measures*

The Kiddie-SADS Present and Lifetime Version (Kaufman et al., 1997) was administered at follow-up to each participant and his/her parent to assess the presence of ADHD. Evaluators were PhD-level psychologists or trained psychology graduate students blind to group membership. Based on this interview, the ADHD group was subdivided into those who continued to meet diagnostic criteria for ADHD ('persisters' $n$ = 44) and those who did not ('remitters' $n$ = 29). Remitters were operationalized to provide adequate separation from persisters, with three or fewer symptoms of inattention *and* hyperactivity-impulsivity. Those with more than three symptoms in either domain, but fewer than six symptoms in one domain, were not included in analyses that focused on outcome status ($n$ = 25). Persistence was defined as meeting DSM-IV criteria for ADHD with at least six symptoms of inattention and/or hyperactivity-impulsivity, plus evidence of impairment across multiple settings. Although all subjects likely met criteria for ADHD-C in childhood, there was a distribution of subtypes at follow-up among the persisters: 31.8% ADHD-C, 47.7% ADHD-Inattentive Type, and 20.5% ADHD-Hyperactive-Impulsive Type. Severity of ADHD symptoms was determined by self- and parent-ratings on a 4-point rating scale made up of all 18 DSM-IV Inattentive and Hyperactive-Impulsive items. Figure 1 highlights the dimensional separation of groups based on self- and parent-report.

### *Neuropsychological measures*

*Wechsler Adult Intelligence Scale, Third Edition (WAIS-III).* The WAIS-III was administered to generate the four Index scores: Verbal Comprehension (VCI), Perceptual Organization (POI), Working Memory (WMI), and Processing Speed (PSI), which served as dependent variables to determine specificity of dysfunction. The WMI, which is of particular interest to ADHD, is comprised of the Digit Span, Arithmetic, and Letter-Number Sequencing subtests.

*Stroop Color-Word Test.* The Stroop (Golden, 1978) was administered to measure basic cognitive speed and interference control. Outcome measures were Word-Reading, Color-Naming, Color-Word, and Interference *T*-scores.

*CPT.* We administered a computerized identical pairs CPT which was sufficiently difficult to safeguard against ceiling effects oftentimes seen in young adults. Examinees monitored a series of 4-digit numbers and responded whenever the same 4-digit sequence appeared twice in succession. A total of 400 stimuli were presented for 200ms, with a 1500ms inter-stimulus interval and a 10% target frequency, and lasted approximately 12 minutes. Performance measures were correct responses ('hits'), false alarms, perceptual sensitivity ($d'$), response bias ($\ln\beta$), response latency (RT) and response variability (RTSD). Calculation of $d'$ and $\ln\beta$ followed the methods of Stanislaw and Todorov (1999). Higher values of $d'$ indicate greater ability to distinguish signals from noise; the response bias ratio,

**Table 2** Adolescent/Young adult sample characteristics

| | ADHD ($n$ = 98) | | Control ($n$ = 85) | | | | |
|---|---|---|---|---|---|---|---|
| *Measure* | Mean | *SD* | Mean | *SD* | *t* | *p* | *d* |
| Age (years) | 18.30 | 1.60 | 18.51 | 1.66 | .86 | .39 | .13 |
| SES | 44.10 | 17.94 | 40.89 | 16.60 | −1.23 | .22 | .19 |
| WAIS-III FSIQ | 93.05 | 14.87 | 96.79 | 15.33 | 1.64 | .10 | .25 |
| Parent report | | | | | | | |
| DSM-IV Inattention | 14.50 | 7.30 | 3.81 | 4.36 | −11.51 | <.001 | 1.77 |
| DSM-IV Hyper.-Impuls. | 9.59 | 7.72 | 1.89 | 4.08 | −8.06 | <.001 | 1.24 |
| CBCL Attention problems | 61.44 | 10.37 | 51.52 | 2.89 | −8.36 | <.001 | 1.30 |
| Adolescent self-report | | | | | | | |
| DSM-IV Inattention | 9.83 | 6.40 | 2.89 | 3.32 | −8.88 | <.001 | 1.36 |
| DSM-IV Hyper.-Impuls. | 7.84 | 6.91 | 1.93 | 2.48 | −7.41 | <.001 | 1.13 |
| YSR Attention problems | 57.48 | 9.73 | 50.53 | 1.54 | −6.34 | <.001 | .99 |

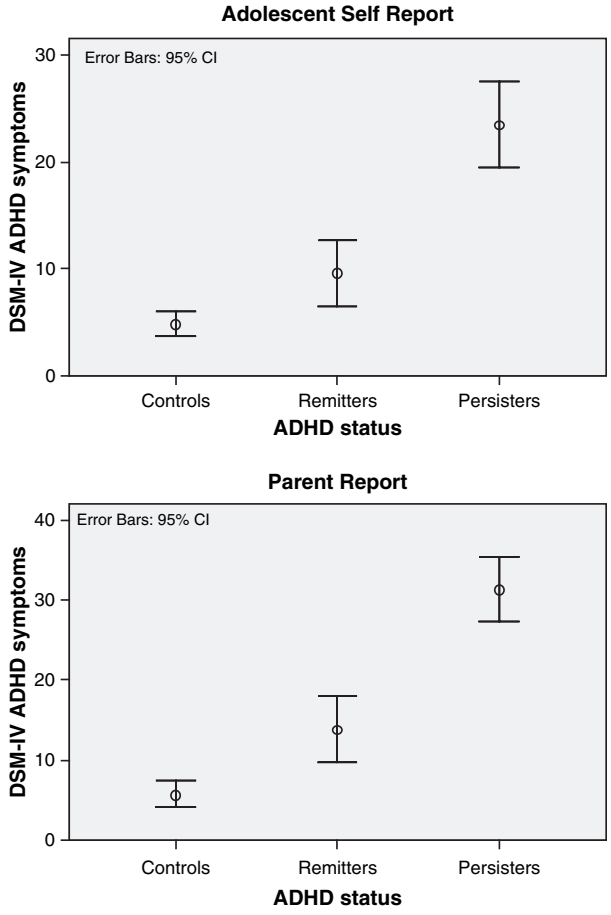


**Figure 1** DSM-IV ADHD symptom checklist scores. All three groups significantly differ from each other at $p < .001$ except controls vs. remitters on the adolescent/young adult self-report ($p < .02$)

$\beta$, is quantified using the natural logarithm of $\beta$ ($\ln\beta$), such that lower values signify an over-response bias (Stanislaw & Todorov, 1999).

*Solid-state actigraph.* Movement was recorded throughout the evaluation using two acceleration-sensitive devices with solid-state memory that store movements per minute (Santos, Guerra, Ribeiro, Duarte, & Mota, 2003). Actigraphs were placed on the non-dominant ankle and waist, and mean activity counts were calculated. To our knowledge, actigraphs have not been used with adolescents/adults with ADHD.

### *Statistical procedures*

Multivariate analysis of variance (MANOVA), with childhood status (ADHD versus control) serving as the independent variable, examined neuropsychological outcome as a function of baseline ADHD status. The MANOVA included the four WAIS-III Index scores, four Stroop *T*-scores, six CPT variables, and the two actigraph variables. Follow-up analyses were conducted using *t*-tests (two-tailed) for each outcome.

To examine the relation between symptom change over development and neuropsychological functioning, we compared persisters, remitters and controls, eliminating those with childhood ADHD who did not meet criteria for persistence or remission. To minimize loss of statistical power from subcategorizing the ADHD group and excluding 25 previously ADHD non-classified participants, separate planned orthogonal contrasts against a common, large sample control group were obtained for remitters vs. controls and persisters vs. controls. Effect sizes for between-group comparisons are reported using Cohen's $d$ statistic based on Hedges' adjustment for sample size (Devilly, 2004).

## Results

### *Childhood ADHD status*

Probands and controls differed on multiple neuropsychological measures. The omnibus test was significant (MANOVA Wilks' $\lambda$ significance = .003) with a medium effect size ($\eta_p^2 = .237$). Follow-up comparisons indicated that the groups differed significantly on the WMI, but not other WAIS-III factor scores, five of six CPT parameters, both actigraph measures, and on the control conditions of the Stroop, but not Interference (see Table 3).

### *Adolescent/Young adult ADHD status*

Neuropsychological outcome as a function of adolescent/young adult ADHD status is presented in Table 4.

There were no significant group differences on the VCI, POI or PSI from the WAIS-III; however, persisters, but not remitters, had significantly lower WMI scores than controls. On the Stroop, remitters, but not persisters, had significantly lower Word Reading scores as compared to controls. There were no significant group differences on the Stroop Color Naming, Color-Word, or Interference scores.

Patterns of CPT performance and actigraph recordings varied across groups. Persisters, but not remitters, differed significantly from controls on CPT Hits, False Alarms, RT and ln$\beta$. In contrast, RTSD and $d'$ significantly distinguished both persisters and remitters from controls. Thus, as compared to controls, both groups with childhood ADHD were significantly more variable in their consistency of response speed on the CPT and had significantly lower perceptual sensitivity. Finally, only persisters differed from controls on actigraph counts recorded from the waist, but both subgroups with childhood ADHD differed from controls on activity level recorded from the ankle.

## Discussion and conclusion

These findings indicate that adolescents/young adults with childhood ADHD have broad neuropsychological deficits relative to same-age peers with no history of ADHD, which is independent of IQ and sociodemographic factors. When the childhood ADHD group is further subdivided based on follow-up status, both persisters and remitters show deficient performance compared to controls on some measures (i.e., RTSD, $d'$ and ankle movement), but only persisters differed from controls on others (i.e., WMI; CPT Hits, False Alarms, RT and ln$\beta$; waist movement). This differential pattern of performance in individuals with childhood ADHD who vary as a function of later status can help differentiate the core or causal deficits of the disorder versus those that might be more epiphenomenal (Carr et al., 2006) or potentially contribute to the remission of ADHD symptoms (Halperin & Schulz, 2006).

Notably, despite similar FSIQ scores, the ADHD group had selectively poorer WMI than controls. However, this finding was carried by persisters and not remitters, and is strengthened by the fact that this pattern was evident on all three subtests that comprise the WMI (Digit Span, Arithmetic and Letter-Number Sequencing), whereas the groups did not differ significantly on any other WAIS-III subtest (data not shown). This supports the contention that working memory is impaired in ADHD (Barkley, 1997; Castellanos & Tannock, 2002). Yet, because deficits diminish in those who no longer meet criteria for the disorder in adulthood, our data suggest that that working memory deficits may be epiphenomenal or secondary to the core deficits of ADHD (Carr et al., 2006), and that perhaps there is a specific role for working memory in compensatory mechanisms or symptomatic 'recovery' from ADHD (Halperin & Schulz, 2006).

Data from the Stroop are consistent with those from the meta-analysis of van Mourik et al. (2005), which suggests that interference control problems

**Table 3** Neuropsychological profiles based on childhood status

| | ADHD ($n$ = 98) | | Control ($n$ = 85) | | | |
|---|---|---|---|---|---|---|
| *Measure* | Mean | *SD* | Mean | *SD* | *p* | *d* |
| WAIS-III Index Scores | | | | | | |
| Verbal Comprehension | 94.69 | 16.50 | 97.46 | 15.94 | .26 | .17 |
| Perceptual Organization | 96.00 | 14.89 | 98.01 | 13.85 | .36 | .14 |
| Processing Speed | 88.57 | 12.60 | 90.40 | 14.08 | .37 | .14 |
| Working Memory | 91.72 | 15.20 | 97.96 | 15.12 | .007 | .41 |
| Stroop Test | | | | | | |
| Word Reading | 39.89 | 7.86 | 43.89 | 8.46 | .001 | .49 |
| Color Naming | 40.34 | 8.01 | 43.73 | 9.05 | .01 | .40 |
| Color-Word | 41.72 | 10.17 | 45.72 | 9.52 | .008 | .40 |
| Interference | 49.54 | 7.14 | 50.84 | 6.56 | .22 | .19 |
| CPT | | | | | | |
| Hits (%-correct) | 71.38 | 16.74 | 80.70 | 12.93 | <.001 | .62 |
| False Alarms (%-made) | 11.54 | 14.01 | 5.84 | 9.49 | .002 | .47 |
| $d'$ | 2.01 | .97 | 2.71 | .90 | <.001 | .75 |
| ln$\beta$ | 3.33 | 2.71 | 4.85 | 4.33 | .007 | .42 |
| Hit Reaction Time (msec) | 613.35 | 108.14 | 633.66 | 93.52 | .20 | .20 |
| Hit RTSD (msec) | 229.11 | 70.18 | 191.77 | 56.69 | <.001 | .58 |
| Actigraph | | | | | | |
| Ankle Activity | 1.71 | .43 | 1.44 | .38 | <.001 | .66 |
| Waist Activity | 1.56 | .44 | 1.37 | .44 | .01 | .43 |

**Table 4** Neuropsychological profiles based on adolescent/young adult ADHD status

| | ADHD-remitters ($n$ = 29) | | ADHD-Persisters ($n$ = 44) | | Planned Contrasts | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Controls vs. ADHD-R | | | Controls vs. ADHD-P | | |
| *Measure* | Mean | *SD* | Mean | *SD* | *t* | *p* | *d* | *t* | *p* | *d* |
| WAIS-III Index Scores | | | | | | | | | | |
| Verbal Comprehension | 95.90 | 15.75 | 94.66 | 15.74 | .46 | .65 | .10 | .93 | .35 | .18 |
| Perceptual Organization | 99.17 | 15.52 | 94.15 | 12.73 | .39 | .70 | .08 | 1.46 | .15 | .29 |
| Processing Speed | 90.19 | 13.23 | 89.07 | 13.39 | .07 | .94 | .02 | .51 | .61 | .10 |
| Working Memory | 94.79 | 16.14 | 91.45 | 15.02 | .95 | .34 | .20 | 2.10 | .03 | .43 |
| Stroop Test | | | | | | | | | | |
| Word Reading | 39.03 | 9.00 | 40.76 | 7.63 | 2.70 | .01 | .55 | 1.92 | .06 | .39 |
| Color Naming | 40.10 | 7.41 | 41.55 | 9.21 | 1.91 | .06 | .44 | 1.27 | .21 | .24 |
| Color-Word | 41.72 | 9.82 | 42.55 | 11.26 | 1.85 | .07 | .41 | 1.62 | .11 | .30 |
| Interference | 50.03 | 7.63 | 49.58 | 6.89 | .54 | .59 | .11 | .94 | .35 | .19 |
| CPT | | | | | | | | | | |
| Hits (%-correct) | 75.19 | 16.74 | 70.90 | 15.45 | 1.74 | .09 | .37 | 3.43 | .001 | .68 |
| False Alarms (%-made) | 11.13 | 17.60 | 13.87 | 14.33 | 1.49 | .15 | .37 | 3.08 | .003 | .66 |
| $d'$ | 2.33 | 1.09 | 1.89 | .96 | 2.09 | .04 | .44 | 4.59 | <.001 | .94 |
| ln$\beta$ | 4.01 | 3.18 | 2.76 | 2.29 | .67 | .50 | .17 | 2.67 | .009 | .57 |
| Hit Reaction Time (msec) | 626.59 | 109.29 | 590.78 | 112.10 | .32 | .75 | .07 | 2.12 | .04 | .41 |
| Hit RTSD (msec) | 226.85 | 65.93 | 229.33 | 77.83 | 2.47 | .02 | .57 | 2.93 | <.001 | .55 |
| Actigraph | | | | | | | | | | |
| Ankle Activity | 1.66 | .49 | 1.74 | .42 | 2.25 | .03 | .50 | 3.64 | .001 | .75 |
| Waist Activity | 1.50 | .53 | 1.59 | .40 | 1.22 | .23 | .26 | 2.34 | .02 | .52 |

are not central to ADHD. Our finding that remitters were generally slower than controls on the control conditions of the Stroop suggests that they may have employed a more cautious approach to the task, perhaps emphasizing accuracy over speed.

Our CPT differentiated probands from controls on almost all parameters based on childhood status. Yet, distinct patterns across measures emerged depending on current ADHD status. Hits, False Alarms, RT and ln$\beta$ paralleled adolescent/young adult status, with only persisters differing from controls; RTSD and $d'$ paralleled childhood ADHD status. Fischer et al. (2005) reported identical results with regard to Hits and False Alarms, but did not measure any other CPT parameters.

The identical pairs CPT used in this study is a demanding task that likely challenges multiple neurocognitive processes. Participants must respond quickly when the same 4-digit stimulus appears twice in a row. As such, successful task performance, as measured by Hits and False Alarms, demands considerable resources from working memory, which needs to be updated every 2sec, and effortful control. Thus, these effortful processes, which are impaired in children with ADHD, continue to be impaired in persisters, but not remitters, suggesting that they are not central to the etiology of ADHD (Carr et al., 2006; Halperin & Schulz, 2006), but instead are epiphenomenal characteristics (Carr et al., 2006). These findings are also consistent with Halperin and Schulz' (2006) hypothesis that improvements in effortful control seen in remitters contributes to their symptom remission. However, confirmation of this causal inference will require further data.

These findings also provide support for Sergeant's cognitive-energetic model (Sergeant et al., 1999), which posits that deficits characteristic of ADHD are secondary to impairments in arousal and activation. Sergeant has suggested that $d'$ may reflect arousal mechanisms (i.e., readiness to receive information), whereas ln$\beta$ may reflect activation (i.e., readiness to respond), and several investigators have suggested that RTSD reflects some aspect of state regulation (Castellanos & Tannock, 2002; Russell et al., 2006). As compared to controls, adolescents/young adults with childhood ADHD demonstrated an impaired ability to detect CPT targets ($d'$), a bias toward responding liberally (ln$\beta$), and overall poorer state regulation (RTSD). Persisters performed worse than controls on all three measures. Remitters performed worse than controls on $d'$ and RTSD, but not ln$\beta$. Thus, poor state regulation, perceptual sensitivity and/or weak arousal mechanisms may be at the core of ADHD. It is interesting given that remitters were not different from controls on Hits and False Alarms, despite the fact that $d'$ and ln$\beta$ are typically confounded by hit and false alarm rate (Stanislaw & Todorov, 1999). Perhaps remitters again used a more conservative and less impulsive response strategy, despite continued problems with perceptual sensitivity.

Notably, the childhood ADHD group was significantly more fidgety than controls as measured by solid-state actigraphs. This finding is interesting in view of the commonly held belief that activity level declines in adults with ADHD (Hart et al., 1995). Even more compelling is that elevated activity level was detectible irrespective of current ADHD status. Not only did persisters exhibit elevated

ankle and waist activity, but remitters were also significantly more active as measured by the ankle actigraph. The smaller effect size for waist activity may reflect a greater degree of effortful regulation, as they were instructed to sit in a chair, whereas ankle activity may be less consciously mediated. It is unclear whether adults with a history of ADHD do not consistently report being fidgety because they are unaware of it or because it does not cause clinically significant impairment in adulthood.

Based on the premise that core deficits of ADHD should persist irrespective of symptom adaptation or remission (Carr et al., 2006; Halperin & Schulz, 2006), these findings suggest that state regulatory mechanisms associated with arousal and fidgetiness are central to the emergence of the disorder. In contrast, commonly seen impairments in working memory and effortful control are less likely to be causal factors in the emergence of ADHD. While the data are largely consistent with the prefrontal recovery hypothesis of Halperin and Schulz (2006) in that Remitters did not differ from controls on several measures of executive control, a stronger test of that model would directly compare persisters to remitters and demonstrate that they differ significantly on measures of controlled, but not less effortful processes. Because of the subdivision of our sample, we did not have the power to make direct contrasts between the two ADHD subgroups, and with our sample size, none would have reached traditional levels of significance (i.e., $p < .05$). Closer scrutiny with larger samples and more direct contrasts is warranted. Yet, it is difficult to know how such contrasts would turn out. One could posit that it would require exceptional prefrontal executive control (better than controls) to override the underlying ADHD deficit to result in recovery. However, ADHD remitters are still symptomatic relative to never-ADHD controls (see Figure 1) and it is unlikely that prefrontal executive mechanisms would develop independent of the integrity of lower subcortical systems. Thus, it is possible that relative improvements in executive control contribute to recovery, although it is likely that a wide array of neural and environmental factors are also in play as individuals with ADHD adapt to their environment and have reduced impairment.

These findings and conclusions must be viewed within the context of several limitations. First, despite considerable effort, we were unable to follow all 169 youth from the childhood study, although available data suggest that our subsample was representative of the original group. Second, we do not have childhood data on controls. Third, as discussed above, subdividing ADHD participants into remitters and persisters resulted in the exclusion of 25 youth and thus the loss of statistical power for analyses that were based on adolescent/adult status, preventing us from directly contrasting the two groups. This latter limitation could have been avoided by using dimensional analyses, but the model being tested lends itself to a more categorical analytic approach.

Despite limitations, these results have potentially far-reaching implications. The pattern of neurocognitive deficits in this study of adults who had childhood ADHD suggests a fundamental and enduring subcortical deficit in ADHD, with key aspects of longitudinal course being associated with differences in the development of cortical processes that impose enhanced mental control. These findings are consistent with preliminary functional magnetic resonance imaging findings, which indicate that prefrontal activation in response to inhibition in adolescents with childhood ADHD corresponds to the persistence of symptoms such that those who are less symptomatic appear more like never-ADHD controls (Schulz, Newcorn, Fan, Tang, & Halperin, 2005a; Schulz et al., 2005b). Further studies will be necessary to elucidate whether or not compensatory processes are at work, and if yes, the mechanisms by which some, but not all, children with ADHD develop these strategies.

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