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# Executive Functions in Girls With ADHD Followed Prospectively Into Young Adulthood

Meghan Miller
University of California, Berkeley

Jennifer Ho
San Diego State University/University of California, San Diego

Stephen P. Hinshaw
University of California, Berkeley

***Objective:*** We prospectively followed an ethnically and socioeconomically diverse sample of girls with ADHD ($n = 140$) and a matched comparison group ($n = 88$) into young adulthood ($M_{age} = 19.6$), 10 years after childhood initial assessments, to evaluate neuropsychological functioning. We hypothesized that neuropsychological deficits would persist through young adulthood for those with ADHD, and that those with continuing ADHD symptomatology in young adulthood would show the largest impairments. ***Method:*** Neuropsychological measures at follow-up emphasized executive functions (EF) including planning, organization, inhibitory control, sustained attention, working memory, and set shifting. ***Results:*** Parallel to findings from childhood and adolescence, the girls with childhood-diagnosed ADHD displayed medium to large deficits in EF relative to comparisons at follow-up, even with statistical control of baseline demographic and comorbidity variables. The addition of IQ as a covariate attenuated differences but several remained significant. Comparisons between the inattentive and combined subtypes of ADHD yielded nonsignificant results with small effect sizes. EF impairments were evident in both participants whose ADHD diagnoses persisted and in those whose ADHD symptoms had remitted to a nondiagnosable level; both subgroups had more EF deficits than those who did not meet criteria for ADHD in either childhood or young adulthood. ***Conclusions:*** Those in both the persistent and remitted ADHD groups showed impairments in EF relative to comparisons and generally did not differ from each other. Overall, childhood ADHD in girls portends neuropsychological/EF deficits that persist for at least 10 years.

*Keywords:* attention-deficit/hyperactivity disorder (ADHD), girls, females, executive function, longitudinal research

Attention-deficit/hyperactivity disorder (ADHD) is a prevalent and impairing neurodevelopmental disorder characterized by developmentally extreme levels of (a) hyperactivity-impulsivity and/or (b) inattention-disorganization (American Psychiatric Association, 2000). A large number of studies have documented neuropsychological deficits in individuals with ADHD, particularly within the executive function (EF) domain (e.g., Berlin, Bohlin, & Rydell, 2004; Scheres et al., 2004; Thorell, 2007). These deficits have been found across multiple studies of children and adolescents with ADHD (e.g., Rapport, Alderson, Kofler, Sarver, Bolden, & Sims, 2008; Re, De Franchis, & Cornoldi, 2010; for a review, see Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005), as well as adults with ADHD (e.g., Rohlf et al., 2011; for a review, see Boonstra, Oosterlaan, Sergeant, & Buitelaar, 2005). Although there is some debate over exactly which abilities constitute the construct of EF, it is generally conceptualized as an umbrella term that encompasses skills such as planning, organization, response inhibition, sustained attention, set shifting, working memory, and reasoning. Performance of these EFs appears to rely on the prefrontal cortex and its extensive interconnections with other brain regions (Tranel, Anderson, & Benton, 1994).

Recent studies have focused on the ability of EF to predict functioning in multiple domains. That is, EFs have been linked to academic performance (Barry, Lyman, & Klinger, 2002; Biederman et al., 2004; Miller & Hinshaw, 2010), social/interpersonal skills (Clark et al., 2002; Diamantopoulou et al., 2007; Gilotty, Kenworthy, Sirian, Black, & Wagner, 2002; Miller & Hinshaw, 2010; Rinsky & Hinshaw, 2011), occupational functioning (Barkley & Fischer, 2011), and global functioning (Miller & Hinshaw, 2010). Recent examination of the present sample indicated that EF abilities in childhood are predictive of academic and occupational functioning in young adulthood (Miller, Nevado, & Hinshaw, 2011). Thus, EFs appear to be implicated in the performance of critical cross-domain abilities.

Several issues are relevant when studying EF impairments in ADHD. First, although deficits in EF are common in ADHD, such

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Meghan Miller and Stephen P. Hinshaw, Department of Psychology, University of California, Berkeley; Jennifer Ho, Department of Psychology, San Diego State University/University of California, San Diego.

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Correspondence concerning this article should be addressed to Meghan Miller, Department of Psychology, Tolman Hall #1650, University of California, Berkeley, CA 94720-1650. E-mail: meghanmiller@berkeley.edu

deficits do not characterize all individuals with this disorder (Biederman et al., 2004; Nigg, Willcutt, Doyle, & Sonuga-Barke, 2005), and those with EF impairment may constitute a distinct subgroup (Lambek et al., 2010; Nigg et al., 2005). Second, these types of deficits are not unique to ADHD. Indeed, EF deficits have been found across multiple neurodevelopmental disorders (for a classic review, see Pennington & Ozonoff, 1996). Third, there is question as to whether the subtypes of ADHD–Predominantly Inattentive type versus Combined (or Hyperactive-Impulsive) show differential EF deficits. These subtypes, which have been posited to comprise distinct entities (see Milich, Balantine, & Lynam, 2001), have not consistently yielded neuropsychological or EF differences (e.g., Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2005; Hinshaw, Carte, Fan, Jassy, & Owens, 2007; Hinshaw, Carte, Sami, Treuting, & Zupan, 2002; O'Brien et al., 2010; Pasini, Paloscia, Alessandrelli, Porfirio, & Curatolo, 2007). In short, although EF deficits are not found in all individuals with ADHD, nor are they specific to ADHD, they have consistently been shown to be of critical importance by predicting impairment across a range of domains of functioning and could constitute a treatment target in those who do exhibit impairment.

Despite an increased focus on girls with ADHD in the past decade (see Hinshaw & Blachman, 2005), there is still a large gap in our knowledge about the presentation and course of ADHD in females, particularly in those followed prospectively. In terms of EF, studies have shown girls with ADHD to be impaired relative to age-matched female controls (Castellanos et al., 2000; Hinshaw et al., 2002; Seidman et al., 2005) and at least as impaired as boys with ADHD (O'Brien et al., 2010; Seidman et al., 2005). Longitudinal investigations of girls with ADHD have shown that these deficits appear to persist over time. Hinshaw et al. (2007) followed a sample of carefully ascertained girls ages 6–12 into adolescence and found that those with childhood-diagnosed ADHD continued to exhibit EF impairment in adolescence. Similarly, Biederman et al. (2007) followed a sample of girls ages 6–17 over 5 years and found that girls with ADHD showed continued impairment in neuropsychological functioning at follow-up, with the exception of set shifting. Thus, although it is now apparent that deficits in EF persist into adolescence in at least some childhood-diagnosed girls with ADHD, it is unknown whether such impairments continue to be present in young adulthood.

The transition to young adulthood is known to be a time of critical importance and increased demands (see Arnett, 2000), yet few studies have followed cohorts of girls with ADHD into emerging adulthood. Although the extant literature is limited, it is quite clear that cross-domain impairment persists into late adolescence and young adulthood in childhood-diagnosed girls with ADHD. Such continued impairment extends across multiple domains including ADHD symptoms, academic functioning, comorbid psychopathology, occupational functioning, and romantic and family relationships (Babinski et al., 2011; Biederman et al., 2010; Chronis-Tuscano et al., 2010; Hinshaw et al., 2011; Mick et al., 2011). However, no investigations to our knowledge have reported on the possibility of continuing EF impairments in young adulthood. Thus, we aim to examine whether deficits in EF persist into young adulthood in females with ADHD.

Our team collected EF data on a large, well-characterized, and diverse sample of preadolescent girls with ADHD and a matched comparison sample, ages 6–12 years. In childhood, the ADHD group showed clear EF deficits relative to the comparison group with stringent statistical control of demographic variables, comorbid disorders, and Full-Scale IQ (FSIQ). Comparisons between ADHD-Combined (ADHD-C) and ADHD-Inattentive (ADHD-I) subtypes were rarely significant (Hinshaw et al., 2002). This sample was then followed prospectively into adolescence with similar findings. Specifically, the childhood-defined ADHD group again showed clear EF deficits relative to comparisons under statistical control of demographic variables and comorbid disorders (but not with the addition of FSIQ); subtype differences were again minimal (Hinshaw et al., 2007). When examining EF abilities using adolescent diagnostic status (ADHD-C, ADHD-I, and no ADHD), EF deficits emerged in the ADHD groups with full statistical control, including IQ as a covariate.

At the present 10-year follow-up, parallel to our previous reports in childhood and adolescence, we aim to determine (a) whether preadolescent girls with ADHD continue to show EF impairment in young adulthood, relative to a matched comparison group; (b) whether such deficits are robust to statistical control of age, socioeconomic status, maternal education, comorbidities, and full-scale intelligence yielded from baseline measures during childhood; and (c) whether the girls who continue to meet diagnostic criteria for ADHD at follow-up show particularly strong EF deficits in young adulthood. Our main hypotheses are as follows: (1) EF deficits will persist through young adulthood for the participants with ADHD; (2) they will be robust to statistical control of demographics, comorbidities, and IQ; and (3) participants meeting criteria for ADHD in young adulthood will show the largest degree of EF impairment at follow-up. We also predict that ADHD-C and ADHD-I differences will continue to be small.

## Method

### Overview of Procedure

We used data from a longitudinal study of behavioral, neuropsychological, social, and family functioning in 228 girls, 140 with rigorously diagnosed childhood ADHD and 88 matched comparison girls. All participated in summer research programs and extensive testing during childhood (ages 6–12) and were followed prospectively into adolescence (ages 11–17) and late adolescence/young adulthood (ages 17–25), completing extensive evaluations at each time point. At young adulthood, 97% of participants were off stimulant medication for at least 24 hours before completing the neuropsychological battery. Well-trained graduate students and bachelor's-level research assistants, all of whom were closely supervised by a licensed clinical psychologist, administered the assessments and tests. Follow-up assessments had received full approval from the institution's Committee for the Protection of Human Subjects, as had all other previous phases of the research.

At the 10-year follow-up, the key goal was to appraise, via multiinformant and multimethod procedures, levels of symptomatology and adjustment/impairment in key domains of psychiatric, academic, neuropsychological, relational, and occupational functioning. For this follow-up, 216 of 228 girls (95% overall; 93% of the ADHD group, 98% of the comparison group) were retained with at least some outcome measures. Participants in this follow-up sample and those lost to attrition were statistically indistinguishable with respect 18 of 23 baseline demographic

variables examined, but the nonretained sample had lower family incomes and FSIQ scores, and higher baseline rates of teacher-rated ADHD, externalizing, and internalizing symptoms. Attrition was slightly higher in the ADHD groups than the comparison group (7% vs. 2%).

## Participants

A multigated procedure was used to recruit participants from pediatric practices, school referrals, and community advertisements to participate in free summer enrichment programs. Those in the ADHD group had to surpass sex-specific thresholds for the Swanson, Nolan, and Pelham scale (SNAP-IV; Swanson, 1992) and meet full *DSM–IV* criteria for ADHD based on the Diagnostic Interview Schedule for Children – Parent version (4th ed., DISC-IV; Shaffer, Fisher, Dulcan, & Schwab-Stone, 2000). Those in the comparison group could not meet SNAP-IV or DISC-IV criteria for ADHD; neither group could have a history of neurological damage, psychosis, pervasive developmental disorder, IQ less than 70, or medical conditions precluding participation in a summer camp. The comparison sample was matched, at a group level, with the clinical group in terms of age and ethnicity. The overall sample is both socioeconomically and ethnically diverse (family incomes ranging from public assistance to upper-middle class; 53% White, 27% African American, 11% Latina, 9% Asian American). At baseline, these 228 girls had a mean age of 9.6 years. The mean age of the comparison group was 113.2 months (*SD* = 19.8, range = 80–153), for the ADHD-I group was 118.0 months (*SD* = 20.2, range = 80–161), and for the ADHD-C group was 114.4 months (*SD* = 20.2, range = 79–160). In childhood, both the ADHD-I and ADHD-C groups had significantly lower FSIQ scores than the comparison group. At baseline, mean FSIQ for the comparison group was 112.0 (*SD* = 12.7, range = 72–140), for the ADHD-I group was 99.8 (*SD* = 14.3, range = 72–122), and for the ADHD-C group was 99.6 (*SD* = 13.2, range = 74–134). At the 10-year follow-up, the mean age of the 216 retained young women was 19.6 years. In some cases, follow-up assessments occurred via home visits or telephone interviews; in other cases, measures were missing because of fatigue or refusal; and in other instances, computer failures occurred. Thus, the sample size for the present EF battery ranges from 153–208. For secondary analyses involving follow-up diagnostic status, the DISC-IV was readministered during young adulthood, with the Young Adult version. Diagnoses were based on a combination of DISC-IV symptom endorsements and SNAP-IV symptom endorsements (young adult and parent report).

## Neuropsychological/EF Measures

We used several well-established, well-validated neuropsychological/EF measures, most of which were identical or parallel to those administered at baseline and the 5-year follow-up. We selected, a priori, one to two dependent measures from each test to minimize problems of multiple statistical tests.

## Response Inhibition

**Conners' Continuous Performance Task (CPT; Conners, 1995).** The CPT is a computerized task of attentional processing and response inhibition that requires participants to press the spacebar when target letters appears on the screen (all letters except 'X'), and not respond to the letter 'X.' Trials are presented in six blocks (interstimulus intervals: 1 s, 2 s, and 4 s) with stimuli being displayed for 250 ms. A neural network including frontal, cingulate, parietal, occipital, and temporal regions as well as the basal ganglia and cerebellum appears to be involved in the performance of this task (Ogg et al., 2008). To measure response inhibition, we selected the percentage of commission errors. Our prior work has shown significantly higher percentages of such errors in girls with ADHD than in the comparison sample, with effect sizes in the medium range (Hinshaw et al., 2002; Hinshaw et al., 2007). Conners (1995) provided criterion-related validity data based on known-groups differentiation. Higher scores indicate worse performance.

**Cancel Underline (CUL).** The CUL is a modified version of the Underlining Task (Rourke & Orr, 1977). It measures inhibitory control and rapid, accurate visual discrimination, abilities that recruit anterior cingulate and prefrontal regions (Cabeza & Nyberg, 1997). Participants were instructed to underline targets (shape or consonant sequences) and cancel out nontargets (ratio of 1:5). Our analysis is based on correct minus incorrect responses (Nigg, Hinshaw, Carte, & Treuting, 1998). Previous research has shown medium-sized differences between ADHD and comparison individuals on this measure, with those with ADHD performing worse (Carte, Nigg, & Hinshaw, 1996; Hinshaw et al., 2002, 2007; Nigg et al., 1998). Higher scores on this measure indicate better performance.

## Sustained Attention

**Conners' Continuous Performance Task (CPT; Conners, 1995).** The CPT is described above. To measure sustained attention, we selected the percentage of omission errors. As with commission errors, higher scores indicate worse performance.

## Working Memory

**WAIS-III Digit Span – Forward and Backward (Wechsler, 1997).** This is a widely used measure of auditory working memory that requires participants to immediately recall digit sequences of increasing length either in their original presentation order (Digits Forward) or in their reverse presentation order (Digits Backward). These abilities rely on frontostriatal and cerebellar regions (Martinussen et al., 2005). We analyzed raw scores for each subtest separately (Digits Forward Total Score, Digits Backward Total Score). For both Digits Forward and Digits Backward, higher scores indicate better performance.

**WAIS-III Letter-Number Sequencing (Wechsler, 1997).** This is a widely used measure of auditory working memory that requires participants to listen to sequences of letters and numbers of increasing length, immediately apply a rule to—and perform a manipulation on—the information, and repeat the new information back. We analyzed raw scores (Total Raw Score); higher scores indicate better performance.

## Set-Shifting

**Trail Making Test: Conditions 2 and 4, Delis-Kaplan Executive Functioning (Delis, Kaplan, & Kramer, 2001).** The Trail Making Test requires participants to draw lines connecting

consecutively numbered circles (Condition 2) or alternating numbered and lettered circles (Condition 4). Dependent variables are the time taken to complete each condition. Condition 4 is viewed as an assessment of cognitive flexibility/set-shifting. The ratio of Condition 4 to Condition 2 (4/2) in the traditional version of the Trail Making Test is more associated with validated set-switching tasks than either condition alone or a commonly used discrepancy score (Arbuthnott & Frank, 2000). Thus, we used this ratio as the dependent variable in the present study. Higher scores indicate worse performance.

## Global EF

**Rey Osterrieth Complex Figure (ROCF; Osterrieth, 1944).** The ROCF is a complex cognitive task that requires an individual to copy and later recall a complex figure composed of 64 segments. We analyze the Copy condition (participants draw the figure with no delay), which appears to tap multiple domains of EF such as planning, working memory, inhibitory control, attention to detail, and organization (Sami, Carte, Hinshaw, & Zupan, 2004; Shin, Park, Park, Seol, & Kwon, 2006). ROCF scores have been significantly correlated with other measures of EF (Somerville, Tremont, & Stern, 2000; Troyer & Wishart, 1997; Watanabe et al., 2005) and have been successfully used to distinguish patients with frontal lobe lesions from those without (Lezak, 1995), as well as children with ADHD from those without (Carte et al., 1996; Nigg et al., 1998; Sami et al., 2004). We used the Error Proportion Score (EPS; number of errors/total number of segments drawn), a validated method of scoring the ROCF that is a measure of efficiency (Sami et al., 2004). The intraclass correlation between the two primary scorers was .91 on a sample of 70 drawings. Among all of the EF measures in our battery, the EPS from the ROCF showed the largest effect size ($d = .90$) in differentiating the girls from ADHD from the comparison sample during childhood (Hinshaw et al., 2002; Sami et al., 2004). Thus, we believe that the EPS is a superior measure compared to other scoring systems for the ROCF. Higher scores on this measure indicate worse performance.

## Covariates

To maintain consistency with our previous reports of neuropsychological functioning in this sample at baseline and 5-year follow-up and to ascertain whether neuropsychological impairments in young adulthood are related specifically to the girls' original ADHD status without the potential influence of comorbid disorders or other confounding variables in childhood, we included two sets of covariates in secondary analyses. The first set includes demographic information (family income, maternal education), participant age, and comorbid disorders (conduct disorder or oppositional defiant disorder, mood or anxiety disorders, and reading disorder; each scored dichotomously as present vs. absent) at baseline. Because Barkley (1997) has suggested conducting neuropsychological analyses with and without statistical control of IQ, the second set of covariates includes the addition of baseline FSIQ from the WISC–III (Wechsler, 1991). Subtests comprising the WISC–III FSIQ score include Information, Similarities, Arithmetic, Vocabulary, Comprehension, Picture Completion, Coding, Picture Arrangement, Block Design, and Object Assembly. At young adult follow-up, the Wechsler Abbreviated Scales of Intelligence (WASI; Wechsler, 1999) was administered as a brief estimate of young adult IQ.

## Data Analytic Plan

All statistical analyses were performed with SPSS for Macintosh, Version 18. Correlations among dependent measures averaged $r = .18$ and ranged from $r = .00$ to .55, with the largest correlation occurring between Digits Backward and Letter-Number Sequencing. The first primary analysis was a multivariate analysis of variance (MANOVA) across the eight dependent measures, with childhood diagnostic status (comparison, ADHD-C, ADHD-I) as the independent variable. A significant MANOVA was followed up with ANOVAs for each dependent measure, using Tukey's post hoc comparisons to determine subgroup differences. Effect sizes were calculated as Cohen's *d,* with .2 constituting a small effect, .5 as medium, and .8 or above as large (Cohen, 1988). Next, we completed two multivariate analyses of covariance (MANCOVAs), first using demographic/comorbidity variables as covariates and then adding IQ as a covariate. When significant, these analyses were followed up with univariate analyses of covariance and post hoc pairwise contrasts. Finally, primary analyses were repeated with diagnostic status at young adulthood as the independent variable, using three groups: (1) those who continued to meet diagnostic criteria for ADHD in young adulthood, (2) those who met diagnostic criteria for ADHD in childhood but no longer met criteria in young adulthood, and (3) those who did not meet criteria in childhood or young adulthood. This method is consistent with other follow-up studies of participants with childhood-diagnosed ADHD (e.g., Barkley & Fischer, 2011).

# Results

The overall MANOVA yielded a statistically significant finding for the effect of baseline diagnostic status on neuropsychological performance, $F(16, 284) = 4.13$, $p < .001$. Table 1 presents group means and the results of the eight univariate ANOVAs. Six of the eight dependent measures, spanning global EF (ROCF EPS), response inhibition (CPT commissions, CUL), and working memory (Digits Forward, Digits Backward, Letter-Number Sequencing), yielded significant results. For all these variables, both ADHD subtypes scored worse than the comparison group but did not differ themselves. Table 2 reveals that effect sizes for the contrasts of (a) ADHD-C versus comparison and (b) ADHD-I versus comparison ranged from small to large in magnitude (*d* values ranging from .04 to .99). Effect sizes for ADHD-C versus ADHD-I contrasts (which were not significant) were, in general, small.

When the first set of covariates (demographics, comorbidity) was included, the MANCOVA yielded a statistically significant finding for the effect of baseline diagnostic status on young adult neuropsychological performance, $F(16, 256) = 2.80$, $p < .001$. Univariate analyses of covariance revealed that all six previously significant dependent measures continued to show significance: global EF (ROCF EPS), $F(2, 189) = 6.63$, $p < .01$; response inhibition, including CPT commissions, $F(2, 136) = 5.43$, $p < .01$ and CUL, $F(2, 189) = 6.47$, $p < .01$; and working memory, including Digits Forward, $F(2, 189) = 4.10$, $p < .05$, Digits Backward, $F(2, 188) = 3.26$, $p < .05$, and Letter-Number Se-

Table 1
*Neuropsychological Performance in Young Adulthood by Childhood Diagnostic Status*

| Measure | Comparison | | | ADHD-I | | | ADHD-C | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | *n* | *M* | *SD* | *n* | *M* | *SD* | *n* | *M* | *SD* | *F* | *p*[a] |
| ROCF error proportion score | 85 | $0.15_a$ | 0.08 | 40 | $0.23_b$ | 0.09 | 83 | $0.20_b$ | 0.10 | 11.63 | .001 |
| CPT % omissions | 71 | 2.59 | 7.31 | 31 | 4.82 | 10.94 | 51 | 2.15 | 3.57 | 1.40 | .249 |
| CPT % commissions | 71 | $28.17_a$ | 19.09 | 31 | $39.16_b$ | 22.37 | 51 | $36.05_b$ | 14.67 | 4.84 | .009 |
| Cancel Underline | 85 | $43.20_a$ | 12.28 | 40 | $33.90_b$ | 16.52 | 84 | $37.61_b$ | 15.88 | 6.29 | .002 |
| Digits Forward | 85 | $10.69_a$ | 1.80 | 40 | $9.18_b$ | 2.12 | 83 | $9.39_b$ | 1.80 | 13.87 | .001 |
| Digits Backward | 85 | $7.13_a$ | 2.07 | 40 | $5.83_b$ | 1.99 | 82 | $5.91_b$ | 2.20 | 8.76 | .001 |
| Letter-Number Sequencing | 85 | $11.20_a$ | 2.30 | 40 | $8.75_b$ | 2.38 | 82 | $9.72_b$ | 2.53 | 16.17 | .001 |
| Trails Ratio (Trails 4/Trails 2) | 84 | 2.32 | 0.65 | 39 | 2.62 | 0.89 | 82 | 2.41 | 0.86 | 1.94 | .146 |

*Note.* In a given row, means with different subscripts differ significantly on the basis of Tukey's post hoc comparisons. ROCF = Rey-Osterrieth Complex Figure; CPT = Continuous Performance Test.
[a] Significance based on one-way analysis of variance after significant omnibus multivariate analysis of variance.

quencing, $F(2, 188) = 6.46$, $p < .01$. Effect sizes for the adjusted means regarding ADHD-C versus comparison and ADHD-I versus comparison contrasts decreased slightly for ROCF, Digits Forward, Digits Backward, and Letter-Number Sequencing but were maintained for CPT commissions and CUL. Once again, contrasts between the two ADHD subtypes were generally small. When the MANCOVA was reconducted with FSIQ in the set of covariates, the overall results were attenuated but still significant, $F(16, 248) = 2.04$, $p < .05$. However, univariate analyses of covariance revealed that only three variables continued to show significance: CPT commissions, $F(2, 134) = 4.99$, $p < .01$, CUL, $F(2, 187) = 3.07$, $p < .05$, and Letter-Number Sequencing, $F(2, 186) = 3.76$, $p < .05$. The ROCF EPS showed marginal significance, $F(2, 187) = 2.89$, $p < .10$. Effect sizes ranged from small to medium for comparison versus ADHD contrasts. Notably, when covarying follow-up rather than baseline IQ, results remained the same with one exception (i.e., the CUL only showed marginal significance when using follow-up WASI-estimated IQ).

Finally, we reconducted primary analyses with the independent variable of follow-up diagnostic status (persistent ADHD, remitted ADHD, and persistent comparisons). On the basis of diagnoses that were determined in young adulthood, we found that 73 continued to meet criteria for ADHD, 54 no longer met diagnostic criteria for ADHD in young adulthood, and 75 maintained their comparison group status. In other words, 57% of the girls with childhood ADHD for whom we had young adult diagnoses continued to meet diagnostic criteria and 88% of comparisons maintained their status. Thus, as the sample of girls with childhood-diagnosed ADHD matured, a significant percentage (43%) failed to meet diagnostic criteria for ADHD in young adulthood (see also Hinshaw et al., 2011).

With young adult diagnostic status as the new independent variable, the MANOVA was statistically significant, $F(16, 260) = 3.66$, $p < .01$. Follow-up univariate analyses (see Table 3) revealed that six of the eight dependent measures yielded significant results, spanning global EF (ROCF EPS), response inhibition (CPT commissions, CUL), and working memory (Digits Forward, Digits Backward, and Letter-Number Sequencing). For five of these six dependent measures (ROCF EPS, CPT commissions, Digits Forward, Digits Backward, and Letter-Number Sequencing), both the persistent ADHD and remitted ADHD groups scored worse than the comparison group but did not differ from each other. For the CUL, the comparison group scored better than the remitted group but differed only marginally significantly from the

Table 2
*Neuropsychological Performance Effect Sizes in Young Adulthood by Childhood Diagnostic Status*

| Measure | No covariates | | | Demographic and comorbidity set[a] | | | Full set[b] | | |
|---|---|---|---|---|---|---|---|---|---|
| | ES 0–1 | ES 0–2 | ES 1–2 | ES 0–1 | ES 0–2 | ES 1–2 | ES 0–1 | ES 0–2 | ES 1–2 |
| ROCF error proportion score | −.94 | −.55 | .32 | −.70 | −.49 | .20 | −.47 | −.30 | .14 |
| CPT % omissions | −.24 | .08 | .33 | −.28 | .01 | .23 | −.11 | .09 | .22 |
| CPT % commissions | −.53 | −.46 | .16 | −.71 | −.51 | .18 | −.70 | −.50 | .19 |
| Cancel Underline | .64 | .39 | −.23 | .70 | .21 | .36 | .48 | .17 | −.31 |
| Digits Forward | .77 | .72 | −.11 | .54 | .36 | −.16 | .40 | .24 | −.15 |
| Digits Backward | .64 | .57 | −.04 | .43 | .40 | −.01 | .28 | .29 | .03 |
| Letter-Number Sequencing | .99 | .61 | −.40 | .70 | .27 | −.42 | .50 | .09 | −.42 |
| Trails Ratio (Trails 4/Trails 2) | −.39 | −.12 | .24 | −.19 | .06 | .25 | −.06 | .14 | .21 |

*Note.* ES = effect size (Cohen's *d*). Positive value of effect sizes reflects greater deviance in the first subgroup. 0 = comparisons; 1 = inattentive; 2 = combined; ROCF = Rey-Osterrieth Complex Figure; CPT = Continuous Performance Test.
[a] Covariates include baseline family income, child age, reading disorder, disruptive disorder, and internalizing disorder. [b] Covariates include demographic and comorbidity set plus child FSIQ.

Table 3
*Neuropsychological Performance in Young Adulthood by Young Adult Diagnostic Status*

| | Comparison | | | Remitted ADHD | | | Persistent ADHD | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Measure | *n* | *M* | *SD* | *n* | *M* | *SD* | *n* | *M* | *SD* | *F* | *p*[a] |
| ROCF error proportion score | 74 | $0.15_a$ | 0.09 | 52 | $0.21_b$ | 0.10 | 70 | $0.21_b$ | 0.10 | 9.96 | .001 |
| CPT % omissions | 60 | 2.56 | 7.93 | 37 | 4.74 | 10.36 | 44 | 1.89 | 2.77 | 1.54 | .218 |
| CPT % commissions | 60 | $26.57_a$ | 16.27 | 37 | $37.99_b$ | 18.73 | 44 | $37.37_b$ | 16.79 | 7.25 | .001 |
| Cancel Underline | 74 | $43.65_a$ | 12.08 | 52 | $33.79_b$ | 18.22 | 71 | $38.56_{a,b}$ | 14.18 | 7.03 | .001 |
| Digits Forward | 74 | $10.61_a$ | 1.76 | 52 | $9.44_b$ | 1.70 | 70 | $9.23_b$ | 2.07 | 11.26 | .001 |
| Digits Backward | 74 | $6.97_a$ | 1.82 | 51 | $5.86_b$ | 2.10 | 70 | $5.91_b$ | 2.17 | 6.56 | .002 |
| Letter-Number Sequencing | 74 | $11.14_a$ | 2.09 | 51 | $9.57_b$ | 2.50 | 70 | $9.31_b$ | 2.53 | 12.22 | .001 |
| Trails Ratio (Trails 4/Trails 2) | 73 | 2.26 | 0.59 | 51 | 2.42 | 0.82 | 69 | 2.52 | 0.92 | 2.03 | .135 |

*Note.* In a given row, means with different subscripts differ significantly on the basis of Tukey's post hoc comparisons. ROCF = Rey-Osterrieth Complex Figure; CPT = Continuous Performance Test.
[a] Significance based on one-way analysis of variance after significant omnibus multivariate analysis of variance.

persistent group. Table 4 reveals that effect sizes for the contrasts of (a) persistent ADHD versus comparison and (b) remitted ADHD versus comparison ranged from small to medium in magnitude (*d* values ranging from .11 to .79). Effect sizes for the persistent and remitted ADHD group contrasts (which were not significant) were generally small, ranging from .00–.38.

When the first set of covariates (demographics, comorbidity) was included, the MANCOVA yielded a statistically significant finding for the effect of young adult diagnostic status, $F(16, 232) = 2.31$, $p < .01$. Univariate analyses of covariance revealed that four of the previously significant dependent measures continued to show significance: a measure of global EF (ROCF EPS), $F(2, 177) = 5.76$, $p < .01$, two measures of response inhibition including CPT commissions, $F(2, 124) = 7.70$, $p < .01$ and CUL, $F(2, 177) = 6.70$, $p < .01$, and a measure of working memory (Letter-Number Sequencing), $F(2, 176) = 3.45$, $p < .05$. Another measure of working memory (Digits Forward) showed marginal significance. Effect sizes for the adjusted means regarding persistent ADHD versus comparison and remitted ADHD versus comparison contrasts continued to fall in the small to medium range (ranging from .05–.79). Again, contrasts between the persistent and remitted ADHD groups were generally small (ranging from .03–.33).

Finally, when the MANCOVA was reconducted with FSIQ in the set of covariates, the overall results remained significant, $F(16, 228) = 1.72$, $p < .05$. However, univariate analyses of covariance revealed that only CPT commissions continued to show significance, $F(2, 122) = 6.33$, $p < .01$, whereas the ROCF EPS showed marginal significance, $F(2, 175) = 2.38$, $p < .10$, along with the CUL, $F(2, 175) = 2.90$, $p < .10$. As in previous analyses, effect sizes ranged from small to medium for comparison versus remitted and persistent ADHD contrasts (ranging from .02-.77), and were small for persistent versus remitted ADHD contrasts (ranging from .02–.30). When covarying follow-up rather than baseline IQ, the MANCOVA was nonsignificant, $F(8, 113) = 1.45$, $p = .12$.

## Discussion

At our 10-year follow-up, featuring a high retention rate of participants, we aimed to determine whether young adult females who had been diagnosed with ADHD in childhood would continue

Table 4
*Neuropsychological Performance Effect Sizes in Young Adulthood by Young Adult Diagnostic Status*

| | No covariates | | | Demographic and comorbidity set[a] | | | Full set[b] | | |
|---|---|---|---|---|---|---|---|---|---|
| Measure | ES 0–1 | ES 0–2 | ES 1–2 | ES 0–1 | ES 0–2 | ES 1–2 | ES 0–1 | ES 0–2 | ES 1–2 |
| ROCF error proportion score | −.63 | −.63 | .00 | −.59 | −.51 | .07 | −.39 | −.36 | .03 |
| CPT % omissions | −.24 | .11 | .38 | −.25 | .05 | .31 | −.10 | .19 | .30 |
| CPT % commissions | −.65 | −.65 | .03 | −.74 | −.79 | −.10 | −.68 | −.77 | −.11 |
| Cancel Underline | .64 | .46 | −.23 | .69 | .35 | −.33 | .46 | .20 | −.26 |
| Digits Forward | .68 | .72 | .11 | .39 | .41 | .03 | .21 | .28 | .09 |
| Digits Backward | .56 | .53 | −.02 | .38 | .32 | −.05 | .21 | .19 | −.02 |
| Letter-Number Sequencing | .68 | .79 | .10 | .42 | .46 | .07 | .17 | .30 | .15 |
| Trails Ratio (Trails 4/Trails 2) | −.22 | −.33 | −.11 | −.06 | −.18 | −.16 | .02 | −.10 | −.12 |

*Note.* ES = effect size (Cohen's *d*). Positive value of effect sizes reflects greater deviance in the first group. 0 = comparisons; 1 = remitted ADHD; 2 = persistent ADHD; ROCF = Rey-Osterrieth Complex Figure; CPT = Continuous Performance Test.
[a] Covariates include baseline family income, child age, reading disorder, disruptive disorder, and internalizing disorder. [b] Covariates include demographic and comorbidity set plus child FSIQ.

to exhibit EF impairments relative to a matched comparison sample. With statistical control of relevant baseline comorbidities and demographic characteristics, impairments in EF persisted into young adulthood in females with childhood-diagnosed ADHD. These deficits emerged on tasks of global EF (ROCF), response inhibition (CPT commissions, CUL), and working memory (Digits Forward, Digits Backward, and Letter-Number Sequencing). When the additional covariate of FSIQ was included, young adult females with childhood ADHD continued to show deficits relative to comparisons on a task of response inhibition (CPT commissions, CUL) and working memory (Letter-Number Sequencing). With childhood diagnostic status as the independent variable, effect sizes for comparison versus ADHD contrasts were small to medium, whereas effect sizes for ADHD subtype contrasts (ADHD-C vs. ADHD-I) were small. Additionally, when using young adult diagnostic status (persistent ADHD, remitted ADHD, or comparison), results indicated that those in the remitted and persistent ADHD groups both performed worse than the comparison group on our measures of EF. However, the persistent and remitted ADHD groups not differ themselves. Here, effect sizes for comparison versus remitted *or* persistent ADHD contrasts were small to medium, but effect sizes for persistent versus remitted ADHD contrasts were small.

In general, our findings of continued impairment on tests of EF in females with ADHD are consistent with our previous work and the work of others who have found that neuropsychological/EF impairment is persistent in females with ADHD through at least mid-adolescence (Biederman et al., 2007; Hinshaw et al., 2007). It has been suggested that prefrontally mediated mechanisms may account for symptom reduction and potential improvements in EF throughout development in individuals with ADHD (Halperin & Schulz, 2006). Indeed, some longitudinal studies have found neuropsychological impairments to be greatest in those who continue to meet diagnostic criteria for ADHD at the follow-up time point (e.g., Fischer, Barkley, Smallish, & Fletcher, 2005; Hinshaw et al., 2007). Yet we found that those whose ADHD symptoms had remitted to a nondiagnosable level exhibited comparable EF deficits to those who continued to meet diagnostic criteria. Although these findings could suggest that a childhood diagnosis of ADHD portends continued deficits in EF regardless of whether ADHD symptoms persist at a diagnosable level, there are clear issues to be considered surrounding the use of current *DSM–IV* criteria for young adults (see Barkley, Murphy, & Fischer, 2008; Faraone, Biederman, & Mick, 2006). The *DSM–IV* criteria for ADHD have not been validated in adults and do not take into consideration developmental differences in symptom expression in adulthood, including decreases in symptoms of hyperactivity-impulsivity (McGough & Barkley, 2004). Using alternative criteria to define ADHD in young adulthood may yield different results.

In childhood, our group found that EF impairments were present in the ADHD groups even with statistical control of baseline demographic characteristics, comorbidity, and FSIQ (Hinshaw et al., 2002). In adolescence, the findings were similar, but the addition of FSIQ as a covariate eliminated the effect of childhood ADHD diagnostic group status on adolescent neuropsychological performance (Hinshaw et al., 2007). Somewhat parallel to the findings of Hinshaw et al. (2007), the addition of baseline FSIQ attenuated the effect of childhood ADHD on young adult EF, but the present omnibus findings remained significant and some group differences persisted in terms of individual dependent measures. Why this discrepancy in significance of findings with the addition of IQ exists is unclear. It may be attributable to a higher correlation between baseline IQ and adolescent performance than young adult performance, but we highlight the importance of conducting analyses of cognitive abilities in individuals with ADHD with and without covarying IQ. Indeed, there is extensive debate over whether IQ scores should be covaried when comparing individuals with ADHD with comparison groups (see Barkley, 1997). It may be that controlling for IQ constitutes overcontrol (see Miller & Chapman, 2001), given that deficits in IQ are inherent to the ADHD construct. Thus, even though we performed our analyses with and without covarying IQ, we emphasize the findings without IQ as a covariate.

The pattern of intact versus impaired EF measures in young adulthood was identical when using childhood or young adult diagnostic status and covarying demographic and comorbidity variables (but excluding IQ). Specifically, only two of the EF domains assessed appeared to be intact in the ADHD group: sustained attention and set shifting. Impairments were present in global EF, response inhibition, and working memory. Thus, in females with childhood-diagnosed ADHD, impairments across several domains of EF appear to persist, at least until young adulthood. Others have found sustained attention deficits in adults with ADHD (Marchetta, Hurks, De Sonneville, Krabbendam, & Jolles, 2007) and have revealed that adults with ADHD require increased cortical activation during sustained attention tasks (Loo et al., 2009). Additionally, previous studies have found set shifting deficits in adults with ADHD (Boonstra, Kooij, Oosterlaan, Sergeant, & Buitelaar, 2010; Rohlf et al., 2011). Yet few of these studies included significant numbers of women. It may be that the EF profile of females differs from that of males. Indeed, Hinshaw et al. (2007) did not find differences in sustained attention during adolescence in the present sample upon introduction of demographic and comorbidity covariates. Similarly, Biederman et al. (2007) did not find deficits in sustained attention in their midadolescent sample of females with ADHD.

We emphasize our findings without IQ as a covariate, because of both concerns about overcontrol and the contention that IQ should not be included as a covariate in studies of cognitive abilities in neurodevelopmental disorders (e.g., Barkley, 1997; Dennis et al., 2009). However, when including the full set of covariates in the present study, only response inhibition was impaired in the ADHD group (childhood-defined) relative to comparisons. A continued focus on examining large samples of females with ADHD, as well as comparisons between males and females with ADHD, may help to clarify questions concerning potentially distinct neuropsychological profiles.

Consistent with earlier findings from the present sample as well as the work of others (Geurts et al., 2005; O'Brien et al., 2010; Pasini et al., 2007), we found little evidence of ADHD subtype differences in EF. Notably, most previous studies of subtype differences focus on children and adolescents, but similar results have emerged in studies of adults with ADHD (Schweitzer, Hanford, & Medoff, 2006). In our study, the subtypes generally did not differ significantly, even for measures on which the ADHD-C group should have, by definition, performed worse than the ADHD-I group (e.g., tasks related to response inhibition/impulsivity). Thus, although there has been contention that the

inattentive subtype is decidedly distinct from the combined subtype (Milich et al., 2001), the two subtypes were generally indistinguishable during childhood, adolescence, and young adulthood in terms of the EF variables we selected. Further efforts to improve subtype definitions are warranted.

The current findings have important implications in light of the current development of revised diagnostic criteria as well as a focus on specific criteria for research purposes. First, our findings highlight concerns regarding the validity of current *DSM–IV* criteria for adults. Current proposals for DSM-5 criteria partially address these concerns by requiring a reduced number of symptoms for individuals age 17 and up, but symptom descriptions continue to lack integration of developmental science. Careful attunement to developmental appropriateness will continue to be important for clinicians. Furthermore, the DSM-5 proposes an enriched set of impulsivity-related symptoms, which may be essential for capturing this critical domain of functioning, both symptomatically and neuropsychologically. Additionally, a focus on dimensions of functioning rather than categorical definitions, as proposed by NIMH's Research Domain Criteria project, will likely be beneficial. For example, a focus on understanding neural and genetic correlates of EF deficits—regardless of subject population—could lead to important findings related to underlying mechanisms and treatments that would address EF impairments transdiagnostically.

Limitations of this investigation include the time-limited nature of our follow-up neuropsychological battery, which was necessary to obtain a multidomain assessment of functioning in this sample. Because we truncated the neuropsychological measures in our battery, we cannot address whether young adult females with ADHD exhibit difficulties in non-EF neuropsychological domains. For example, motor problems were not evaluated, but others have found greater motor overflow in girls with ADHD than boys with ADHD (O'Brien et al., 2010). Second, while the retention rate of 95% for the larger follow-up was high, home visits, equipment failure, and missed tests for some participants reduced the amount of neuropsychological data available. Finally, the sample was clinically ascertained, and it is not clear whether our results would be similar in a community sample of females with ADHD.

Overall, the findings of the present study are consistent with other work showing that neuropsychological problems are persistent in individuals with ADHD (e.g., Fischer et al., 2005). Although few studies have prospectively followed large samples of females with ADHD, our results are in line with the few longitudinal studies of females with ADHD that have found continuing neuropsychological deficits in adolescence (Biederman et al., 2007; Hinshaw et al., 2007). Thus, these findings add to the evidence that EF impairment persists into young adulthood in females with childhood-diagnosed ADHD. A continued focus on the female ADHD phenotype and its developmental course across various symptom and functioning domains is of great importance, given potential unique risks for this group (Hinshaw & Blachman, 2005; Hinshaw et al., 2011; Rucklidge, 2010). In particular, future work should focus on examining (a) how EF impairments are related to other domains of functioning in individuals with ADHD; (b) the developmental trajectories of EF in females with and without childhood-diagnosed ADHD; and (c) how these trajectories relate to changes in ADHD symptoms over time. Finally, the development of valid diagnostic criteria for ADHD in adulthood is of high relevance, both clinically and conceptually.

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