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Executive functions in girls with and without childhood ADHD: developmental trajectories and associations with symptom change

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Background: We prospectively followed an ethnically and socioeconomically diverse sample of girls with attention-deficit/hyperactivity disorder (ADHD) (n = 140) and a matched comparison sample (n = 88) from childhood through young adulthood to evaluate developmental trajectories of executive functions (EF) and associations between EF trajectories and dimensional measures of ADHD symptoms. We hypothesized that (a) EF trajectories would be similar in girls both with and without childhood ADHD, with the ADHD group showing greater impairment across time; and (b) changes in EF abilities would predict changes in ADHD symptoms across time, consistent with the theory that ADHD symptom reductions partially result from prefrontally mediated EF development. **Method:** Latent growth curve models were used to evaluate development of sustained attention, response inhibition, working memory, and global EF abilities, and associations between EF trajectories and ADHD symptom trajectories. **Results:** Girls with childhood-diagnosed ADHD showed greater improvement across development on measures of sustained attention and global EF, but similar rates of improvement on measures of working memory and response inhibition. Changes in the global EF measure predicted changes in both inattentive and hyperactive-impulsive symptoms across time, whereas changes in response inhibition predicted changes in hyperactive-impulsive symptoms; associations between changes in other EF variables and symptoms were not significant. **Conclusions:** Findings suggest variability in patterns of EF improvement over time in females with ADHD histories and indicate that EF development may play a role in symptom change. **Keywords:** Attention-deficit/hyperactivity disorder, females, development, longitudinal, executive functions, symptoms.

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a prevalent and impairing neurodevelopmental disorder characterized by developmentally extreme levels of inattention-disorganization and/or hyperactivity-impulsivity (American Psychiatric Association, 2000). Neuropsychological deficits, particularly those related to executive function (EF), are common in individuals with ADHD (Willcutt, Doyle, Nigg, Faraone & Pennington, 2005). EF encompasses skills such as planning, organization, response inhibition, sustained attention, set shifting, working memory, and reasoning; performance of these abilities relies on the prefrontal cortex (PFC) and its interconnections with other brain regions (Tranel, Anderson & Benton, 1994).

Executive function deficits are consistently found in youth with ADHD (e.g. Hinshaw, Carte, Fan, Jassy & Owens, 2007; Rapport et al., 2008); the body of literature focusing on adults with ADHD is smaller, but such impairments have also been documented in adulthood (e.g. Rohlf et al., 2012). PFC dysfunction has been suggested to be central to the development of EF impairments in ADHD (Barkley, 1997; Benson, 1991). Alternatively, ADHD symptoms may result primarily from noncortical dysfunction (e.g. subcortical regions related to attention, impulsivity, reward sensitivity), with prefrontally mediated mechanisms accounting for ADHD symptom reduction across development (Halperin & Schulz, 2006). Although longitudinal studies of children with ADHD suggest that EF deficits persist into adulthood (e.g. Biederman et al., 2007) and that hyperactive-impulsive symptoms decrease more rapidly than inattentive symptoms (e.g. Biederman, Mick & Faraone, 2000; Hinshaw et al., 2012; Kessler et al., 2010), sorely lacking is research documenting developmental changes in EF abilities and how such changes are associated with symptom changes over time, particularly in females.

One study focusing on the course of attention in primarily boys with ADHD revealed that the largest group differences existed prior to adolescence (Drechsler, Brandeis, Földényi, Imhof & Steinhausen, 2005). In a longitudinal neuroimaging study of boys and girls with ADHD, Shaw et al. (2006) found that children with ADHD who had worse clinical outcomes at follow-up showed thinning of the left medial PFC, a region involved in EF, whereas diagnosed children with better clinical outcomes showed normalization of the right parietal region. Indeed, associations between ADHD symptoms and neuropsychological variables have been suggested (Nigg, 2005; Seidman, 2006). An examination of EF in those with persistent versus remitted ADHD found that only those with persistent ADHD into adolescence showed deficits (Halperin, Trampush, Miller,

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Marks & Newcorn, 2008), suggesting that developmental improvements in symptoms are associated with EF improvements. Others have found no differences in EF performance between persisters and remitters (Biederman et al., 2009; Miller, Ho & Hinshaw, 2012).

A key concern pertains to current diagnostic criteria for defining ADHD in adulthood (McGough & Barkley, 2004). The use of categorical variables to define the persistence of ADHD symptoms may mask relevant associations, given that ADHD symptoms exist along a continuum and that ADHD status can fluctuate across development (Lahey, Pelham, Loney, Lee & Willcutt, 2005). A focus on associations between changes in EF and changes in dimensional measures of ADHD symptoms may be informative. A relevant longitudinal study did not find associations between improvements in EF and change in ADHD symptoms (Vaughn et al., 2011), yet the focus was solely on variables from a continuous performance task.

Females with ADHD exhibit significant functional impairments across development (Biederman et al., 2008, 2010; Hinshaw et al., 2012), and understanding (a) trajectories of EF and (b) associations between such trajectories and developmental changes in ADHD symptoms is a scientific and clinical priority in girls and women. Previous cross-sectional work in the present sample has shown that EF deficits are present in females with ADHD during childhood (Hinshaw, Carte, Sami, Treuting & Zupan, 2002), adolescence (Hinshaw et al., 2007), and young adulthood (Miller, Ho, et al., 2012). Although studies of the female ADHD phenotype are becoming more common, the developmental course of neuropsychological deficits (vs. cross-sectional comparisons of group differences) in female samples is not well characterized. Thus, we aimed to assess (a) developmental trajectories of EF in females with and without ADHD, and (b) associations between change in EF and change in ADHD symptoms (inattentive, hyperactive-impulsive) between childhood and young adulthood, evaluating whether EF abilities act as a mechanism underlying symptom change in females. The latter question could have important implications for the conceptualization of EF deficits in ADHD, as simultaneous reductions in neuropsychological deficits and ADHD symptoms may indicate that such neuropsychological impairments are epiphenomenal to ADHD (e.g. Carr, Nigg & Henderson, 2006).

Using latent growth curve modeling (LGM), we expected to find similar rates of change in participants with ADHD and comparison participants over time. Based on the model proposed by Halperin and Schulz (2006), we expected greater improvement in EF scores to be associated with greater reductions in hyperactive-impulsive symptoms, and, to a lesser extent, inattentive symptoms, given that hyperactive-impulsive symptoms have been reported to decrease more rapidly (e.g. Biederman et al., 2000).

Method

Overview of procedure

The present investigation utilizes data from a longitudinal study of behavioral, neuropsychological, social, and family functioning in 228 girls, 140 with 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 11–18) and late adolescence/young adulthood (ages 17–24), completing multi-domain evaluations. An extensive neuropsychological battery was performed during childhood, with repeated measures of key domains at adolescent and young-adult follow-ups. Medicated participants were off stimulant medication during testing (washout period ≥ 24 h), with the exception of three participants in young adulthood who were tested while receiving stimulant medications.[1] Well-trained graduate students and bachelor's-level research assistants, supervised by a licensed clinical psychologist, administered assessments.

For the adolescent follow-up, 209 girls (92%) were retained for evaluation (93% of comparison group, 91% of ADHD group). Those retained and those lost to attrition were statistically indistinguishable with respect to nearly all baseline variables examined (Hinshaw, Owens, Sami & Fargeon, 2006). At the young adult follow-up, 216 girls (95%) were retained (98% of comparison group, 93% of ADHD group). Retained participants and those lost to attrition were again statistically indistinguishable with respect to nearly all baseline variables examined (Hinshaw et al., 2012). Data loss for neuropsychological variables was higher than the overall attrition rate resulting from home-based assessments, fatigue/refusal, and computer failures. Neuropsychological data loss was consistent across time and measures (~5–10%) with the exception of a Continuous Performance Task in young adulthood (~30%). All assessments received approval from UC Berkeley's Committee for the Protection of Human Subjects; all participants provided written informed assent/consent.

Participants

A multi-gated procedure was used to recruit participants from pediatric practices, schools, and the community to participate in summer enrichment programs. Those in the ADHD group had to surpass initial, sex-specific thresholds for the Swanson, Nolan, and Pelham scale (SNAP-IV; Swanson, 1992) and meet full DSM-IV criteria for either the Combined (ADHD-C) or Inattentive (ADHD-I) subtype based on the Diagnostic Interview Schedule for Children–Parent version (4th edn., DISC-IV; Shaffer, Fisher, Lucas, Dulcan & Schwab-Stone, 2000); those who met criteria for the Hyperactive-Impulsive subtype were excluded. 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 sample was group-matched on age and ethnicity.

The overall sample is 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, the sample had a mean age of 9.6 years (*SD* = 1.7). Of those with ADHD, 93 had ADHD-C and 47 had ADHD-I. The ADHD-I (*M* = 99.8, *SD* = 14.3) and ADHD-C (*M* = 99.6, *SD* = 13.2) groups had lower FSIQ scores than the comparison group (*M* = 112.0, *SD* = 12.7; see Hinshaw, 2002). At the adolescent follow-up, the mean age of the 209 retained girls was 14.2 years (*SD* = 1.6), and at the young adult follow-up, the mean age of the 216 retained young women was 19.6 years (*SD* = 1.7). Between childhood and adolescent assessments, 27% of the ADHD-I and 45% of the ADHD-C group were on stimulant medication for at least some of the interval; between adolescent and young adult assessments, the rates were 44% of the ADHD-I and 58% of the ADHD-C group.

Diagnostic classification and symptom measures

The parent version of the DISC-IV was used for ADHD classification at baseline. The parent-completed SNAP-IV (Swanson, 1992) rating scale was used at all time points, yielding dimensional counts of inattentive and hyperactive-impulsive symptoms. Parents of participants on medication were asked to complete symptom ratings based on unmedicated behavior.

Measures of EF

Well-established, well-validated measures of EF were selected based on (1) evidence that individuals with ADHD show impairments in these domains and (2) repeated administration in the present sample.

Rey-Osterrieth Complex Figure Test (RCFT; Osterrieth, 1944; childhood, young adulthood). The RCFT is a complex cognitive task requiring an individual to copy and recall a complex figure composed of 64 segments. It assesses multiple domains of EF, including planning, response inhibition, attention, and organization, serving as a comprehensive measure of EF. The copy condition was used; only it differentiated the two groups at baseline (Sami, Carte, Hinshaw & Zupan, 2003) and was most predictive of outcomes in adolescence (Miller & Hinshaw, 2010) and young adulthood (Miller, Nevado & Hinshaw, 2012). We used the error proportion score (EPS), a validated scoring method defined as the number of errors divided by total number of segments drawn, indexing efficiency (Sami et al., 2003). Intraclass correlations between pairs of the three primary scorers at baseline ranged from 0.91 to 0.94 (drawings n = 84–195 across rater pairs); at young adult follow-up, intraclass correlations between the two primary scorers were 0.91 on a sample of 70 drawings. Higher scores indicate greater impairment.

Taylor Complex Figure Test (TCFT; Taylor, 1969; adolescence). This measure is the only major alternative to the RCFT in test–retest situations (Helmes, 2000) and thus served as a parallel form of the RCFT in adolescence. The copy condition was utilized, again using the EPS. Intraclass correlations between pairs of the three scorers ranged from 0.77 to 0.94, with a mean of 0.84 on a subsample of 60 drawings. Higher scores indicate greater impairment.

Conners' Continuous Performance Task (CPT; Conners, 1995; all time points). The CPT is a computerized visual task of attentional processing and response inhibition requiring the participant to press the spacebar when target letters appears on the screen (all except 'X'), and not respond to the letter 'X'. The 14-min task consists of trials presented in six blocks (interstimulus intervals: 1, 2, 4 s); stimuli are displayed for 250 ms. We analyzed percentage of omission errors (sustained attention) and percentage of commission errors (response inhibition); higher scores indicate greater impairment. Conners (1995) provided criterion-related validity data based on known-groups differentiation.

WISC-III Digit Span (Wechsler, 1991; all time points). This measure of auditory working memory requires participants to immediately recall digit sequences of increasing length in original (Digits Forward) or reverse order (Digits Backward). Given the importance of manipulation of information to the construct of working memory, we analyzed Digits Backward, using raw rather than age-standardized scores. This variable was reverse-scored to align with the direction of all other measures.

Covariates

In analyses of developmental trajectories of EF, we included baseline oppositional defiant disorder (ODD) or conduct disorder (CD) ascertained from the DISC-IV, coded as 1 versus 0 for their presence versus absence. We intentionally did not include IQ as a covariate because IQ deficits are likely to be associated with EF impairments; removing IQ-associated variance would diminish potential associations between EF and ADHD measures (see Barkley, 1997; Dennis et al., 2009; Vaughn et al., 2011). No covariates were included in analyses concerning associations between EF change and ADHD symptom change because of sample size requirements of structural equation modeling (SEM; see Kline, 2005).

Data analytic plan

EF trajectories. Developmental change across the four EF domains was examined with separate LGMs. Following Bollen and Curran (2006), data were re-organized so that change was modeled on participant age rather than assessment period. Accordingly, participants' ages across all three assessment periods were first divided into quartiles, approximating developmental periods of childhood (Mdn_{age} = 8.7 years), early adolescence (Mdn_{age} =12.1 years), adolescence (Mdn_{age} = 16.0 years), and young adulthood (Mdn_{age} = 20.0 years). Participants were assigned to a respective group based on age at each assessment. Those who completed each assessment had a maximum of three observations; the fourth observation and other missing data were estimated using maximum-likelihood procedures (see below). This strategy improves upon analytic techniques that confound assessment period and age (see Bollen & Curran, 2006).

Executive function growth curve trajectories were freely estimated from the data rather than specified *a priori*, allowing for growth trajectories to take nonlinear forms. Childhood ADHD diagnostic status was included as a predictor of EF intercept and slope factors. Significant associations indicated that mean EF scores in childhood and/or rate of EF change across development differed by diagnostic group. Childhood ODD/CD was also included as a predictor (i.e. covariate) of slope and intercept factors. Predictor variables were allowed to covary.

Associations between EF and ADHD symptom change. These analyses tested the hypothesis that developmental trajectories of EF abilities are predictive of ADHD symptom change across time. Separate models were constructed to examine hypothesized relations between each EF variable and each ADHD symptom domain (inattention, hyperactivity-impulsivity), resulting in eight analyses. Each model contained separate LGMs of EF abilities and ADHD symptoms, with individual trajectories modeled according to the analytic procedures outlined above, except without the inclusion of ODD/CD. Next, pathways were included from EF intercept and slope factors to ADHD symptom intercept and slope factors. Primary hypotheses were tested by examining regression coefficients predicting ADHD symptom change from EF change. Significant associations between these latent constructs represented a moderated effect, such that ADHD symptom change differed as a function of EF change.[2]

Missing data. Missing data emanated from subject attrition, the organizational structure of the data, and incomplete EF and symptom data. Missing data were estimated utilizing full maximum likelihood procedures, which yield unbiased and efficient population estimators (the recommended strategy for handling missing data with LGMs; Bollen & Curran, 2006). Thus, data from all 228 participants were utilized in all growth curve analyses. Model fit was evaluated with the model chi-square statistic, comparative fit index (CFI; values >0.90), standardized root mean square residual (SRMR; <0.10), and root mean square error of approximation (RMSEA[3]; values <0.08 with 90% CI containing 0) (Hu & Bentler, 1999; Kline, 2005). We appraised significance of model parameters using conventional standards (α = 0.05). Analyses were conducted using EQS v.6.1 software (Bentler, 2006).

Because SEM requires data be distributed multivariate normal, we first examined univariate displays as a preliminary check on this assumption. One variable, CPT omissions, evidenced significant positive skew and kurtosis and was thus transformed ($-1/\sqrt{(1 + \text{variable})}$). For each model, we also examined the normalized estimate of Mardia's coefficient to check for multivariate kurtosis (Bentler, 2006). For the only model violating this assumption (i.e. CPT omissions; $g_{2,\ p} = -3.59$, $p < 0.001$), we report the Yuan and Bentler (1998)scaled chi-square statistic and robust versions of the CFI and RMSEA and test the significance of model parameters utilizing robust standard errors (see Bentler, 2006).

Results

EF trajectories

Global EF (RCFT). The baseline LGM for RCFT showed an excellent fit to the data, $\chi^2(7, N = 228) = 2.50$, *ns*, CFI = .989, SRMR = 0.029, RMSEA = 0.000, $CI_{0.90}$ {0.000, 0.025}. Total RCFT scores decreased (i.e. improved) over time ($b = -0.10$, $p < 0.001$), with the largest decrease (~82% of total change) occurring between childhood and early adolescence (factor loading = 0.82, $p < 0.001$). Childhood ADHD status was predictive of intercept and slope factors, indicating that RCFT levels in childhood and the rate at which RCFT scores improved differed between groups (see Table 1; Figure 1A). Specifically, girls with ADHD had higher (worse) RCFT scores at baseline and evidenced greater improvement than did comparison participants. The variance of both intercept and slope terms was significant (intercept = 0.03, $p < 0.001$; slope = 0.03, $p < 0.001$), indicating individual differences in baseline RCFT scores and rate of RCFT change not accounted for in this model.

Sustained attention (CPT omissions). The baseline LGM of CPT omissions also showed an excellent fit to the data, Y-B$\chi^2(7, N = 228) = 6.19$, *ns*, CFI = .967, SRMR = 0.059, RMSEA = 0.000, $CI_{0.90}$ {0.000, 0.076}. Results indicated that CPT omissions

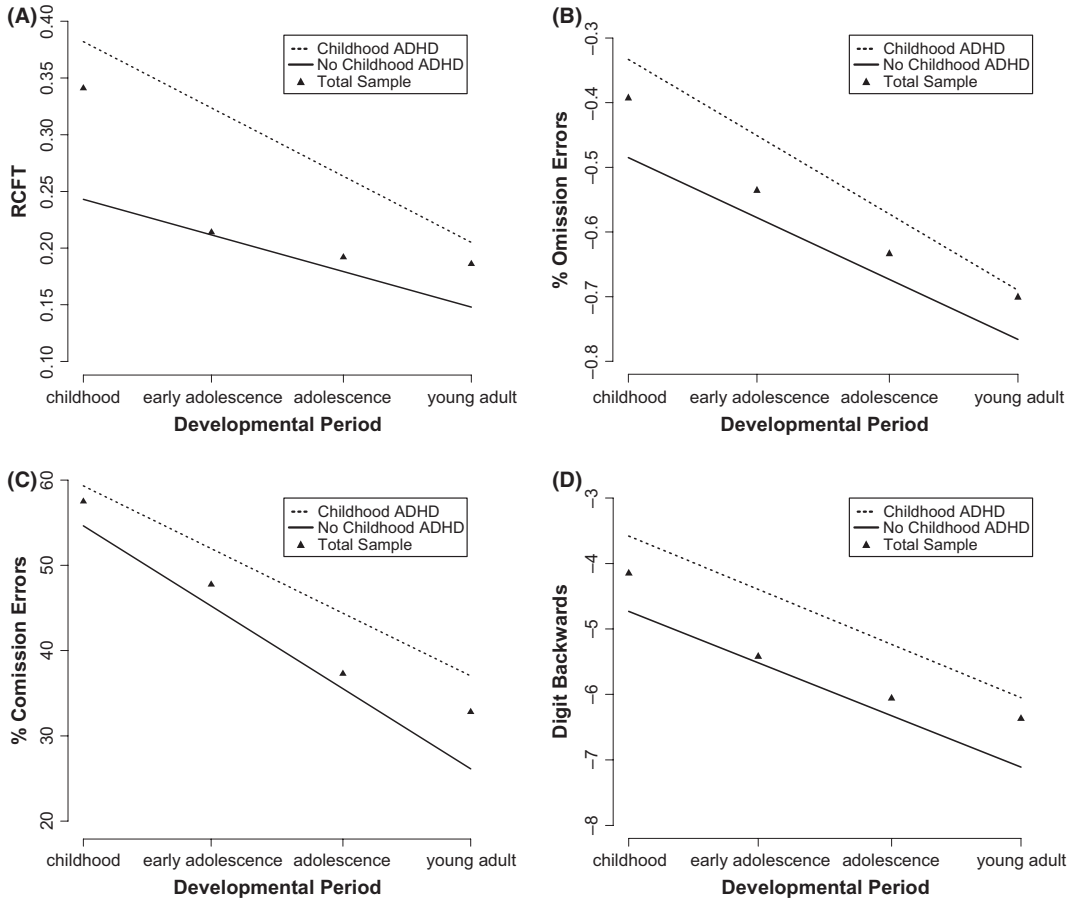
Table 1 Summary of moderating effects of childhood ADHD status on developmental trajectory of executive functions from childhood through young adulthood

Model	*b*	*SE b*	*Z*	*β*
Global EF (ROCF)				
ADHD → Intercept	0.14	0.03	4.88***	0.36
ADHD → Slope	−0.08	0.03	−2.96**	−0.24
Sustained attention (CPT % Omissions)[a]				
ADHD → Intercept	0.15	0.02	7.28***	0.77
ADHD → Slope	−0.07	0.03	−2.23*	−0.23
Response inhibition (CPT % Commissions)				
ADHD → Intercept	4.84	2.52	1.92	0.21
ADHD → Slope	5.83	3.16	1.84	0.21
Working memory (Digit Span Backward)				
ADHD → Intercept	1.17	0.23	5.01***	0.76
ADHD → Slope	−0.13	0.31	−0.41	−0.11

ADHD, attention-deficit/hyperactivity disorder; ROCF, Rey-Osterrieth Complex Figure Test; CPT, Continuous Performance Test; EF, executive function. ADHD was coded 0 = comparison and 1 = ADHD.
All models included baseline ODD/CD diagnostic status as an additional predictor of EF slope and intercept factors, except for Response Inhibition.
[a]Robust standard errors were utilized in these models to account for departures from multivariate normality.
*$p < 0.05$,
**$p < 0.01$,
***$p < 0.001$.

decreased (i.e. improved) over time for all participants ($b = -0.27$, $p < 0.001$), with nearly half of total change occurring by early adolescence (factor loading = 0.46, $p < 0.001$), and an additional 30% by late adolescence (factor loading = 0.79, $p < 0.001$). Childhood ADHD status was again associated with CPT omissions in childhood and the rate at which these scores decreased over time (see Table 1; Figure 1B); girls with ADHD evidenced worse performance at baseline and greater improvement than comparison girls. Variance of intercept ($\sigma^2 = 0.003$, $p < 0.05$) and slope ($\sigma^2 = 0.02$, $p < 0.001$) were again significant, reflecting individual variability in these terms.

Response inhibition (CPT commissions). Initial estimates of the baseline LGM of CPT commissions produced a model that did not fit the data well, $\chi^2(7, N = 228) = 13.25$, $p = 0.07$, CFI = .864, SRMR = 0.052, RMSEA = 0.063, $CI_{0.90}$ {0.00, 0.114}. We omitted ODD/CD as a covariate, which resulted in improved model fit, $\chi^2(5, N = 228) = 1.58$, *ns*, CFI = .996, SRMR = 0.031, RMSEA = 0.000, $CI_{0.90}$ {0.00, 0.038}. CPT commission errors decreased (i.e. improved) on average (estimate = −28.38, $p < 0.001$); the rate of change was relatively consistent across development, with approximately 40% occurring between childhood and early adolescence



Figures 1 (A–D) Developmental trajectories of EF for girls with and without childhood ADHD. (A) global EF (moderated effect), (B) sustained attention (moderated effect), (C) response inhibition (no moderated effect), and (D) working memory (no moderated effect).

(factor loading = 0.39, $p < 0.001$), and an additional 40% between early adolescence and adolescence (factor loading = 0.82, $p < 0.001$). Childhood diagnostic status was not predictive of initial CPT commission scores or rate of change (see Table 1; Figure 1C), reflecting equivalent developmental trajectories of response inhibition in girls with and without childhood ADHD. Both intercept ($\sigma^2 = 125.07$, $p < 0.01$) and slope ($\sigma^2 = 182.04$, $p < 0.01$) variance terms were significant, again indicating individual variability in growth factors.

Working memory (Digits Backwards). The baseline LGM showed an excellent fit to the data, $\chi^2(7, N = 228) = 5.89$, *ns*, CFI = 0.974, SRMR = 0.035, RMSEA = 0.000, $CI_{0.90}$ {0.000, 0.073}. On average, working memory scores improved (estimate = −2.33, $p < 0.001$), with nearly 60% of change occurring by early adolescence (factor loading = 0.57, $p < 0.001$) and an additional 25% by adolescence (factor loading = 0.86, $p < 0.001$). Childhood diagnostic status was predictive of initial Digits Backwards scores, such that girls with childhood ADHD recalled fewer digits, but diagnostic status did not predict rate of change (see Table 1; Figure 1D). Neither intercept nor slope variances were significant ($\sigma^2 = .31$ and $\sigma^2 = 0.30$, respectively).

Associations between EF and ADHD symptom change

There was no evidence of departures from multivariate normality in the following models. Relevant path coefficients are summarized in Table 2.

Global EF (RCFT). Hyperactive-impulsive symptoms: This model fit the data well, $\chi^2(22, N = 228) = 22.34$, *ns*, CFI = 0.934, SRMR = 0.042, RMSEA = 0.008, $CI_{0.90}$ {0.000, 0.056}. The latent RCFT intercept factor was positively associated with rate of hyperactive-impulsive symptom change, indicating that higher (worse) scores on the RCFT at baseline were associated with greater reductions in hyperactive-impulsive symptoms across development. In addition, there was a positive association between RCFT and hyperactive-impulsive slope factors, signifying that the more that RCFT scores decreased across development, the faster hyperactive-impulsive symptoms decreased. There was no association between baseline RCFT scores and baseline hyperactive-impulsive symptoms. Variance terms for intercept ($\sigma^2 = .16$, $p < 0.001$) and slope ($\sigma^2 = .19$, $p < 0.001$) factors of the hyperactive-impulsive symptom trajectory were both significant, suggesting individual differences in symptom growth not accounted for by EF development.

Inattentive symptoms: This model also fit the data exceptionally well, $\chi^2(22, N = 228) = 18.28$, *ns*, CFI = 0.971, SRMR = 0.031, RMSEA = 0.000, $CI_{0.90}$ {0.000, 0.044}. Baseline RCFT scores were not predictive of baseline inattentive symptoms but were positively associated with rate of inattentive symptom change, such that higher (worse) RCFT scores in childhood were associated with greater decreases in inattentive symptoms. In addition, rate of RCFT change was predictive of inattentive symptom change, suggesting that the more that RCFT

Table 2 Summary of moderating effects of change in EF on change in ADHD symptoms across development

Pathways	Hyperactive-impulsive symptoms			Inattentive symptoms		
	b (*SE b*)	*Z*	*β*	*b* (*SE b*)	*Z*	*β*
Global EF (RCFT)						
Intercept → ADHD Intercept	−0.41 (0.67)	−0.60	−0.09	−0.56 (0.58)	−0.95	−0.10
Intercept → ADHD Slope	2.45 (0.78)	3.13**	0.76	2.78 (0.97)	2.87**	0.78
Slope → ADHD Slope	2.97 (0.85)	3.47***	0.83	2.41 (1.07)	2.26*	0.60
Sustained attention (CPT% Omissions)						
Intercept → ADHD Intercept	4.38 (5.77)	0.76	0.48	0.81 (1.18)	0.69	0.08
Intercept → ADHD Slope	−2.98 (4.44)	−0.67	−0.48	−1.34 (1.78)	−0.75	−0.21
Slope → ADHD Slope	1.04 (1.04)	1.01	0.26	0.12 (0.46)	0.26	0.03
Response inhibition (CPT% Commissions)						
Intercept → ADHD Intercept	0.008 (0.006)	1.26	0.11	0.002 (0.004)	0.66	0.03
Intercept → ADHD Slope	0.003 (0.006)	0.59	0.07	0.008 (0.006)	1.28	0.15
Slope → ADHD Slope	0.01 (0.004)	2.39*	0.27	0.004 (0.005)	0.78	0.09
Working memory (digit span backwards)						
Intercept → ADHD Intercept	−0.18 (0.47)	−0.39	−0.17	−0.03 (0.08)	−0.41	−0.02
Intercept → ADHD Slope	0.44 (1.01)	0.44	0.57	0.11 (0.11)	0.98	0.10
Slope → ADHD Slope	−0.29 (0.84)	−0.40	−0.35	0.01 (0.08)	0.12	0.01

RCFT, Rey-Osterrieth Complex Figure Test; ADHD, attention-deficit/hyperactivity disorder; CPT, Continuous Performance Test; EF, executive function.
*$p < 0.05$,
**$p < 0.01$;
***$p < 0.001$.

scores decreased across development, the faster inattentive symptoms decreased (see Table 2). Symptom intercept ($\sigma^2 = 0.12$, $p < 0.001$) and slope ($\sigma^2 = 0.32$, $p < 0.001$) were again significant, suggesting additional influences on the rate of symptom change.

Sustained attention (CPT omissions). Hyperactive-impulsive symptoms: This model adequately fit the data, $\chi^2(22, N = 228) = 21.74$, *ns*, CFI = 0.934, SRMR = 0.062, RMSEA = 0.000, $CI_{0.90}$ {0.000, 0.054}. Baseline CPT omissions scores were not related to baseline hyperactive-impulsive symptoms or rate of hyperactive-impulsive symptom change. Rate of CPT omissions change was not related to hyperactive-impulsive symptom change (*p*s > 0.25). Variances of symptom intercept ($\sigma^2 = 0.08$) and slope factors ($\sigma^2 = .17$) were not significant, indicating this model adequately captured individual variability in these growth curve parameters.

Inattentive symptoms: This model also adequately fit the data, $\chi^2(22, N = 228) = 29.09$, *ns*, CFI = 0.938, SRMR = 0.057, RMSEA = 0.038, $CI_{0.90}$ {0.000, 0.071}. Baseline CPT omission errors were not associated with either baseline inattentive symptoms or rate of symptom change. Rate of CPT omissions change was also not predictive of inattentive symptom change (*p*s > 0.22). Symptom intercept ($\sigma^2 = 0.11$, $p < 0.001$) and slope ($\sigma^2 = 0.36$, $p < 0.01$) variances were significant.

Response inhibition (CPT commissions). Hyperactive-impulsive symptoms: This model showed a marginal fit to the data, $\chi^2(22, N = 228) = 27.12$, *ns*, CFI = .889, SRMR = 0.052, RMSEA = 0.032, $CI_{0.90}$ {0.000, 0.067}, with the CFI being below its recommended cut-off (Hu & Bentler, 1999; Kline, 2005). Therefore, the following should be interpreted cautiously. There were no significant associations between baseline CPT commission errors and hyperactive-impulsive symptoms in childhood or their rate of change (*p*s > 0.10). Rate of change in CPT commissions scores was predictive of hyperactive-impulsive symptom change, indicating that hyperactive-impulsive symptoms decreased more rapidly as a function of decreases in CPT commission errors. Variances of symptom intercept ($\sigma^2 = 0.13$, $p < 0.01$) and slope ($\sigma^2 = 0.19$, $p < 0.001$) were both significant.

Inattentive symptoms: This model did not fit the data well, $\chi^2(23, N = 229) = 42.63$, $p < 0.01$, CFI = 0.852, SRMR = 0.046, RMSEA = 0.064, $CI_{0.90}$ {0.034, 0.093}. Thus, we did not interpret the findings.

Working memory (Digits Backwards). Hyperactive-impulsive symptoms: The model demonstrated an adequate fit to the data, $\chi^2(22, N = 228) = 18.44$, *ns*, CFI = 0.947, SRMR = 0.042, RMSEA = 0.000, $CI_{0.90}$ {0.000, 0.045}. There were no significant associations between the Digits Backwards intercept and the hyperactive-impulsive intercept or slope. Rate of change for Digits Backwards was not predictive of hyperactive-impulsive symptom change (*p*s > 0.33). The variance of the latent symptom intercept factor was significant ($\sigma^2 = 0.20$, $p < 0.001$), although the variance of the latent slope was not ($\sigma^2 = 0.32$, *ns*).

Inattentive symptoms: This model fit the data exceptionally well, $\chi^2(22, N = 228) = 22.19$, *ns*, CFI = 0.966, SRMR = 0.042, RMSEA = 0.006, $CI_{0.90}$ {0.000, 0.056}. Baseline Digits Backwards did not predict baseline inattentive symptoms or the rate of symptom change. Rate of change for Digits Backward was not associated with rate of change for inattentive symptoms (all *p*s > 0.16). Variances of both intercept ($\sigma^2 = 0.10$, $p < 0.001$) and slope ($\sigma^2 = .32$, $p < 0.01$) factors were significant.

Discussion

In this unprecedented investigation, we found that EF abilities improved in females with and without ADHD, with trajectories differing by childhood ADHD status in some cases, controlling for key baseline externalizing comorbidities. In addition, in contrast to previous studies examining associations between ADHD symptom change and change in specific EF domains (e.g. Vaughn et al., 2011), we found that those who showed greater improvement on a global EF measure showed greater decline in both the inattentive and hyperactive-impulsive domains, and those who showed greater improvement on a measure of response inhibition showed greater decline in hyperactive-impulsive symptoms. Associations between rates of change on EF and symptom variables were not significant for the measures of sustained attention or working memory.

The developmental trajectory of some EF variables differed between females with childhood ADHD and comparisons: Those with ADHD showed greater improvement (i.e. steeper slopes) on a global EF measure and a measure of sustained attention. However, rates of response inhibition and working memory improvement did not differ between the groups. In the two models showing this moderated effect, those with the greatest EF impairment in childhood (i.e. the ADHD group) revealed the greatest improvement over time, potentially reflecting an artifact of regression toward the mean. That is, because the ADHD group was most impaired in childhood, they had more room to improve in their global EF and sustained attention scores. Alternatively, this pattern may reflect the heterogeneity inherent to ADHD, with the possibility of subgroups of females with ADHD who exhibit high levels of EF

impairment in childhood and rapid symptom reduction. It is noteworthy that at least one previous study has found improvement in neuropsychological abilities over time to be more pronounced for children with ADHD than controls (Drechsler et al., 2005). Continued follow-up of the present sample will allow for a more complete understanding of developmental trajectories, moderators of EF change, identification of differences in patterns between EF subdomains, and exploration of potential subgroups that can be distinguished by patterns of EF and symptom change.

Previous longitudinal studies in females with ADHD have shown that EF impairments persist into adulthood (Biederman et al., 2007; Hinshaw et al., 2002, 2007; Miller, Ho, et al., 2012), but many of these studies have not evaluated rates of change per se. Neuroimaging studies have indicated that brain development may occur at different rates in individuals with ADHD (Castellanos et al., 2002) and are suggestive of a delay in brain development (Shaw et al., 2007), with such delays ranging from 3–5 years. A persistent question concerns whether individuals with ADHD eventually attain similar levels of neurocognitive (and other) functioning to those without ADHD. A recent cross-sectional investigation in the present sample found that those with childhood ADHD continued to show impairments in response inhibition, working memory, and global EF (but not sustained attention) relative to comparisons in young adulthood, regardless of persistence of diagnosis (Miller, Ho, et al., 2012). These findings are important given that young adulthood is a time during which the PFC is reaching its developmental endpoint. The present findings suggest that there may not be a consistent pattern of delayed development across EF subdomains in females with ADHD, and rates of neurocognitive improvement may vary depending upon the construct being evaluated, with dramatic improvements in some areas and slower change (and persistent impairment) in others, relative to those without ADHD.

Although some studies have examined EF impairments in individuals with persistent versus remitted ADHD, few have examined associations between changes in EF and changes in dimensional measures of ADHD symptoms. This was a major aim of the current investigation, to identify potential mechanisms underlying symptom improvement. In this study, those who showed the greatest improvement on a global EF measure also showed the greatest reduction in both inattentive and hyperactive-impulsive symptoms across development; this model fit the data well. In addition, in contrast to previous studies (i.e. Vaughn et al., 2011), those who showed the greatest improvement on a response inhibition task showed the greatest reduction in hyperactive-impulsive symptoms, although notably this model showed only a marginal fit to the data. These findings suggest the potential relevance of neuropsychological variables in the progression of symptom change. Notably, relative to other EF measures, the global EF measure that was associated with ADHD symptom change has best distinguished the ADHD and comparison groups in previous investigations (e.g. Hinshaw et al., 2002, 2007; Miller, Ho, et al., 2012) and best predicted functional outcomes in adolescence (Miller & Hinshaw, 2010) and young adulthood (Miller, Nevado, et al., 2012). Despite evidence of at least partial separability of EF components (e.g. Miyake et al., 2000), our findings suggest the potential importance of using measurement strategies that capture the integrated nature of EF abilities, at least with respect to clinical relevance. That is, the combination of impairments across multiple domains of EF may best account for symptom reduction over time. Larger samples may be required to model trajectories of EF subdomains with greater precision and sensitivity.

Although capturing the integrated nature of EF abilities may be of great clinical relevance, it provides little basis for understanding core neuropsychological impairments in ADHD. With respect to EF subdomains, previous theoretical models of ADHD have highlighted response inhibition (Barkley, 1997) and working memory (Rapport, Chung, Shore & Isaacs, 2001) as core deficits. In addition, it has been suggested that simultaneous attenuation of EF deficits and symptom improvement may indicate an epiphenomenal deficit rather than a core impairment (see Carr et al., 2006). Using this framework, our findings are in contrast to the response inhibition model, in that change in response inhibition was associated with hyperactive-impulsive symptom change over time. It is also possible, however, that the measurement overlap between response inhibition and hyperactive-impulsive symptoms could account for this finding. On the other hand, working memory trajectories did not differ between groups and were not associated with symptom change, lending some support to the notion that a deficit in working memory may be core to the ADHD syndrome. This result is in contrast to findings from studies using categorical variables (i.e. persisters, remitters) to evaluate group differences (e.g. Halperin et al., 2008), highlighting the importance of also utilizing dimensional measurement strategies. Future studies should examine still other subdomains of EF (e.g. set shifting, planning, fluency; see Pennington & Ozonoff, 1996), contrasted with response inhibition and working memory, to evaluate core deficits versus epiphenomena, and identify specific domains that may be particularly important to harness for treatments aimed at reducing ADHD symptoms.

Although few investigations have prospectively followed large samples of females with ADHD, the present findings are generally consistent with previous studies of individuals with ADHD that demonstrate EF impairments at various development time

points (e.g. Biederman et al., 2007; Hinshaw et al., 2002; Miller, Ho, et al., 2012; Nigg, Butler, Huang-Pollock & Henderson, 2002; Seidman 2006). We extend these findings by examining growth trajectories of EF abilities and their associations with ADHD symptom trajectories in the understudied population of females with childhood-diagnosed ADHD. A continued focus on the female ADHD phenotype and its developmental course is of great importance given potential unique risks for this group (Hinshaw et al., 2012; Rucklidge, 2010). Continued evaluation of associations between changes in EF and changes in ADHD symptoms could lead to treatment development work aimed at simultaneously addressing both domains.

Limitations include clinical ascertainment of the sample; whether our results would be similar in a community sample of females with ADHD is unclear. In addition, because the sample is entirely female, comparisons between sexes are impossible. Although the retention rates for follow-up assessments were high, home visits, equipment failure, and missed tests for some participants reduced the amount of EF data available at follow-up. Additionally, the data analytic strategy employed resulted in a large degree of missing data. Whereas maximum-likelihood procedures were used to mitigate this, it remains a significant limitation. Larger samples may help clarify potential relations between EF variables and symptom change. Finally, we did not include covariates in models of associations between EF and symptom growth trajectories, as this would have resulted in a subject-to-variable ratio untenable for SEM procedures (Kline, 2005). Thus, we cannot rule out the possibility that comorbid conditions – or additional unmeasured factors – account for the association between change in EF scores and change in ADHD symptoms over time. Relatedly, IQ was not included in any of the models for both theoretical and pragmatic (i.e. subject-to-variable ratio) reasons. Thus, we cannot conclude whether results are EF-specific or related to general cognitive ability; this is an important area for future investigation.

Overall, the present investigation provides insight into (a) the development of EF abilities in females with ADHD, (b) how such development is associated with symptom change, and (c) the female ADHD phenotype. Females with ADHD appear to exhibit variable improvement in EF abilities over time, with greater improvement than comparisons on some measures and similar improvement on others. Those who showed the greatest improvement over time on a global EF measure and a measure of response inhibition also exhibited the greatest reduction in ADHD symptoms, indicating that changes in EF abilities may, to some extent, underlie developmental changes in ADHD symptoms and suggesting that neuropsychological functioning may be one relevant area in which to focus future treatment development efforts.

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Key points

- Females with a history of ADHD show variability in patterns of EF improvement over time.
- Changes in EF abilities may underlie developmental changes in ADHD symptoms, providing some insight into core versus secondary deficits.
- Neuropsychological functioning may be one area in which to focus future treatment development efforts.
- A continued focus on the female ADHD phenotype and its developmental course is of great importance given potential unique risks.

Notes

1. Data were analyzed including and excluding participants on stimulant medication during testing. Findings did not differ, thus results are presented based on the full sample.
2. Our analytic approach precluded the further investigation of simple slopes following a significant moderated effect. Procedures for determining the standard error of conditional effects have not yet been developed for the special case when factor loadings are freely estimated (see Curran, Bauer & Willoughby, 2004). Graphical displays of moderated

effects were included for descriptive purposes utilizing online software freely available at http://www.quantpsy.org/ (Preacher, Curran, & Bauer, 2006).
3. RMSEA was computed based upon covariance and mean structures.

References

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th edn). Washington, DC: American Psychiatric Association.

Barkley, R.A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, *121*, 65–94.

Benson, D.F. (1991). The role of frontal dysfunction in attention deficit hyperactivity disorder. *Journal of Child Neurology*, *6*, S9–S12.

Bentler, P.M. (2006). *EQS 6 Structural equations program manual*. Encino, CA: Multivariate Software, Inc.

Biederman, J., Mick, E., & Faraone, S.V. (2000). Age-dependent decline of symptoms of attention deficit hyperactivity disorder: Impact of remission definition and symptom type. *American Journal of Psychiatry*, *157*, 816–818.

Biederman, J., Petty, C.R., Ball, S.W., Fried, R., Doyle, A.E., Cohen, D., … & Faraone, S.V. (2009). Are cognitive deficits in attention deficit/hyperactivity disorder related to the course of the disorder? A prospective controlled follow-up study of grown up boys with persistent and remitting course. *Psychiatry Research*, *170*, 177–182.

Biederman, J., Petty, C.R., Doyle, A.E., Spencer, T., Henderson, C.S., Marion, B., … & Faraone, S.V. (2008). Stability of executive function deficits in girls with ADHD: A prospective longitudinal followup study into adolescence. *Developmental Neuropsychology*, *33*, 44–61.

Biederman, J., Petty, C.R., Fried, R., Doyle, A.E., Spencer, T., Seidman, L.J., … & Faraone, S.V. (2007). Stability of executive function deficits into young adult years: A prospective longitudinal follow-up study of grown up males with ADHD. *Acta Psychiatrica Scandinavica*, *116*, 129–136.

Biederman, J., Petty, C.R., Monuteaux, M.C., Fried, R., Byrne, D., Mirto, T., … & Faraone, S.V. (2010). Adult psychiatric outcomes of girls with attention deficit hyperactivity disorder: 11-year follow-up in a longitudinal case-control study. *American Journal of Psychiatry*, *167*, 409–417.

Bollen, K.A., … & Curran, P.J. (2006). *Latent curve models: A structural equation modeling approach*. Wiley Series in Probability and Mathematical Statistics. New York: Wiley.

Carr, L.A., Nigg, J.T., & Henderson, J.M. (2006). Attentional versus motor inhibition in adults with attention-deficit/hyperactivity disorder. *Neuropsychology*, *20*, 430–441.

Castellanos, F.X., Lee, P.L., Sharp, W., Jeffries, N.O., Greenstein, D.K., Clasen, L.S., … & Rapoport, J.L. (2002). Developmental brain trajectories of brain volume abnormalities in children and adolescents with ADHD. *Journal of the American Medical Association*, *288*, 1740–1748.

Conners, C.K. (1995). *Conners' continuous performance test computer program: User's manual*. Toronto, Ont., Canada: Multi-Health Systems.

Curran, P.J., Bauer, D.J., & Willoughby, M.T. (2004). Testing and probing main effects and interactions in latent curve analysis. *Psychological Methods*, *9*, 220–237.

Dennis, M., Francis, D.J., Cirino, P.T., Schachar, R., Barnes, M.A., & Fletcher, J.M. (2009). Why IQ is not a covariate in cognitive studies of neurodevelopmental disorders. *Journal of the International Neuropsychological Society*, *15*, 331–343.

Drechsler, R., Brandeis, D., Földényi, M., Imhof, K., & Steinhausen, H.C. (2005). 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*, *46*, 824–836.

Halperin, J.M., & Schulz, K.P. (2006). Revisiting the role of the prefrontal cortex in the pathophysiology of attention-deficit/hyperactivity disorder. *Psychological Bulletin*, *132*, 560–581.

Halperin, J.M., Trampush, J.W., Miller, C.J., Marks, D.J., & Newcorn, J.H. (2008). Neuropsychological outcome in adolescents/young adults with childhood ADHD: Profiles of persisters, remitters, and controls. *Journal of Child Psychology & Psychiatry*, *49*, 958–966.

Helmes, E. (2000). Learning and memory. In G. Groth-Marnat (Ed.), *Neuropsychological assessment in clinical practice: A guide to test interpretation and integration* (pp. 293–334). New York: Wiley.

Hinshaw, S.P. (2002). Preadolescent girls with attention-deficit/hyperactivity disorder: I. Background characteristics, comorbidity, cognitive and social functioning, and parenting practices. *Journal of Consulting & Clinical Psychology*, *70*, 1086–1098.

Hinshaw, S.P., Carte, E.T., Fan, C., Jassy, J.S., & Owens, E.B. (2007). Neuropsychological functioning of girls with attention-deficit/hyperactivity disorder followed prospectively into adolescence: Evidence for continuing deficits?. *Neuropsychology*, *21*, 263–273.

Hinshaw, S.P., Carte, E.T., Sami, N., Treuting, J.J., & Zupan, B.A. (2002). Preadolescent girls with attention-deficit/hyperactivity disorder: II. Neuropsychological performance in relation to subtypes and individual classification. *Journal of Consulting and Clinical Psychology*, *70*, 1099–1111.

Hinshaw, S.P., Owens, E.B., Sami, N., & Fargeon, S. (2006). Prospective follow-up of girls with attention-deficit/hyperactivity disorder into adolescence: Evidence for continuing cross-domain impairment. *Journal of Consulting and Clinical Psychology*, *74*, 489–499.

Hinshaw, S.P., Owens, E.B., Zalecki, C., Perrigue-Huggins, S., Nevado-Montenegro, A., Schrodek, E., & Swanson, E.N. (2012). Prospective follow-up of girls with attention-deficit/hyperactivity disorder into early adulthood: Continuing impairment includes elevated risk for suicide attempts and self-injury. *Journal of Consulting and Clinical Psychology*, online first, *80*, 1041–1051.

Hu, L., & Bentler, P.M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*, *6*, 1–55.

Kessler, R.C., Green, J.G., Adler, L.A., Barkley, R.A., Chatterji, S., Faraone, S.V., … & Van Brunt, D.L. (2010). Structure and diagnosis of adult attention-deficit/hyperactivity disorder: Analysis of expanded symptom criteria from the adult ADHD clinical diagnostic scale. *Archives of General Psychiatry*, *67*, 1168–1178.

Kline, R.B. (2005). *Principles and practice of structural equation modeling* (2nd edn). New York: Guilford Press.

Lahey, B.B., Pelham, W.E., Loney, J., Lee, S.S., & Willcutt, E. (2005). Instability of the *DSM-IV* subtypes of ADHD from preschool through elementary school. *Archives of General Psychiatry*, *62*, 896–902.

McGough, J.J., & Barkley, R.A. (2004). Diagnostic controversies in adult attention deficit hyperactivity disorder. *American Journal of Psychiatry*, *161*, 1948–1956.

Miller, M., & Hinshaw, S.P. (2010). Does childhood executive function predict adolescent functional outcomes in girls with and without ADHD? *Journal of Abnormal Child Psychology*, *38*, 315–326.

Miller, M., Ho, J., & Hinshaw, S.P. (2012). Executive functions in girls with ADHD followed prospectively into young adulthood. *Neuropsychology*, *26*, 278–287.

Miller, M., Nevado, A.J., & Hinshaw, S.P. (2012). Childhood executive function continues to predict outcomes in young

adult females with and without childhood-diagnosed ADHD. *Journal of Abnormal Child Psychology*, *40*, 657–668.

Miyake, A., Friedman, N.P., Emerson, M.J., Witzki, A.H., Howerter, A., & Wager, T.D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology*, *41*, 49–100.

Nigg, J.T. (2005). Neuropsychologic theory and findings in attention-deficit/hyperactivity disorder: The state of the field and salient challenges for the coming decade. *Biological Psychiatry*, *57*, 1424–1435.

Nigg, J.T., Butler, K.M., Huang-Pollock, C.L., & Henderson, J.M. (2002). Inhibitory processes in adults with persistent childhood onset ADHD. *Journal of Consulting & Clinical Psychology*, *70*, 153–157.

Osterrieth, P.A. (1944). Le test de copie d'une figure complex [A test of copying a complex figure]. *Archives de Psychologie*, *30*, 206–256.

Pennington, B.F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology & Psychiatry*, *37*, 51–87.

Preacher, K.J., Curran, P.J., & Bauer, D.J. (2006). Computational tools for probing interaction effects in multiple linear regression, multilevel modeling, and latent curve analysis. *Journal of educational and behavioral Statistics*, *31*, 437–448.

Rapport, M.D., Alderson, R.M., Kofler, M.J., Sarver, D.E., Bolden, J., & Sims, V. (2008). Working memory deficits in boys with attention-deficit/hyperactivity disorder (ADHD): The contribution of central executive and subsystem processes. *Journal of Abnormal Child Psychology*, *36*, 825–837.

Rapport, M.D., Chung, K., Shore, G., & Isaacs, P. (2001). A conceptual model of child psychopathology: Implications for understanding attention deficit hyperactivity disorder and treatment efficacy. *Journal of Clinical Child & Adolescent Psychology*, *30*, 48–58.

Rohlf, H., Jucksch, V., Gawrilow, C., Huss, M., Hein, J., Lehmkuhl, U., & Salbach-Andrae, H. (2012). Set shifting and working memory in adults with attention-deficit/hyperactivity disorder. *Journal of Neural Transmission*, *119*, 95–106.

Rucklidge, J.J. (2010). Gender differences in attention-deficit/hyperactivity disorder. *Psychiatric Clinics of North America*, *33*, 357–373.

Sami, N., Carte, E.T., Hinshaw, S.P., & Zupan, B.A. (2003). Performance of girls with ADHD and comparison girls on the Rey-Osterrieth Complex Figure: Evidence for executive processing deficits. *Child Neuropsychology*, *9*, 237–254.

Seidman, L.J. (2006). Neuropsychological functioning in people with ADHD across the lifespan. *Clinical Psychology Review*, *26*, 466–485.

Shaffer, D., Fisher, P., Lucas, C.P., Dulcan, M.K., & Schwab-Stone, M.E. (2000). NIMH Diagnostic Interview Schedule for Children Version IV (NIMH DISC-IV): Description, differences from previous versions, and reliability of some common diagnoses. *Journal of the American Academy of Child & Adolescent Psychiatry*, *39*, 28–38.

Shaw, P., Eckstrand, K., Sharp, W., Blumenthal, J., Lerch, J.P., Greenstein, D., … & Rapoport, J.L. (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *Proceedings of the National Academy of Sciences*, *104*, 19649–19654.

Shaw, P., Lerch, J., Greenstein, D., Sharp, W., Clasen, L., Evans, A., … & Rapoport, J. (2006). Longitudinal mapping of cortical thickness and clinical outcome in children and adolescents with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, *63*, 540–549.

Swanson, J.M. (1992). *Assessment and treatment of ADD students*. Irvine, CA: K.C. Press.

Taylor, L.B. (1969). Localization of cerebral lesions by psychological testing. *Clinical Neurosurgery*, *16*, 269–287.

Tranel, D., Anderson, S.W., & Benton, A. (1994). Development of the concept of "executive function" and its relationship to the frontal lobes. In F. Boller, & J. Grafman (Eds.), *Handbook of neuropsychology* (Vol. *9*, pp. 125–148). New York: Elsevier.

Vaughn, A.J., Epstein, J.N., Rausch, J., Altaye, M., Langberg, J., Newcorn, J.H., … & Wigal, T. (2011). Relation between outcomes on a continuous performance test and ADHD symptoms over time. *Journal of Abnormal Child Psychology*, *39*, 853–864.

Wechsler, D. (1991). *Manual for the Wechsler Intelligence Scale for children* (3rd edn). New York: Psychological Corporation/Harcourt Brace.

Willcutt, E.G., Doyle, A.E., Nigg, J.T., Faraone, S.V., & Pennington, B.F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. *Biological Psychiatry*, *57*, 1336–1346.

Yuan, K.-H., & Bentler, P.M. (1998). Robust mean and covariance structure analysis. *British Journal of Mathematical and Statistical Psychology*, *70*, 147–167.

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