

Executive Functions and ADHD in Adults: Evidence for Selective Effects on ADHD Symptom Domains

Joel T. Nigg, Gillian Stavro, Mark Ettenhofer, David Z. Hambrick, Torri Miller, and John M. Henderson
Michigan State University

Dual-process models of attention-deficit/hyperactivity disorder (ADHD) suggest that both executive functioning and regulatory functions (e.g., processing speed) are involved and that executive function weaknesses may be associated specifically with symptoms of inattention–disorganization but not hyperactivity–impulsivity. Adults aged 18–37 (105 with ADHD, 90 controls) completed a neuropsychological battery. The ADHD group had weaker performance than did the control group ($p < .01$) on both executive and speed measures. Symptoms of inattention–disorganization were uniquely related to executive functioning with hyperactivity–impulsivity controlled. Inattention was associated with slower response speed, and hyperactivity–impulsivity with faster output speed. Results were not accounted for by IQ, age, gender, education level, or comorbid disorders. Findings are discussed in terms of developmental and dual-process models of ADHD leading into adulthood.

Keywords: attention-deficit/hyperactivity disorder, ADHD, executive functioning, regulatory functions, dual-process model

As a group, children with attention-deficit/hyperactivity disorder (ADHD) have established and replicated weaknesses in neuropsychological *executive functioning* (EF), defined broadly as the ability to regulate behavior to context and maintain a response set (Barkley, 1997; Nigg, 2001; Pennington & Ozonoff, 1996). Discussion continues about whether executive deficits may represent a proximal causal deficit in the disorder (Barkley, 1997; Nigg, 2001; Sergeant et al., 1999). At the same time, the expression of ADHD in adults has become a central focus of investigation and controversy (Faraone et al., 2000; Sachdev, 1999), particularly now that longitudinal data have established that a substantial percentage of children with ADHD continue to show persistent problems into adulthood, including notable impairment in occupational and social functioning (Barkley, 2002; Faraone et al., 2000; Murphy & Barkley, 1996).

Whether EF deficits are detected in adults with persistent and impairing ADHD symptoms is critical to theories concerning the role of executive functioning in the disorder and how executive deficits may relate to the course of the disorder over development. Physical and neurological development, including myelination of the frontal cortices, is relatively complete by young adulthood

(Benes, 2001). Developmental theories suggest that frontally mediated operations, such as those described in most models of executive functioning, continue to become more efficient during this period. ADHD symptoms either decrease or take somewhat modified forms of expression with maturation into early adulthood between the ages of 18 and 30 years (Faraone et al., 2000). A key question is whether executive neurocognitive deficits occur in ADHD during early adulthood, consistent with the continued centrality of EF to the disorder, or whether they may normalize even as impairment continues, suggesting EF is a secondary feature only.

Accordingly, several studies have examined neuropsychological executive dysfunction in adults with ADHD (Corbett & Stanczak, 1999; Lovejoy et al., 1999; Weyandt et al., 1995). Unfortunately, results have been rather mixed. Although several studies have found some type of EF weakness in adults with ADHD on tasks assessing set shifting (Gansler et al., 1998), focused attention (Sandson et al., 2000), and response inhibition—including the antisaccade (Nigg et al., 2001) and stopping tasks (Aron et al., 2003)—several studies failed to find executive deficits (Johnson et al., 2001; Seidman et al., 1998; Weyandt et al., 1995), although Seidman et al. (1998) found deficits on some measures that require intact executive control. In one of the largest studies to date, Murphy, Barkley, and Bush (2001) examined 105 adults with ADHD and 64 controls. The ADHD group had deficits on several measures of executive functioning.

On the other hand, relatively consistent findings have emerged in adults with ADHD regarding slow response speed (Johnson et al., 2001) and continuous performance task errors (Epstein et al., 2001; Gansler et al., 1998; Seidman et al., 1998), all suggestive of difficulties in output regulation that may be related to vigilance, alertness, (Berger & Posner, 2000), activation, effort, or other state regulation mechanisms (Sergeant et al., 1999).

In all, several issues emerge from the existing literature on executive and related neurocognitive functioning in adults with

Joel T. Nigg, Gillian Stavro, Mark Ettenhofer, David Z. Hambrick, Torri Miller, and John M. Henderson, Department of Psychology, Michigan State University.

This research was supported by National Institute of Mental Health Grant R01-MH63146 to Joel Nigg, John Henderson, and Fernanda Ferreira. We thank the participants who made this study possible; Colleen Schmitt, Andrew Homa, Timothy Goth-Owens, Julie Ann Stawicki, and Linna Leslie for invaluable assistance in carrying out this work; and Michelle Martel, Laurie Carr, and Steve Faraone for helpful discussions about earlier versions of this article.

Correspondence concerning this article should be addressed to Joel Nigg, Department of Psychology, Room 115-C, Psychology Building, Michigan State University, East Lansing, MI 48824-1116. E-mail: nigg@msu.edu

ADHD. First, what accounts for the mixed findings on executive measures? Several explanations are possible, including (a) generally small sample sizes (most studies had $n < 60$ per group, and many had $n < 40$ per group), resulting in limited power to detect the potentially smaller effect sizes that might be expected in adults if they have partially compensated for their executive problems; (b) limited reliability of some executive measures, which might be addressed by a latent variable or factor-based approach (Kuntsi, Stevenson, Oosterlaan, & Sonuga-Barke, 2001); (c) effects may be present only in some domains of EF; and (d) comorbidity may explain positive findings. Indeed, specificity of effects to ADHD is a key question (Sergeant et al., 1999), especially for adults (Gallagher & Blader, 2001) for whom executive functioning deficits may be a general marker of psychopathology. It is essential to clarify whether any deficits hold when major comorbid disorders are controlled either by exclusion or by covariance.

Second, ADHD as defined in the *DSM-IV* (American Psychiatric Association, 1994) is characterized by problems in two behavioral domains—inattention-disorganization and hyperactivity-impulsivity—leading *DSM-IV* to specify three behavioral subtypes: primarily inattentive (ADHD-I), primarily hyperactive-impulsive (ADHD-H), and combined (ADHD-C). Yet, as children mature into adulthood, developmental changes in ADHD expression may occur, with reductions in overt hyperactivity but continued problems in organization and attention (Faraone et al., 2000). As a result, theorists have begun to suggest that EF weakness may be particularly relevant to inattention-disorganization, rather than hyperactivity-impulsivity per se (e.g., Sonuga-Barke, 2002). If so, executive deficits could be masked by failure to separately analyze these two partially separable behavioral domains. It appears to be theoretically important to clarify whether executive deficits, if present in adults with ADHD, are more strongly related to the inattentive-disorganization domain of problems rather than to hyperactivity-impulsivity. Whereas some initial studies of ratings data support this supposition (Nigg et al. 2002b), studies of laboratory executive measures that address this question are few.

However, it is unclear that the *DSM-IV* subtypes of ADHD should be expected to be neuropsychologically distinguishable in adults. In childhood, these subtypes, especially the ADHD-C versus ADHD-I subtypes, have distinct external and clinical correlates (Milich et al., 2001). Theories generally predict that executive dysfunction will be characteristic of the ADHD-C subtype and one study did show differences in subtypes on response inhibition in children (Nigg, Blaskey, Huang-Pollock, & Rappley, 2002a). However, for the most part, the subtypes proposed in the *DSM-IV* have yielded ambiguous, limited, or at best partial neuropsychological distinctions in children (Chhabildas et al., 2001; Faraone, Biederman, Weber, & Russell, 1998; Hinshaw, Carte, Sami, Treuting, & Zupan, 2002; Klorman et al., 1999; Nigg et al., 2002a), so it is unclear whether this distinction will be informative with regard to neuropsychological effects in adults (Dinn et al., 2001; Gansler et al., 1998; Murphy et al., 2001). Indeed, from a developmental point of view, subtypes might not be preserved as validly distinct, because in adulthood symptoms change with maturation, placing more importance on inattentive symptoms as noted earlier. Crucially, then, many prior studies relied on the *DSM-III-R* (American Psychiatric Association, 1987) or on other definitions of ADHD, and few if any separately considered these two behavioral domains as currently conceptualized in the *DSM-*

IV. We sought to clarify relations to the behavioral domains, considering subtypes a secondary focus.

Third, from a neuropsychological point of view, contemporary dual-process models stipulate that ADHD overall likely involves more than one distributed neural system from the broad set of systems involved in regulatory control (Berger & Posner, 2000; Sonuga-Barke, 2002; Nigg, Goldsmith, & Sachek, 2004). One system is thought to be a frontal-striatal network involved in EFs, which provides for response inhibition-suppression, protection of working memory, focusing of attention, temporal organization of behavior, and related abilities and functions (Barkley, 1997; Berger & Posner, 2000; Pennington & Ozonoff, 1996; Posner & Peterson, 1990). A second network is related to vigilance or alertness (Berger & Posner, 2000; Posner & Peterson, 1990; Sandson et al., 2000) or, in a related but somewhat alternative view, to activation (response readiness and preparation of motor output) or effort (Sergeant et al., 1999). In either conception, the second weakness would be expected to result in difficulty mobilizing rapid responses, leading to slow response on rapid timed tasks. As we noted, this particular response profile is well recognized in children with ADHD and is now fairly well replicated in adults with ADHD. Broadly speaking then, when we consider a neuropsychological battery, we viewed it as essential to distinguish response or output speed (as an index of alertness, vigilance, or activation) from executive control per se (here intended to involve the four domains of set shifting, planning, response inhibition, and working memory; Pennington & Ozonoff, 1996). However, we planned to empirically evaluate this approach.

Fourth, the nature of the EF construct has faced extensive discussion. Of particular interest in the field is whether it is best thought of as multicomponential only, or as also including a higher order executive ability that may be captured as a latent factor. Whereas there is considerable evidence for multicomponentiality as one aspect of EFs (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000; Murphy et al., 2001; Pennington & Ozonoff, 1996), a number of considerations also commend the possibility of a single latent ability that may converge for at least a substantial subset of executive measures. Such measures have correlated positively and moderately in a number of studies (Delis, Kaplan, & Kramer, 2001; Hanes, Andrewes, Smith, & Pantelis, 1996), although this is not always the case (Lowe & Rabbitt, 1998), and many such tasks may all be impaired in those with frontal lobe injury (Duncan et al., 1997). Use of composite or factor scores, if justified, may provide more robust and reliable indices of correlates to ADHD than individual measures, which can have varying reliability.

The present study sought to address the question of EFs in a relatively large and inclusive sample of ADHD adults using a broad clinical neuropsychological executive battery. Our conceptual model (a) viewed EFs as including multiple components, but as potentially including also a higher order general factor that would capture at least a substantial subset of the EF construct, and (b) anticipated a second cognitive domain, related to a vigilance network (Posner & Peterson, 1990) or state regulation (Sergeant et al., 1999) partially distinct from EFs and indexed by output speed. Our primary hypotheses were that (a) ADHD would be related to weaker performance on executive tasks and on a composite executive factor if one could be identified, and (b) that executive weakness would be specifically related to symptoms of inattention-disorganization rather than hyperactivity-impulsivity

(Sonuga-Barke, 2002). Secondly, we evaluated ADHD subtype effects and potential gender differences (Katz et al., 1998). The main results are presented without covarying IQ or comorbid disorders. However, to assure that results were not due to their shared overlap with EFs (Miller & Chapman, 2001), we rechecked results after covarying IQ and comorbid disorders (i.e., antisocial personality disorder, substance dependence, and mood and anxiety disorders).

Method

Participants

Recruitment. We deliberately sought to recruit adults who met criteria for varying subtypes of ADHD to maximize variation in the two behavioral domains of interest and in order that secondary analyses could look at those effects. However, we were prepared to study the adult ADHD sample as a whole in view of both the relatively weak evidence for important subtype differences in neuropsychological performance, especially in adults, and our interest in the behavioral symptom domains. Prospective participants were recruited from the community via public advertisements and then evaluated in a standard multistage screening and diagnostic evaluation procedure. Separate advertisements were used to recruit possible ADHD participants (these ads asked for volunteers who had been told or believed they had difficulties with inattention, impulsivity, overactivity, ADD, or ADHD) and possible controls (which asked for volunteers in good health for a study of adult development). In the multistage screening procedure, prospective participants contacted the project office, at which point key rule outs were checked by telephone (ages 18–40, no sensory-motor handicap, no neurological illness, and native English speaking). Eligible participants were then scheduled for the diagnostic visit wherein they completed semistructured clinical interviews.

Assessment of ADHD symptoms by self and informant reports. Assessment of ADHD in adults requires retrospective assessment of their childhood ADHD status to establish childhood onset and inclusion of informant interviews to verify symptoms and impairment (Wender, Wolf, & Wasserstein, 2001). A retrospective Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS; Puig-Antich & Ryan, 1986) was administered by a masters-level clinician after extensive training, following previously published procedures for assessing adults (Biederman et al., 1992; Biederman, Faraone, Keenan, Kneec, & Tsuang, 1990) to assess their childhood ADHD, conduct disorder (CD), and oppositional defiant disorder (ODD) symptoms and impairment. Because self-report recall of these symptoms may lead to underreporting (Murphy & Barkley, 1996), the same semistructured modules were administered to the participant and an informant who had known them as a child (usually a parent) to ensure cross-informant convergence. The informant reported on the participant's childhood behaviors via an ADHD rating scale and a retrospective K-SADS ADHD module adapted to be appropriate for an informant interview.

Current (adult) ADHD symptoms were assessed by self-report and by interview with a second informant, who knew the participant well currently (Wender et al., 2001). We again used K-SADS ADHD questions worded appropriately for current adult symptoms following Biederman et al. (1992). This interview was supplemented with the Barkley and Murphy (1998) Current ADHD Symptoms Rating Scale. To ensure that ADHD participants exceeded normative cutoffs for level of ADHD symptoms, participants also completed the Conners' Young Adult ADHD Rating Scale (Conners, Erhardt, & Sparrow, 1999), Achenbach's (1991) Young Adult Self-Report Scale, and the Brown (1996) Adult ADHD Rating Scale. The informant (usually a spouse or friend) completed the Conners peer rating as well as Barkley and Murphy peer ratings on adult symptoms, and a brief screen of antisocial behavior, drug and alcohol use. They also completed a structured interview about the target participant's current ADHD symptoms with the modified K-SADS for current symptoms. All

informant interviews were conducted by telephone after appropriate consent procedures; informants were paid for their time.

Assessment of comorbid psychopathology. Comorbid Axis I disorders were assessed with the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1995) administered by a trained masters-level clinician. Assessed were major depressive disorder, dysthymic disorder, bipolar disorder, substance abuse and dependence, psychotic symptoms, generalized anxiety disorder (GAD), posttraumatic stress disorder, obsessive-compulsive disorder, panic disorder, agoraphobia, simple phobia, social phobia, and eating disorders. Antisocial personality disorder and other personality disorders were assessed with the SCID-II. We covaried common comorbid conditions most likely to account for findings. Autistic disorder was screened by the clinician using added symptom questions and was a rule out.

Establishment of best estimate diagnosis for ADHD. A diagnostic team that included a licensed clinical social worker, a licensed clinical psychologist, and a board certified psychiatrist then arrived at a "best estimate" diagnosis (Faraone, 2000) as follows. Each team member independently reviewed all available information from SCID, K-SADS, and rating scales to arrive at a clinical judgment about ADHD present or absent, ADHD subtype, and comorbid disorders. Their rates of agreement were then computed for all cases that they reviewed (including cases that were eventually ruled ineligible for study participation), and then disagreements were conferenced to arrive at a consensus diagnosis. Agreement between the two primary clinicians on the team (the psychologist and the psychiatrist) for presence or absence of ADHD (any type) was satisfactory ($k = .80$). Likewise, their agreement on ADHD subtype (combined, inattentive, or hyperactive) in childhood and adulthood was also adequate, ranging from $k = .74$ to $.85$, with the exception of current ADHD-H, which had $k = .74$ despite a high percentage of agreement (85%) due to its small n (14). Finally, the clinicians' interrater reliability for comorbid disorders was excellent (past major depression, $k = .96$; any current anxiety disorder, $k = 0.98$; antisocial personality disorder, $k = 0.93$; substance or alcohol dependence, $k = 0.97$).

Note that because no agreed upon criteria exist for ADHD in adults, the team followed DSM-IV criteria for children by requiring the same symptoms in adults but allowed the adult ADHD-residual category as well because it was allowed in earlier editions of the DSM. The DSM-IV criteria regarding comorbidity were carefully followed in all cases, however, so that although comorbid disorders were diagnosed when present, ADHD was not diagnosed if clinicians judged that symptoms were better explained by a co-occurring mood or other major disorder. This was intended to provide some control against obtaining a sample with extreme levels of comorbid disorders. Onset in childhood was required; however, to enhance validity in light of concern about the age of 7 onset criteria in retrospective assessments (Barkley & Biederman, 1997), we operationalized this as a finding that two reporters (both participant and their retrospective informant) independently reported that the age of onset was age 12 or younger. Clinical interviewers rated and noted evidence of impairment when interviewing participants and their informants; the clinical team then required evidence of notable impairment for the ADHD diagnosis. Sixty-five percent of the ADHD sample reported that they had been previously diagnosed with or treated for ADHD as children or adolescents (agreement between self and reporter, $k = .91$).

Exclusionary criteria. Potential participants were excluded from both groups if they had a current major depressive or manic-hypomanic episode, current substance dependence preventing sober testing, history of psychosis, history of autism, full-scale IQ (FSIQ) <75, history of head injury with loss of consciousness, sensory-motor handicap, neurological illness, native language not English, or currently prescribed antipsychotic, antidepressant, or anticonvulsant medications. For the control group, additional exclusions were antisocial or borderline personality disorder, past bipolar disorder, or a previously diagnosed learning disorder. Other psychiatric disorders were free to vary.

Medication washout. Participants prescribed psychostimulant medications (20% of the ADHD group; medications were Adderall, Ritalin, Concerta, and Focalin in this sample) were tested after a minimum of 24 hr (for short-acting preparations) to 48 hr washout (for long-acting preparations); actual mean washout time was 63.8 hr.

Final sample. Four hundred twenty-four adults passed initial screen and completed the screening rating scale and the diagnostic screen visit. The diagnostic procedures qualified 195 of them (46%) between the ages of 18 and 37 for the study, grouped into an ADHD group and a non-ADHD control group. Primary reasons for rule out after the initial screen were failure of self-informant convergence on symptoms, current major depression, or taking long-acting psychoactive medications that would affect neuropsychological test performance.

Neuropsychological Test Battery

Full Scale IQ was estimated with a five subtest short form of the Wechsler Adult Intelligence Scale (3rd. ed.; WAIS-III; Wechsler, 1997): Picture Completion, Vocabulary, Similarities, Arithmetic, and Matrix Reasoning. Reading was assessed with the Wide Range Achievement Test (3rd. ed.; WRAT-III; Wilkinson, 1993). The remainder of the battery was constructed to assess the EF construct, following the four component model suggested by Pennington and Ozohoff (1996): set shifting, interference control, working memory and planning, and response inhibition. The tests were administered in the same fixed order to all participants.

Trail Making Test. The Trail Making Test (*Trails*) is a widely used, timed paper-and-pencil test consisting of two parts (Reitan, 1958). Part A requires the participant to draw a line connecting numbered circles in sequential order. Part B requires the participant to draw a line connecting numbered and lettered circles in alternating sequential-alphabetical order. Scores on each part of the Trails test are determined by the time required to complete each trial. Whereas performance on Part A depends largely on psychomotor speed and visual search abilities, Part B places additional demands on the participant's working memory and cognitive flexibility by requiring the participant to maintain two mental sets and alternate between them (Arbuthnott & Frank, 2000). We viewed Trails A time as a measure of motor speed. To index set shifting, we created a Trails B time residual score variable by regressing Trails B on Trails A. Individuals who have sustained frontal lobe damage show deficits on this task (Stuss et al., 2001).

The Stroop Color-Word Test. The Stroop Test is widely used to measure the ability to shift attention and inhibit interfering information (MacLeod, 1991). Participants are required to rapidly name a list of color words (*blue, red, or green*) in which the color of the text is incongruent with the word itself (Stroop, 1935). Dorsolateral frontal lobe activity was increased during the incongruent color-word condition (Banich et al., 2000). Word reading and color reading scores served as measures of output speed. The Stroop interference score used here was a residual score created by partialling word and color naming speed from the color-word raw score. The (raw) color-word score was not further analyzed.

Wisconsin Card Sorting Test (WCST). The WCST is a widely used computer-administered task assessing working memory, concept formation, and set shifting (Heaton et al., 1993). Up to 64 trials were administered in which the participant sorted cards on the basis of shifting criteria of color, shape, and number. The participant is not told how to sort the cards and instead must deduce the current correct principle with only the computer's feedback (*correct* or *incorrect*). Neuroimaging and other data associate difficulty on this task with injured or underactivated prefrontal cortical regions (Wang, Kakigi, & Hoshiyama, 2001). We analyzed categories completed and perseverative errors as measures of EF. Because of their high correlation ($r = -.60$), they were standardized and averaged (after reversing the categories score).

Logan Stop Task. The Stop Task is a dual-task computer paradigm to assess response suppression or inhibition in a rapid decision context. Procedures were the same as those used by Logan, Schachar, and Tannock (1997). The computer screen displayed an *X* or an *O* on a black-and-white screen, and individuals were required to respond to these stimuli by

pressing designated buttons labeled *X* and *O* as quickly as possible with their dominant hand. They were to withhold responding when they heard a tone. Four blocks of 64 trials were administered following two practice blocks of 32 trials each. We used the tracking version of the stop task, which provides the most valid estimates of stop signal reaction time (RT; Band, Van Der Molen, & Logan, 2003). The time of the stop-signal tone was varied in a stochastic procedure to maintain accuracy at 50% so that stop-signal RT was computed as the difference between stop-signal delay and go speed (Logan, 1994). Performance in adults involves right inferior frontal cortex and basal ganglia (Aron et al., 2003). We calculated stop-signal RT, go RT, and variability of go reaction time (*response variability*) by averaging performances across the last three blocks of trials after cleaning data by the same procedures described by Nigg (1999).

Tower of London (Colorado version; TOL). The TOL is a computer-administered task (Davis & Keller, 2002) that assesses planning ability. Individuals were presented with 10 problems, in which they viewed the target arrangement and matched it by rearranging different colored balls on 3, 4, or 5 different size pegs. The initial starting position was displayed on the left, and the goal position was displayed on the right. Individuals used the computer mouse to move the balls, one at a time, from the starting position to match the final goal position in the fewest possible moves. This task activates prefrontal cortex and associated neural regions (Newman et al., 2003). Total number of moves made was the outcome variable.

Data analysis. We planned to (a) examine our two-factor conception of the neuropsychological battery with confirmatory factor analysis, (b) examine ADHD main effects on the battery with multivariate analysis of variance (MANOVA), (c) examine subtype and then symptom domain effects with multiple regression, and (d) determine whether effects could be explained by a range of potential confounds and covariates. For MANOVA, we report the effect size partial eta-squared (η^2), which indicates the percentage of variance accounted for by a factor in an analysis of variance type of model (Cohen et al., 2003). We preceded all of that by a check on outliers, missing data, and sample descriptive characteristics.

Results

Data Preparation and Variable Consolidation

Outliers and missing values. Extreme outliers ($z \geq 4.00$ and $SD > 0.50$ from next score) were truncated to within 0.50 standard deviations of the next nearest score to prevent undue influence of single scores on linear models and reduce Type I and Type II error (Wilcox et al., 1998). This resulted in the adjustment of one score for Trails B (from $z = 4.90$ to $z = 3.50$) and one score for SSRT (from $z = 5.80$ to $z = 3.60$). Data were missing for 1.7% of the cognitive variables as a result of computer malfunction or examiner error, which occurred early in the project when ADHD participants were primarily being seen. Within the ADHD group, amount of missing neuropsychological scores (Cohen & Cohen, 1983) was not associated with behavioral symptoms or disorders. We imputed the few missing data points and covaried "amount of missingness" from all analyses (as recommended by Cohen & Cohen, 1983) to control for effects of data imputation. That covariate was not significant in those models and never altered results, so it is ignored in the present results. We imputed missing data by using the expectation maximization algorithm, which is one form of maximum likelihood estimation. Maximum likelihood imputation methods are generally viewed as superior to older alternatives such as listwise deletion or regression estimation (Shaffer & Graham, 2002) because they preserve parameter estimates better than regression-based imputation (in our data, mean change in F with imputation was trivial at $F = 0.31$) and avoid the biases introduced by listwise deletion.

Consolidation of Neuropsychological and Behavioral Measures

We planned our analyses in the following two steps: (a) an initial MANOVA to describe group differences on the neuropsychological battery as a whole, and (b) regression analyses to assess subtype and domain-specificity effects that use a composite score to represent the executive functioning and output speed constructs.

With regard to behavioral symptom scores, for the regression models, composite ADHD-symptom domain scores were created by averaging K-SADS symptom totals across the three informants for each participant (informants and probands by definition had some agreement on symptoms, so reliability of these composites was acceptable (Inattention: $\alpha = .93$; Hyperactivity: $\alpha = .89$).

To set the stage for evaluation of the subtype and domain-specific effects, we evaluated the fit of our a priori two-factor model of neuropsychological functioning (executive functioning and response or output speed) by using a confirmatory factor analysis (with AMOS software). Initial model fitting revealed that Stroop interference (residual score) and go RT from the stop task had poor loadings on either factor and so they were eliminated from the model, resulting in a significant improvement in fit, $\Delta\chi^2(15, N = 195) = 173.61, p < .05$. Response variability, which we had initially assigned to the state-regulation factor, instead loaded on the EF factor. Forcing it to load on the speed factor resulted in a significant worsening of model fit and allowing it to load on both factors did not significantly improve fit. Dropping it entirely did not alter the two-factor solution. We therefore assigned it to the EF factor. This resulted in a model in which EF was indexed by Trails B-residual, stop RT, WCST (perseverative errors and categories composite), Tower total points, and response variability. The state regulation or Speed factor in turn was indexed by Trails A, Stroop word, and Stroop color naming. EF and Speed latent factors were permitted to correlate (as Figure 1 indicates, they correlated at .58). This model, which is shown in Figure 1, provided good overall fit, $\chi^2(19, N = 195) = 25.79, p > .05$, normed fit index (NFI) = .90, goodness-of-fit index (GFI) = .99, Comparative fit index (CFI) = .97, root mean square error of approximation (RMSEA) = 0.04. An alternate one-factor model yielded a significantly worse fit, $\Delta\chi^2(1, N = 195) = 23.26, p < .05$, than the two-factor solution shown in Figure 1, and unaccept-

able fit overall, $\chi^2(20, N = 195) = 49.05, p < .01$, NFI = .81, GFI = .94, CFI = .87, RMSEA = 0.09. Thus, the two-factor model was supported as the most reasonable summary of the neuropsychological measures for domain-specific analyses. On the basis of this finding, we created unit-weighted composite variables from standard (z) scores to represent EF and Speed for use in the regression analyses.

Preliminary Sample Description and Check on Covariates

Demographic and descriptive characteristics of the ADHD and control samples are presented in Table 1. Various rating-scale models of attention symptoms all showed marked elevations in our ADHD sample, indicating likely broad consensus on the validity of the ADHD assignments. Ethnic variation was closely similar to the surrounding community from which the sample was obtained. Parental household incomes were nearly identical in the two groups ($p > .80$), indicating they came from similar socioeconomic backgrounds. Despite this and consistent with reports that individuals with ADHD tend not to attend or complete college (Murphy & Barkley, 1996), ADHD individuals were more likely to be in the workforce or seeking work (67% vs. 56%) or attending community or technical college (16% vs. 7%), whereas controls were more likely to be attending university full time (32% vs. 16%, $p < .05$); controls thus had higher educational achievement overall. Covarying of this feature is debatable given that lower educational attainment is considered a hallmark consequence of ADHD (Barkley, 1998). However, we checked all results after covarying education and student status, with no appreciable change in results, as noted later. Also consistent with the literature, personal incomes tended to be lower for the ADHD than for control individuals (nonstudents, $M = \$21,300$ vs. $\$29,400, p < .01$). The group difference in FSIQ was not statistically significant, and covarying IQ may be inappropriate in ADHD studies because lower IQ in ADHD groups could be due to their EF weakness. Therefore, all of the results reported are without IQ covaried. However, to make sure that findings could not be explained by the association of IQ with EF, we also checked all results with IQ covaried as recommended by Murphy and Barkley (1996); any effects are noted in table footnotes later. The slight group differ-

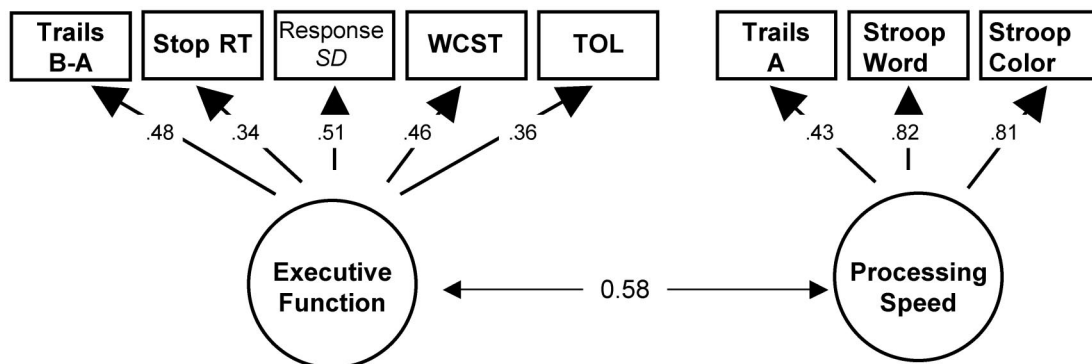


Figure 1. Best-fitting two-factor model for neuropsychological measures. Trails B-A = Trail Making B residual after regressing Trail Making A; Stop RT = Stop task reaction time; Response SD = within-subject variability in reaction time on go trials of the stop task; WCST=Wisconsin Card Sort Test categories and perseverative errors composite; TOL = Tower of London total points.

Table 1
Description of ADHD and Control Groups on Basic Cognitive and Demographic Variables and Key Comorbid and Lifetime Psychopathologies

Variable	ADHD		Control		<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
FSIQ	110.80	11.59	113.23	10.10	.12
WRAT Reading	101.00	11.44	105.10	9.05	.01
Number and percentage					
Reading disorder ^a	8	7.6%	3	3.3%	.20
Male	71	67.6%	32	35.6%	<.001
White	94	89.5%	75	78.9%	.21
Married	13	12.3%	13	14.4%	.53
Age in years	23.70	4.28	24.64	4.77	.15
Inattention–disorganization (per <i>DSM-IV</i>)	6.15	1.93	0.73	1.15	<.001
Hyperactivity–impulsivity (per <i>DSM-IV</i>)	4.75	2.40	0.86	1.10	<.001
Brown Attention Scale <i>T</i> score	74.24	12.09	54.45	8.98	<.001
Conners ADHD <i>T</i> score	69.79	12.40	43.83	10.89	<.001
Achenbach Attention Index <i>T</i> score	65.84	8.64	54.36	6.23	<.001
Number and percentage					
Lifetime alcohol dependence	16	15.2%	4	4.4%	.01
Lifetime drug dependence	11	10.5%	3	3.3%	.05
Lifetime substance dependence	24	22.9%	7	7.8%	.004
Major depressive disorder, past	32	30.5%	11	12.2%	.002
Any anxiety disorder, current ^b	19	18.1%	10	10.5%	.17
Antisocial personality disorder (APD)	5	5%	0		.06
APD symptoms		0.45		0.022	.003

Note. ADHD = attention-deficit/hyperactivity disorder; FSIQ = Full-scale IQ; WRAT = Wide Range Achievement Test.

^a Reading Disorder FSTQ ≤ 85 and at least 15 point difference between FSIQ and WRAT Reading.

^b Any Anxiety Disorder = generalized anxiety disorder, panic disorder, agoraphobia, phobias, posttraumatic stress disorder, obsessive–compulsive disorder, anxiety disorder.

ence in age was not significant. Covarying age also did not affect results; it is ignored in result reporting.

The ADHD group included multiple subtypes, as intended by our broad sampling strategy. They can be described as follows, along with the clinician agreements on the following descriptors: (a) Persistent Inattentive (met criteria for ADHD–I as children and adults; $n = 26$; $k = .84$); (b) Persistent Combined (met criteria for ADHD–C as children and adults; $n = 28$; $k = .83$); (c) Persistent Hyperactive (met criteria for ADHD–H as children and adults; $n = 5$; $k = .74$); (d) Inconsistent Subtype (met criteria for ADHD–H, ADHD–C, or ADHD–I as children but for a different subtype as an adult; $n = 21$; $k = .76$); (e) Residual ADHD (met criteria for ADHD–H, ADHD–C, or ADHD–I as children, as adults fell short of full criteria but continued to have symptoms with marked impairment; American Psychiatric Association, 1987; $n = 25$; $k = .89$). Effects of subtype were evaluated selectively in statistical models later.

Comorbid conditions in the ADHD and control group are presented in Table 1. As expected, the ADHD group had more substance abuse and mood disorder than did controls, but rates were still relatively modest, again consistent with an ADHD sample for whom problems were not able to be better explained by a co-occurring psychiatric condition, as required in the *DSM-IV* (American Psychiatric Association, 1994). Likewise, antisocial personality disorder was rare in this sample, but the ADHD group had significantly more antisocial symptoms and behaviors than did controls.

An initial check on gender effects revealed no Group (i.e., ADHD vs. control) \times Gender interaction in the omnibus

MANOVA on neuropsychological scores, $F = 1.11$, $p = .36$, but gender differences in task performance approached significance, $F = 1.81$, $p = .06$. We therefore retained gender as a covariate in all between-groups analyses, in view of the group differences in gender ratios. As a final preliminary overview of the data, we present in Table 2 the zero-order correlations between (a) the neuropsychological scores (note that Stroop variables have been reversed so that all neuropsychological scores have the same valence, with high scores indicating poor performance); (b) the composite EF and output speed scores; and (c) the composite ADHD-symptom domains (recall that these are composites of all reporters on the K–SADS). We noted that the correlation between the composite EF and Speed scores was $.34$ ($p < .01$). Table 2 indicates that the speed factor is correlated in opposite directions with inattention–disorganization ($r = .19$, $p < .05$) and hyperactivity–impulsivity ($r = -.05$, $p = ns$) although the latter was nonsignificant.

Is ADHD in Adulthood Associated with Overall Neuropsychological Deficit on Executive Functioning and Speed Measures?

A MANOVA was computed on the Neuropsychological Measures \times Diagnosis (ADHD vs. non-ADHD) with gender covaried. The omnibus effect was significant, $F(11, 171) = 2.35$, $p = .015$, $\eta^2 = .125$. Between-subjects effects for diagnosis, as presented in Table 3, showed significant group effects for Trails-residual, Stroop color, stop RT, go RT variability, and WCST categories. The ADHD effect remained significant after covarying FSIQ, $F =$

Table 2
Bivariate Zero-Order Correlations Among ADHD Symptom Domains, Neuropsychological Measures, and Composite Scores

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. ADHD	—																
2. Inattentive	.90	—															
3. Hyperactive	.76	.72	—														
4. Trails A	.01	.05	-.12	—													
5. Trails B residual	.21	.24	.17	.00	—												
6. Stroop word	.18	.20	-.03	.36	.23	—											
7. Stroop color	.18	.22	-.01	.35	.26	.67	—										
8. Stroop interference	.08	.08	.11	.10	.06	.00	.00	—									
9. Stop RT	.18	.23	.16	.17	.08	.23	.17	.26	—								
10. RT variability ^a	.19	.22	.13	.09	.27	.25	.29	.07	.22	—							
11. Go RT	.12	.14	.03	.01	.18	.09	.16	-.05	-.10	.70	—						
12. Perseverative errors	.06	.10	.05	.06	.26	.17	.13	.12	.13	.25	.07	—					
13. Categories ^b	.18	.18	.17	.02	.18	.16	.11	.12	.19	.13	.07	.60	—				
14. TOL moves	.01	.02	.02	.16	.18	.17	.18	.03	.05	.10	.03	.29	.22	—			
15. FSIQ	-.11	-.14	-.14	-.17	-.26	-.25	-.20	-.16	-.21	-.29	-.17	-.44	-.34	-.31	—		
16. EF composite	.23	.28	.19	.14	.56	.35	.33	.19	.48	.56	.23	.72	.66	.20	-.52	—	
17. Speed composite	.15	.19	-.05	.71	.21	.85	.84	.04	.24	.26	.11	.15	.11	.15	-.26	.34	—

Note. Test scores have been reverse coded for this table as needed, so that all neuropsychological variables will have the same directional valence (high score = poor performance) to ease readability, except that IQ was not reversed. Correlations greater than .13 are significant at the .05 level; correlations greater than .18 are significant at the .01 level. ADHD = attention-deficit/hyperactivity disorder; TOL = Tower of London; FSIQ = Full-scale IQ; EF = executive function; Stop RT = stop signal reaction time; Go RT = go reaction time on the stop task; Perseverative errors = Wisconsin Card Sorting Task (WCST) perseverative errors.

^a RT variability refers to reaction time variability on the go trials of the stop task.

^b “Categories” refers to WCST categories completed.

1.86, $p < .05$, $\eta^2 = .10$, and after covarying student–nonstudent status, $F = 2.08$, $p = .024$, $\eta^2 = .119$, with minor differences in results for individual variables as noted in the footnote to Table 3. Past major depressive disorder, current anxiety disorder, lifetime history of alcohol or substance dependence, and symptoms of antisocial personality disorder were covaried, with no change in results for the MANOVA omnibus test, $F = 1.85$, $p < .05$, $\eta^2 = .11$, or the individual tests.

To better isolate EFs, the MANOVA was repeated including only those neuropsychological tests from the EF composite score (Trails-residual, stop RT, response variability, WCST categories and perseverative errors, and TOL), as well as Stroop interference (residual), given its widespread use as an executive measure. The omnibus ADHD diagnosis effect was significant, $F = 3.53$, $p = .003$, $\eta^2 = .12$, with gender covaried (gender main effect was nonsignificant at $p = .38$ for this subset of variables). The same

Table 3
ADHD and Control Means, Standard Deviations, and Between-Group Effects for Individual Neuropsychological Measures from MANOVA (Gender Covaried)

Variable	ADHD		Control		$F(11, 182)$	p
	M	SD	M	SD		
Trails A	25.07	7.79	24.93	7.68	0.20	.66
Trails B (Residual)	2.67	14.91	-3.11	12.40	8.06	.005
Stroop word	98.35	15.55	104.03	15.79	6.16	.01 ^b
Stroop color	75.87	13.59	80.68	13.22	3.43	.07 ^a
Stroop interference	-0.60	9.22	0.70	7.92	0.62	.43
Stop RT	251.9	67.0	230.0	52.6	7.65	.006
RT variability	131.4	42.7	116.6	34.3	9.78	.002
Go RT	593.6	119.0	565.6	106.5	2.97	.09
WCST perseverative errors	6.77	3.33	6.35	3.45	0.89	.35
WCST categories	3.75	1.06	4.13	1.00	6.69	.01 ^{b,c}
TOL moves made	82.40	6.26	82.35	7.84	0.30	.59

Note. MANOVA = multivariate analysis of variance; ADHD = attention-deficit/hyperactivity disorder; RT = reaction time; WCST = Wisconsin Card Sorting Task; TOL = Tower of London; Full-scale IQ (FSIQ) estimated from five subtests of the Wechsler Adult Intelligence Scale—III.

^a $p < .05$ if gender is not covaried.

^b ns when FSIQ is covaried.

^c ns when education level is covaried.

^d ns when student status is covaried.

individual tests remained significant as in our initial MANOVA analysis as presented in Table 3. Covarying major depressive disorder, antisocial personality disorder symptoms, anxiety disorder, or lifetime substance dependence did not alter the effect of ADHD diagnosis, $F = 2.64, p < .05, \eta^2 = .10$; results again were essentially unchanged after covarying IQ, $F = 2.79, p < .01, \eta^2 = .10$, education level, or student status, $F = 3.04, p < .01, \eta^2 = .11$. Thus, these relatively robust ADHD effects were not explained by comorbid psychiatric disorders in the ADHD group or other covariates.

Are Neuropsychological Effects Carried by the ADHD Combined Type?

Given the broad definition of ADHD used in our ADHD sample, it was important to determine whether deficits in task performance could be attributed to the group as a whole or whether they were only accounted for by those participants with ADHD who met criteria for the most severe subtype. Neuropsychological theories of child ADHD suggest that executive type deficits are primarily expected in ADHD-C (Barkley, 1997). However, that has been difficult to demonstrate in children due to similar deficits in other subtypes (Hinshaw et al., 2002) and it is unclear theoretically whether one would expect this differentiation to emerge in adults in any case (Faraone et al., 2000; Murphy et al., 2001).

We tested this effect in regression analyses in which the criterion variable was the EF composite score by using orthogonal contrast codes to determine whether effects were carried by ADHD generally or by ADHD-C subtype. Participant gender was covaried in all models (Gender \times Contrast interactions were never significant and are omitted from further discussion). In the first model, Contrast 1 compared ADHD with controls, and Contrast 2 compared ADHD-C (in childhood, in adulthood, or both; $n = 55$) with the remaining ADHD participants ($n = 50$). The EF composite score was regressed onto gender, Contrast 1, and Contrast 2. The regression model was significant, $F = 4.67, p < .01, R^2 = 0.068$. However, only Contrast 1, comparing ADHD participants with controls, was significant ($\beta = -0.27, p < .001$). Contrast 2 had a trivial effect that was not statistically significant ($\beta = -0.004, p = .96$). This model was repeated with a revised Contrast 2, comparing only persistent ADHD-C (childhood and adulthood, $n = 28$) to the remaining ADHD sample. The overall regression model was significant, $F = 4.95, p < .01, R^2 = 0.072$. Again, Contrast 1 reached statistical significance ($\beta = -.30, p < .001$) and Contrast 2 did not ($\beta = .066, p = .38$). These results suggest that the EF weakness observed in the ADHD group was not carried solely by those with combined-type ADHD. Finally, we compared those who had partially improved (residual ADHD, plus those with ADHD-C in childhood but ADHD-I or ADHD-H in adulthood) versus the others. The ADHD main effect ($p < .01$) but not the subtype effect ($p = .26$) was again significant.

Are EF Weaknesses Specific to the Inattentive-Disorganized Symptom Cluster?

To address the question of whether there are differential relations of ADHD symptom domains to EF and Speed, regression analyses were conducted with the composite variables for these constructs as criterion variables, and inattentive-disorganized symptoms (I-D) and hyperactive-impulsive symptoms (H-I) as

predictor variables (recall that these were reliable composites of all reporters across adulthood and childhood). This approach makes symptoms a predictor instead of an outcome, as recommended by methodologists and followed by Nigg et al. (1998) so as to best evaluate specificity of psychopathological domains to particular correlates. These models used the entire sample of 195, although the same result held within the diagnosed ADHD group. As presented in Table 4, results indicated that only I-D was uniquely related to EF, $F = 8.03, p < .01, R^2 = .07$, whereas both I-D and H-I were related to Speed, $F = 12.27, p < .01, R^2 = .12$. However, H-I and I-D symptom domains were related to Speed in opposite directions: H-I was related to faster output speed (consistent with impulsive response), whereas I-D was related to slower performance (consistent with underaroused response).

Results were essentially unchanged when the model was changed to exclude response variability, as well as when it was altered to include major depressive disorder, any current anxiety disorder, symptoms of antisocial personality disorder, and lifetime history of substance dependence (see Table 4). When reading disorder (defined in Table 1 footnote) was covaried, these effects remained unchanged; reading disorder was related to processing speed ($\beta = 0.15, p < .05$), but not to EF ($\beta = 0.09, p = .22$). Finally, when ADHD symptom domains were separated according to developmental presentation (i.e., childhood ratings only or adult symptoms only entered into the model instead of the lifetime composite), the pattern of results remained unchanged.

Finally, to evaluate further the amount of variance in ADHD symptoms explained by the two composite neuropsychological scores as well as to evaluate whether they contributed additively to ADHD symptoms when they served as independent rather than dependent variables, we conducted further secondary checks. First, we analyzed regression models in which ADHD symptom domains (inattention, hyperactive-impulsive, and total) served as the outcome, and the EF and Speed composites were the predictors. These results revealed that EF carried the variance in these models ($\beta = .24, p < .01$, in both models), and that Speed failed to add value (*ns* in both models). The two variables together explained 9% of the variance in inattention and 5% of the variance in hyperactivity-impulsivity. That result confirmed the importance of EF in relation to ADHD symptoms but did not clarify the contribution of the two cognitive domains to different groups or types of ADHD. To evaluate this latter question, we revisited our subtype regression model, in which we divided the groups into ever ADHD-C ($n = 55$) and other ADHD ($n = 50$) along with controls ($n = 90$). We coded these groups into a 3-level categorical dependent variable and conducted a polytomous logistic regression analysis with EF and Speed composites as the predictors. This analysis compared each group with the control group (which served here as the reference group), and generated beta coefficients reflecting the degree to which being in the different ADHD groups was predicted by the EF and Speed composite variables. We interpreted statistically significant Wald statistics from this analysis as evidence for prediction of group membership by the cognitive variables (see P. Cohen et al., 2003, for a description of polytomous regression). This analysis revealed that membership in the ADHD-C type group was predicted by poor EF ($\beta = .87, SE = .33, p < .01$) but not by slow response speed ($\beta = -.01, SE = .24, p = ns$). In contrast, membership in the other ADHD group was predicted by slow response speed ($\beta = .50, SE = .25, p < .05$) but not by poor EF ($\beta = .65, SE = .34, p = .06$). Because

Table 4
Regression Models: Executive Function and Speed Composite Scores Regressed Upon Symptom Domains and Psychiatric Diagnoses

	R^2	B	$SE B$	β	partial r^2
Model 1: Executive functioning					
Model 1A: ADHD symptoms	0.077				
Inattention–Disorganization		0.03	0.01	0.29**	.040
Hyperactivity–Impulsivity		–0.002	0.01	–0.01	<.001
Model 1B: Comorbidity	0.123				
Inattention–Disorganization		0.03	0.01	0.27**	.035
Hyperactivity–Impulsivity		–0.01	0.01	–0.04	<.001
Past major depressive disorder		–0.07	0.11	–0.05	.002
Any current anxiety disorder		0.02	0.12	0.01	<.001
Symptoms of antisocial personal by disorder		0.04	0.05	0.06	.003
Substance dependence		0.30	0.12	0.18*	.028
Model 2: Speed					
Model 2A: ADHD Symptoms	0.113				
Inattention–Disorganization		0.07	0.01	0.48**	.111
Hyperactivity–Impulsivity		–0.07	0.02	–0.40**	.076
Model 2B: With Comorbidity	0.118				
Inattention–Disorganization		0.07	0.02	0.46**	.100
Hyperactivity–Impulsivity		–0.07	0.02	–0.40**	.073
Past major depressive disorder		–0.08	0.14	–0.04	.002
Any current anxiety disorder		–0.03	0.16	0.01	<.001
Symptoms of antisocial personal by disorder		0.02	0.06	0.02	<.001
Substance dependence		0.18	0.17	0.08	.005

* $p < .05$. ** $p < .01$.

the latter effect was marginal ($p = .06$), in a final check on the role of fast as opposed to slow responding, we noted that the 5 participants with ADHD who had persistent hyperactive type (see Method) were the only group to respond faster than the control group. When those 5 individuals were excluded, the prediction for the other ADHD group was even more clearly related to slow response speed ($\beta = .68$, $SE = .27$, $p < .05$) and not to EF ($\beta = .56$, $SE = .35$, ns) in the polytomous model logistic regression model.

Discussion

Despite the recent contributions to the field of over a dozen studies of neuropsychological functioning in adults with ADHD, consensus as to EF deficits in ADHD has been difficult to achieve. The present study, through the use of a larger sample than in most initial reports in this age range, identified clear deficits in executive functioning in the ADHD adult sample across several key measures and on an aggregate composite EF factor validated by confirmatory factor analysis. Furthermore, this EF deficit was largely related to symptoms of inattention–disorganization but not to hyperactivity–impulsivity in a test of recent dual-process models of ADHD (Sonuga-Barke, 2002). Finally, also partially consistent with such models (Berger & Posner, 2000), Speed emerged as a partially distinct neuropsychological factor that was related to both ADHD symptom domains, although in opposite directions. Results were independent of IQ, comorbid psychiatric disorders, gender, and ADHD subtype. We consider each of these points in turn.

First, we consider the basic finding of EF weaknesses in ADHD. Our findings confirm those of Lovejoy et al. (1999) and Murphy et

al. (2001) and, in some respects, those of Seidman et al. (1998), who concluded that adults with ADHD had important weaknesses in EF functioning. In the most comparable study to ours, Murphy et al. (2001) found a composite EF weakness in a sample of adults with no subtype effects observed. The confirmation of these results with the *DSM-IV* criteria, in a relatively large sample, and using a composite-factor approach lends credence to the theory that EF weakness is important in adult ADHD and that the adult syndrome may share important neurocognitive features with the syndrome in childhood, adding to its conceptual validity. It is important to note that we were careful both to define ADHD in such a way that symptoms could not be explained better by a comorbid condition and to covary coexisting psychiatric disorders when they were diagnosed along with primary ADHD. Our omnibus MANOVA effect of executive and neuropsychological weakness in the ADHD sample was not explained by comorbid antisocial symptoms (nor by antisocial personality disorder, which was rare in this sample), current anxiety disorders, lifetime mood disorder, history of alcohol or drug dependence, or reading disability. Thus, even though these other conditions may also somewhat impair executive functioning, the deficits observed here were also specific to ADHD with those other conditions controlled or excluded.

Second, the isolation of EF deficits to the behavioral domain of inattention–disorganization but not hyperactivity–impulsivity, provides needed validation data for recent two-factor theories of ADHD. For example, Sonuga-Barke (2002) has suggested that executive deficits contribute primarily to symptoms of inattention–disorganization and that problems in reward–response contribute primarily to symptoms of hyperactivity–impulsivity. The present study evaluated the element of those models that links executive

dysfunction with the inattention–disorganization domain. This prediction has been rarely evaluated empirically, and, to our knowledge, it has not been previously tested in adults, with most studies reporting on ADHD in aggregate. We found support for that prediction, supporting the dual-process model. This supposition also is consistent with findings that the Inattention domain, but not the Hyperactivity domain, is uniquely related to the personality domain of Conscientiousness–Constraint in adulthood (Nigg et al., 2002b), which is thought to be related to the same neural circuits involved in executive control (Nigg et al., 2001). Our findings also echo a recent finding in children (Chhabildas et al., 2001) regarding this two-process conception of symptom domains and cognitive functions.

Such findings should give added impetus to such two-factor theories and suggest that they may be able to be extended into adulthood. Indeed, such conceptions may be especially important in the early adult developmental period; when neural maturation is completed, hyperactive–impulsive behaviors tend to become less prominent, and impairment may be relatively more dependent on persisting problems with inattention and disorganization (Barkley, 1998). However, it should be noted that the effect did not hold when the model was reversed (only EF predicted inattention and hyperactivity). Even so, EF and Speed were differentially related to ADHD subgroups in the logistic regression analysis, with the combined type characterized by poor EF and the other group by slow response speed. Thus, although EF and Speed may contribute to different groups of individuals with ADHD, the theory may need to be modified to indicate that EF contributes primarily to the combined subtype, and slow speed, perhaps related to sluggish cognitive tempo, may contribute to other types. Finally, we noted a negative relation of speed to hyperactivity when inattention was partialled. That effect may reflect a small subgroup that may have excess response speed; that group may be a hyper- as opposed to hypoaroused group (see Clark, Barry, McCarthy, & Selikowitz, 2001 for related conclusions).

Third, the conception that at least two neurocognitive mechanisms are involved in ADHD (Berger & Posner, 2000), one in the more pure executive domain and one in a speed domain related to alertness or activation, gained support in our data. The two factor organization of the neuropsychological scores fit the data very well, and these two factors showed differential relations to the ADHD symptom domains. However, several caveats were noted for the Speed factor. Inattention was related to slower response, consistent with an underarousal effect. Hyperactivity–impulsivity was related to faster response speed but only when inattention was partialled, consistent with impulsive response and also with an overarousal response in a subgroup (Clarke et al., 2001).

Another caveat was that response variability did not load on the Speed factor as expected, but instead loaded with EF. This rendered our EF factor slightly different than the intended conceptual model. Indeed, the conceptual structure of the EF domain has received extensive discussion and warrants comment here as well. Whereas EF is usually viewed as a multicomponent domain, a position we view as reasonable, the possibility that a general factor may account for a significant portion of the ability shared across many executive tasks has been widely discussed. We found some support for that possibility and used that result to create a composite variable with which to simplify our regression models testing symptom-domain effects. However, the inclusion of variability in the model, although empirically sound, was not consis-

tent with the conceptual EF model. We retained the empirical model to maximize the power of our analyses. It is important to note that results were unchanged if we excluded variability from this factor (the two-factor solution still fit the data well, and the primary regression findings showing that EF was specific to inattentive symptoms still held). We also note that others have used batteries with more working memory representation (e.g., Murphy et al., 2001). We had limited coverage of the working memory domain in our battery, and may have found two EF factors if we had more working memory measures. A full examination of the structure of EF measures obviously would benefit from even larger samples and more measures of EF. The main point for our study was that when a single latent factor was pulled from the EF domain, it was associated uniquely with inattention–disorganization but not hyperactivity–impulsivity when both were entered as predictors.

Several other limitations should be recognized. Although the sample size for two-group comparisons made this one of the largest studies of adult *DSM-IV* ADHD disorder to be yet reported, our ADHD sample was diverse. This limited our ability to powerfully test subtype differences. However, those effects when tested were small and not close to being significant and help to support the broader clinical generalizability of these results to adults with ADHD disorder in general. This is important because validity and definition of subtypes in adults is still not established. Further work is needed to evaluate subtypes, but the findings here establish EF as a correlate of ADHD in adults. The ADHD group was relatively well functioning, with 16% in college, an average to slightly above average group IQ, and low rates of antisocial personality disorder, yet still showed executive deficits relative to controls; these were not accounted for by IQ or by better educational attainment in the control group. Our community-based sampling was done in a region (central Michigan) in which 38.7% of adults between the ages of 18 and 40 are in college or graduate school, so the fact that we had substantial representation of college students was not surprising and indicated we adequately sampled the local population. Nonetheless, the proportion of students in the samples is higher than in the general national population, and this may limit generalizability. Also, our sample may have been higher functioning than some prospective samples, in that about 35% reported not having been diagnosed before (although all reported impairing symptoms and full criteria met from childhood, confirmed by informant report). Groups differed in gender ratios; however, gender was covaried in all analyses so this gender difference did not explain any results reported. Finally, we did not include a psychiatric comparison group by which to formally test specificity of effects to ADHD. Indeed, EF should be affected secondarily by other conditions, including active mood disorders (Gallagher & Blader, 2001). However, we were able to show that the ADHD executive deficit observed here was not explained by major comorbid conditions.

In conclusion, these data indicate that executive functioning is weakened in ADHD in young adulthood, similar to what is observed in children with ADHD. These findings support the validity of the syndrome of ADHD in adults and support consideration of executive functioning in theories of the syndrome's persistence and expression in adulthood. The data further suggest that the contribution of executive functioning to ADHD in adults is particularly important in the symptom domain of inattention–disorganization. This finding may be consistent with dual-process

models of ADHD, which attempt to account for etiology via multiple pathways of neuropsychological dysfunction.

References

- American Psychiatric Association. (1987). *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.). Washington, DC: Author.
- American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: Author.
- Achenbach, T. (1991). *Manual for the Young Adult Self Report and Young Adult Behavior Checklist*. Burlington, VT: University of Vermont Department of Psychiatry.
- Arbuthnott, K., & Frank, J. (2000). Trail Making Test, Part B as a measure of executive control: Validation using a set-switching paradigm. *Journal of Clinical and Experimental Neuropsychology*, *22*, 518–528.
- Aron, A. R., Fletcher, P. C., Bullmore, E. T., Sahakian, B. J., & Robbins, T. W. (2003). Stop signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nature Neuroscience*, *6*, 115–116.
- Band, G. P. H., Van Der Molen, M. W., & Logan, G. D. (2003). Horse-race model simulations of the stop-signal procedure. *Acta Psychologica*, *112*, 105–142.
- Banich, M. T., Milham, M. P., Atchley, R., Cohen, N. J., Webb, A., Wszalek, T., et al. (2000). fMRI studies of Stroop tasks reveal unique roles of anterior and posterior brain systems in attentional selection. *Journal of Cognitive Neuroscience*, *12*, 988–1000.
- Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive function: Constructing a unified theory of ADHD. *Psychological Bulletin*, *121*, 65–94.
- Barkley, R. A. (1998). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment* (2nd ed.). New York: Guilford Press.
- Barkley, R. A. (2002). Major life activity and health outcomes associated with attention-deficit/hyperactivity disorder. *Journal of Clinical Psychiatry*, *63*(Suppl 12), 10–15.
- Barkley, R. A., & Biederman, J. (1997). Towards a broader definition of the age of onset criterion for attention deficit hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*, 1204–1210.
- Barkley, R. A., & Murphy, K. R. (1998). *Attention-Deficit Hyperactivity Disorder: A Clinical Workbook* (2nd ed.). The Guilford Press.
- Benes, F. M. (2001). The development of prefrontal cortex: The maturation of neurotransmitter systems and their interactions. In C. A. Nelson & M. Luciana (Eds.), *Handbook of developmental cognitive neuroscience* (pp. 79–92). Cambridge, MA: MIT Press.
- Berger, A., Posner, M. I. (2000). Pathologies of brain attentional networks. *Neuroscience and Biobehavioral Reviews*, *24*, 3–5.
- Biederman, J., Faraone, S. V., Keenan, K., Benjamin, J., Krifcher, B., Moore, C., Sprich-Buckminster, S., Ulaglia, K., Jellinek, M. S., Steingard, R., Spencer, T., Norman, D., Kolodny, R., Kraus, I., Perrin, J., Keller, M. B., & Tsuang, M. T. (1992). Further evidence for family-genetic risk factors in attention deficit hyperactivity disorder: Patterns of comorbidity in probands and relatives in psychiatrically and pediatrically referred samples. *Archives of General Psychiatry*, *49*, 728–738.
- Biederman, J., Faraone, S. V., Keenan, K., Knee, D., & Tsuang, M. T. (1990). Family-genetic and psychosocial risk factors in DSM-III attention deficit disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *29*, 526–533.
- Brown, T. E. (1996). *Brown Attention-Deficit Disorder Scales*. San Antonio, TX: The Psychological Corporation.
- Chhabildas, N., Pennington, B. F., & Willcutt, E. G. (2001). A comparison of the neuropsychological profiles of the DSM-IV subtypes of ADHD. *Journal of Abnormal Child Psychology*, *29*, 529–540.
- Clarke, A. Barry, R., McCarthy, R., & Selikowitz, M. (2001). EEG-defined subtypes of children with attention-deficit/hyperactivity disorder. *Clinical Neurophysiology*, *112*, 2098–2105.
- Cohen, P., & Cohen, J. (1983). *Applied multiple regression/correlation analysis for the behavioral sciences*. Hillsdale, NJ: Erlbaum.
- Cohen, P., Cohen, J., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences* (3rd ed.). Hillsdale, NJ: Erlbaum.
- Conners, C. K., Erhardt, D., & Sparrow, E. (1999). *Adult ADHD Rating Scales: Technical manual*. Toronto: Multi-Health Systems, Inc.
- Corbett, B., & Stanczak, D. E. (1999). Neuropsychological performance of adults evidencing attention-deficit/hyperactivity disorder. *Archives of Clinical Neuropsychology*, *14*, 373–387.
- Davis, H. P., Keller, F. (2002). *Colorado Assessment Tests (CATs) Version 1.2*. Colorado Springs, CO: Author.
- Delis, D. C., Kaplan, E., & Kramer, J. H. (2001). *Delis-Kaplan Executive Function System*. New York: Psychological Corporation.
- Dinn, W. M., Robbins, N. C., Harris, C. L. (2001). Adult attention-deficit/hyperactivity disorder: Neuropsychological correlates and clinical presentation. *Brain and Cognition*, *46*, 114–121.
- Duncan, J., Johnson, R., Swales, M., & Freer, C. (1997). Frontal lobes after head injury: Unity and diversity of function. *Cognitive Neuropsychology*, *14*, 713–741.
- Epstein, J. N., Johnson, D. E., Varia, I. M., & Conners, C. K. (2001). Neuropsychological assessment of response inhibition in adults with ADHD. *Journal of Clinical and Experimental Neuropsychology*, *23*, 362–371.
- Faraone, S. V., Biederman, J., Spencer, T., Wilens, T., Seidman, L. J., Mick, E., & Doyle, A. E. (2000). Attention-deficit/hyperactivity disorder in adults: An overview. *Biological Psychiatry*, *48*, 9–20.
- Faraone, S. V., Biederman, J., Weber, W., & Russell, R. L. (1998). Psychiatric, neuropsychological and psychosocial features of DSM-IV subtypes of attention-deficit/hyperactivity disorder: Results from a clinically referred sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, *37*, 185–193.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1995). The Structured Clinical Interview for DSM-III Personality Disorders (SCID-I). *Journal of Personality Disorders*, *9*, 83–91.
- Gallagher, R., & Blader, J. (2001). The diagnosis and neuropsychological assessment of adult attention-deficit/hyperactivity disorder: Scientific study and practical guidelines. *Annals of the New York Academy of Sciences*, *931*, 148–171.
- Gansler, D. A., Fucetola, R., Kregel, M., Stetson, S., Zimering, R., & Makary, C. (1998). Are there cognitive subtypes in adult attention deficit/hyperactivity disorder? *Journal of Nervous and Mental Disease*, *186*, 776–781.
- Hanes, K. R., Andrews, D. G., Smith, D. J., & Pantelis, C. (1996). A brief assessment of executive control dysfunction: Discriminant validity and homogeneity of planning, set shift, and fluency measures. *Archives of Clinical Neuropsychology*, *11*, 185–191.
- Heaton, R. K., Chelune, G. J., Talley, J. L., Kay, G. G., & Curtiss, G. (1993). *Wisconsin Card Sorting Test Manual*. USA: Publishing Assessment Resources.
- 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.
- Johnson, D. E., Epstein, J. N., Waid, L. R., Latham, P. K., Voronin, K. E., & Anton, R. F. (2001). Neuropsychological performance deficits in adults with attention-deficit/hyperactivity disorder. *Archives of Clinical Neuropsychology*, *16*, 587–604.
- Katz, L. J., Goldstein, G., & Geckle, M. (1998). Neuropsychological and personality differences between men and women with ADHD. *Journal of Attention Disorders*, *2*, 239–247.
- Klorman, R., Hazel-Fernandez, L. A., Shaywitz, S. E., Fletcher, J. M., Marchione, K. E., Holahan, J. M., Stuebing, K. K., & Shaywitz, B. A. (1999). Executive functioning deficits in attention-deficit hyperactivity disorder are independent of oppositional defiant disorder or reading

- disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38, 1148–1155.
- Kuntsi, J., Stevenson, J., Oosterlaan, J., & Sonuga-Barke, E. J. S. (2001). Test-retest reliability of a new delay aversion task and executive function measures. *British Journal of Developmental Psychology*, 19, 339–348.
- Logan, G. D. (1994). A user's guide to the stop signal paradigm. In D. Dagenbach & T. Carr (Eds.), *Inhibition in language, memory, and attention* (pp. 189–239). San Diego: Academic Press.
- Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychological Review*, 91, 295–327.
- Logan, G. D., Schachar, R. J., Tannock, R. (1997). Impulsivity and inhibitory control. *Psychological Science*, 8, 60–64.
- Lovejoy, D. W., Ball, J. D., Keats, M., Stutts, M. L., Spain, E. H., Janda, L., et al. (1999). Neuropsychological performance of adults with attention-deficit/hyperactivity disorder (ADHD): Diagnostic classification estimates for measures of frontal lobe/executive functioning. *J Int Neuropsychol Soc*, 5, 222–233.
- Lowe, C., & Rabbitt, P. (1998). Test/re-test reliability of the CANTAB and ISPCD neuropsychological batteries: Theoretical and practical issues. *Neuropsychologica*, 36, 915–923.
- MacLeod, C. M. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin*, 109, 163–203.
- Milich, R., Balentine, A., & Lynam, D. (2001). ADHD combined type and ADHD predominantly inattentive type are distinct and unrelated disorders. *Clinical Psychology Science and Practice*, 8, 463–488.
- Miller, G. M., & Chapman, J. P. (2001). Misunderstanding analysis of covariance. *Journal of Abnormal Psychology*, 110, 40–48.
- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., & Howerter, A. (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.
- Murphy, K., & Barkley, R. A. (1996). Attention deficit hyperactivity disorder adults: Comorbidities and adaptive impairments. *Comprehensive Psychiatry*, 37(6), 393–401.
- Murphy, K., Barkley, R. A., & Bush, T. (2001). Executive functions in young adults with attention deficit hyperactivity disorder. *Neuropsychology*, 15, 211–220.
- Newman, S. D., Carpenter, P. A., Varma, S., & Just, M. A. (2003). Frontal and parietal participation in problem solving in the Tower of London: fMRI and computational modeling of planning and high-level perception. *Neuropsychologia*, 41, 1668–1682.
- Nigg, J. T. (1999). The ADHD response-inhibition deficit as measured by the stop task: Replication with DSM-IV combined type, extension, and qualification. *Journal of Abnormal Child Psychology*, 27, 393–402.
- Nigg, J. T. (2001). Is ADHD an inhibitory disorder? *Psychological Bulletin*, 127, 571–598.
- Nigg, J. T., Blaskey, L., Huang-Pollock, C., & Rappley, M. D. (2002a). Neuropsychological executive functions and DSM-IV ADHD subtypes. *Journal of the American Academy of Child and Adolescent Psychiatry*, 41, 59–66.
- Nigg, J. T., Goldsmith, H. H., & Sachek, J. (2004). Temperament and attention deficit/hyperactivity disorder: The development of a multiple pathway model. *Journal of Clinical Child and Adolescent Psychology*, 33, 42–53.
- Nigg, J. T., Hinshaw, S. P., Carte, E. T., & Treuting, J. J. (1998). Neuropsychological correlates of antisocial behavior and comorbid disruptive behavior disorders in children with ADHD. *Journal of Abnormal Psychology*, 107, 468–480.
- Nigg, J. T., John, O. P., Blaskey, L. G., Huang-Pollock, C. L., Willcutt, E. G., Hinshaw, S. P., & Pennington, B. (2002b). Big five dimensions and ADHD symptoms: Links between personality traits and clinical symptoms. *Journal of Personality and Social Psychology*, 83, 451–469.
- Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 37, 51–87.
- Posner, M., & Petersen, S. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, 13, 25–42.
- Puig-Antich, J., & Ryan, N. (1986). *Kiddie Schedule for Affective Disorders and Schizophrenia*. Pittsburgh, PA: Western Psychiatric Institute.
- Reitan, R. M. (1958). Validity of the Trail Making Test as an indicator of organic brain damage. *Perceptual and Motor Skills*, 8, 271–276.
- Sachdev, P. (1999). Attention deficit hyperactivity disorder in adults. *Psychological Medicine*, 29, 507–514.
- Sandson, T. A., Bachna, K. J., & Morin, M. D. (2000). Right hemisphere dysfunction in ADHD: Visual hemispatial inattention and clinical subtype. *Journal of Learning Disabilities*, 33, 83–90.
- Seidman, L. J., Biederman, J., Weber, W., Hatch, M., & Faraone, S. V. (1998). Neuropsychological function in adults with attention-deficit hyperactivity disorder. *Biological Psychiatry*, 44, 260–268.
- Sergeant, J. A., Oosterlaan, J., & van der Meere, J. (1999). Information processing and energetic factors in attention deficit/hyperactivity disorder. In: *Handbook of disruptive behavior disorders* (pp. 75–104). Quay HC, Hogan A, (Eds.). New York: Plenum Press.
- Shafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods*, 7, 147–177.
- Sonuga-Barke, E. J. S. (2002). Psychological heterogeneity in AD/HD—A dual pathway model of behaviour and cognition. *Behavioural Brain Research*, 130, 29–36.
- Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, 18, 643–662.
- Stuss, D. T., Bisschop, S. M., Alexander, M. P., Levine, B., Katz, D., & Izukawa, D. (2001). The trail making test: A study in focal lesion patients. *Psychological Assessment*, 13, 230–239.
- Wang, L., Kakigi, R., & Hoshiyama, M. (2001). Neural activities during Wisconsin Card Sorting Test – MEG observation. *Cognitive Brain Research*, 12, 19–31.
- Wechsler, D. (1997). *Wechsler Adult Intelligence Scale – III. Administration and scoring manual*. San Antonio, TX: The Psychological Corporation.
- Wender, P. H., Wolf, L. E., & Wasserstein, J. (2001). Adults with ADHD: An overview. In J. Wasserstein, L. E. Wolf, & F. F. LeFever, (Eds.), *Adult Attention Deficit Disorder: Brain Mechanisms and Life Outcomes*, *Annals of the New York Academy of Sciences*, 931, 1–16.
- Weyandt, L. L., Linterman, I., & Rice, J. A. (1995). Reported prevalence of attentional difficulties in a general sample of college students. *Journal of Psychopathology and Behavioral Assessment*, 17, 293–304.
- Wilkinson, G. S. (1993). *The Wide Range Achievement Test administration manual*. Wilmington, DE: Wide Range.
- Wilcox, R. R., Keselman, H. J., & Kowalchuk, R. K. (1998). Can tests for treatment group equality be improved? The bootstrap and trimmed means conjecture. *British Journal of Mathematical and Statistical Psychology*, 51, 123–134.

Received July 22, 2004

Revision received November 15, 2004

Accepted December 2, 2004 ■