Impact of Executive Function Deficits and Attention-Deficit/Hyperactivity Disorder (ADHD) on Academic Outcomes in Children

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The association between executive function deficits (EFDs) and functional outcomes were examined among children and adolescents with attention-deficit/hyperactivity disorder (ADHD). Participants were children and adolescents with ($n = 259$) and without ($n = 222$) ADHD, as ascertained from pediatric and psychiatric clinics. The authors defined EFD as at least 2 executive function measures impaired. Significantly more children and adolescents with ADHD had EFDs than did control participants. ADHD with EFDs was associated with an increased risk for grade retention and a decrease in academic achievement relative to (a) ADHD alone, (b) controlled socioeconomic status, (c) learning disabilities, and (d) IQ. No differences were noted in social functioning or psychiatric comorbidity. Children and adolescents with ADHD and EFDs were found to be at high risk for significant impairments in academic functioning. These results support screening children with ADHD for EFDs to prevent academic failure.

Among the family of mental processes that comprise neuropsychological functioning is the set of higher cortical abilities referred to as executive functions (EFs). This construct has been defined as "the ability to maintain an appropriate problem set for attainment of future goals" (Welsh & Pennington, 1989, p. 201) and includes such abilities as components of attention, reasoning, planning, inhibition, set-shifting, interference control, and working memory (Pennington & Ozonoff, 1996). EFs are considered to be critically important for complex human behavior, and their breakdown is thought to commonly result in behavioral or psychiatric impairment (Goldberg & Seidman, 1991). Studies of Alzheimer's disease (Chen, Sultzer, Hinkin, Mahler, & Cummings, 1998) and schizophrenia (Green, 1996), as well as studies of patients undergoing physical rehabilitation (Cahn-Weiner, Malloy, Boyle, Marran, & Salloway, 2000; Hanks, Rapport, Millis, & Deshpande, 1999), have clearly demonstrated significant impairments in functional outcomes associated with EF deficits (EFDs), supporting the critical role of EFs for sophisticated human behavior.

An emerging literature has repeatedly documented that children with attention-deficit/hyperactivity disorder (ADHD) exhibit EFDs. For example, a recent literature review of 18 studies by Pennington and Ozonoff (1996) concluded that children with ADHD consistently exhibit worse performance on certain cognitive and EF measures. Likewise, using a focal neuropsychological

battery aimed at assessing EFDs in children and adolescents with ADHD, we have shown that as a group, boys with ADHD show significantly poorer executive functioning relative to control participants in referred (Seidman, Biederman, Faraone, Weber, & Ouellette, 1997) and nonreferred (Seidman, Biederman, Monuteaux, Weber, & Faraone, 2000) samples. Other studies have reached similar conclusions (Barkley, 1997; Douglas, 1972). There is less research investigating EFDs in girls with ADHD. However, a growing literature that includes research by our group suggests that EFDs are also found in girls with ADHD (unpublished data).

Despite these consistent data implicating EFDs in ADHD, very little is known about the clinical implications of EFDs in children and adolescents with ADHD. Although impairments on such tests are assumed to relate to real-world functions, the ecological validity of impairment on such tests and in ADHD has yet to be determined. Given the critical importance of EFs for adequate functioning and considering the poor long-term psychiatric, social, and academic outcome associated with ADHD (Barkley, Fischer, Edelbrock, & Smallish, 1990; Biederman et al., 1996; Cantwell, 1985; Edelbrock, Costello, & Kessler, 1984; Faraone et al., 1993; Greene, Biederman, Faraone, Sienna, & Garcia Jetton, 1997; Hart, Lahey, Loeber, Applegate, & Frick, 1995), it is important to assess whether the functional impairment related to ADHD is associated with ADHD itself, independently of EFDs. One approach to address this question is to compare the functional outcomes of ADHD samples with and without EFDs. If children who have ADHD with EFDs perform worse compared with children with ADHD without EFDs, there would be evidence that at least part of the impairment observed in children who have ADHD is associated with EFDs.

Like other neuropsychological functions, executive functioning is usually viewed as a continuously varying trait. Yet, there are several reasons why a categorical definition of EFDs may be useful. Such a classification would (a) allow for comparisons of the prevalence of clinically significant EFD across populations, (b) encourage the standardization of neuropsychological assessment in

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research, (c) aid in the validation of psychiatric diagnoses, and (d) provide a useful diagnostic tool for clinicians. Specifically for ADHD, an EFD classification scheme would provide a useful tool for assessing the association between EFDs and ADHD.

Using the sample with which we demonstrated group neuropsychological deficits in referred (Seidman et al., 1997) and nonreferred (Seidman et al., 2000) children with ADHD, we aimed to test the association between EFD and academic and psychosocial impairments among children with ADHD and control participants at the individual level. On the basis of the literature, we hypothesized that EFDs would be more prevalent in children with ADHD relative to control participants and would be associated with impairments in multiple domains of functioning.

Method

Participants

In this analysis, the data from two identically designed case-control family studies of ADHD were combined. These studies ascertained families on the basis of male (Biederman et al., 1992) and female (Biederman et al., 1999) participants with ($n = 140$ boys; $n = 140$ girls) and without ($n = 120$ boys; $n = 122$ girls) *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; *DSM–III–R*; American Psychiatric Association, 1987) ADHD, as ascertained from pediatric and psychiatric sources. Participants were 6–17 years of age at the time of ascertainment. Male participants were brought in again for a 4-year follow-up assessment (Biederman et al., 1996) in which 128 of the proband participants with ADHD (91%) and 109 of the control proband participants (91%) participated. There were no significant differences between those participants successfully reassessed and those lost to follow-up on psychiatric, cognitive, or functional outcomes (Biederman et al., 1996). Potential participants were excluded if they had been adopted, their nuclear family was not available, they had major sensorimotor handicaps (e.g., paralysis, deafness, blindness, psychosis, autism, inadequate command of the English language), or they had a full scale IQ (Wechsler, 1974) that was less than 80. After a complete description of the study, parents provided informed consent for their children, and children and adolescents provided written assent, and the IRB granted approval for this study. For the present study, we used all proband participants with available neuropsychological data, which included 121 male proband participants with ADHD (95%), 103 male control participants (94%), 138 female proband participants with ADHD (99%), and 122 female control participants (100%). The few participants not assessed were due to time constraints, scheduling problems, or unwillingness on the part of the participants.

A three-stage ascertainment procedure was used to select the proband participants for both studies. For participants with ADHD, the first stage was the patient's referral. The second stage confirmed the diagnosis of ADHD by using a telephone questionnaire administered to the mother. The questionnaire asked about the 14 *DSM–III–R* symptoms of ADHD and questions regarding study-exclusion criteria. The third stage confirmed the diagnosis with face-to-face structured interviews with the mother. Only patients who received a positive diagnosis at all three stages were included. For control proband participants, we ascertained participants from referrals to medical clinics for routine physical examinations. In the second stage, the control mothers responded to the telephone questionnaire. Eligible control participants meeting study-entry criteria were recruited for the study and received the third-stage assessment (structured interview). Only participants classified as not having ADHD at all three stages were included in the control group.

Psychiatric Assessments

All diagnostic assessments used structured interviews based upon the criteria of the *DSM–III–R*. Psychiatric assessments of proband participants

relied on the epidemiologic version of the Schedule for Affective Disorder and Schizophrenia for Children (Orvaschel, 1985). Diagnoses were based on independent interviews with the mothers and direct interviews of proband participants, except for children younger than 12 years of age, who were not directly interviewed. Maternal reports and self-reports were combined by considering a diagnosis positive if it was endorsed by either interview. The structured interviews assessed lifetime history of psychopathology. ADHD symptoms, based on *DSM–III–R* criteria, were those measured at Year 4 for boys and baseline for girls.

The interviewers–psychometricians had undergraduate degrees in psychology; they were trained to high levels of interrater reliability for the assessment of psychiatric diagnosis by Joseph Biederman. We computed kappa coefficients of agreement by having experienced, board-certified child and adult psychiatrists diagnose participants from audiotaped interviews made by the assessment staff. On the basis of 173 interviews from a mixed pediatric and adult data set, the median kappa for all diagnoses was .86, and the kappa for ADHD was .98. In addition, the assessment personnel were blind to proband diagnosis (ADHD or control) and ascertainment site (psychiatric or pediatric). All follow-up assessments were made blindly to prior assessments of the same participants and their family members. Thus, all neuropsychological function assessments were administered and scored by examiners who were unaware of all other data on the participants.

A committee of board-certified child and adult psychiatrists resolved all diagnostic uncertainties. The committee members were blind to the participants' ascertainment group, ascertainment site, all data collected from other family members, and all nondiagnostic data (e.g., neuropsychological tests). Diagnoses were considered positive if, on the basis of the interview results, *DSM–III–R* criteria were unequivocally met to a clinically meaningful degree. We created categories of disorders for this analysis as follows: (a) mood disorder included major depression with severe impairment or bipolar disorder; (b) multiple anxiety was defined as two or more anxiety disorders; (c) speech–language was defined as language disorder or stuttering; (d) disruptive disorder included conduct disorder, oppositional defiant disorder, or antisocial personality disorder; and (e) psychoactive substance use disorder included drug or alcohol abuse or dependence. Rates of disorders reported here are lifetime prevalence.

Psychosocial Assessments

Social functioning was assessed with the Social Adjustment Inventory for Children and Adolescents (SAICA; Orvaschel & Walsh, 1984), a semistructured interview schedule administered to the mother that assesses adaptive functioning. The SAICA consists of 76 items that assess social difficulties at school and in interactions with peers, siblings, and parents. There is evidence supporting the validity (Biederman, Faraone, & Chen, 1993; Greene et al., 1996; John, Gammon, Prusoff, & Warner, 1987), interrater reliability (Greene et al., 1997), and internal consistency (Greene et al., 1997) of the SAICA. As a measure of overall functioning, we used the *DSM–III–R* Global Assessment of Functioning (Orvaschel & Puig-Antich, 1987), a summary score of each participant's overall functioning assigned by the interviewers on the basis of information gathered in the diagnostic structured interview. Socioeconomic status (SES) was assessed with the Hollingshead Scale (Hollingshead, 1975).

Cognitive Assessments

Using the methods of Sattler (1988), we estimated full scale IQ from the vocabulary and block design subtests of Wechsler Intelligence Scales for Children—Revised (WISC–R; Wechsler, 1974) for participants younger than 17 years of age and the Wechsler Adult Intelligence Scales—Revised (Wechsler, 1981) for participants older than 17 years of age. Our interviewers assessed academic achievement with the Arithmetic subtest of the Wide Range Achievement Test—Revised (WRAT–R; Jastak & Jastak, 1985). Participants from the study of boys with ADHD were administered

the Gilmore Oral Reading Test (Gilmore & Gilmore, 1968) at the baseline assessment and the Reading subtest of the WRAT–R (Jastak & Jastak, 1985) at follow-up. Participants from the study of girls with ADHD were administered the Reading subtest of the WRAT–R (Jastak & Jastak, 1985). The definition of learning disabilities under Public Law 94–142 requires a significant discrepancy between a child's potential and achievement (Federal Register, 1977). Recommended by Reynolds (1984), we used a statistically corrected discrepancy between IQ and achievement to define learning disability.

Neuropsychological Assessments

The central theoretical construct guiding our choice of many of the tests in the battery is that key neuropsychological deficits in ADHD are associated with frontal regions or frontal networks, indicating impairment in a widespread cerebral network underlying attention and EFs. The hypothesis that the neuropsychological underpinnings of ADHD are characterized by executive dysfunction was proposed by investigators who recognized similarities in clinical presentation between persons with hyperactivity and adult patients with frontal lobe damage (Mattes, 1980; Shue & Douglas, 1992). EFs are distinct from other mental functions such as sensation, perception, or memory per se. There is, however, considerable overlap with domains such as attention, reasoning, and problem solving and with certain components of learning and memory (Pennington & Ozonoff, 1996). Thus, we chose commonly used clinical neuropsychological tests that assess components of EFs that are thought to be indirect indices of fronto-striatal systems and that have been used in the research literature on ADHD. These components of EFs include (a) vigilance and distractibility, (b) planning and organization, (c) response inhibition, (d) set shifting and categorization, (e) selective attention, (f) visual scanning, and (g) verbal learning. Thus, the neuropsychological battery we developed was based on the empirical and clinical literatures on attention, ADHD, and EFs. Although there is no standard battery of EF measures in the field, we specifically selected tests that have a long track record of use in both clinical settings and the research literature and that are consistent with our theoretical perspective. The tests and variables used are as follows:

1. The copy organization and delay organization of the Rey–Osterrieth Complex Figure (Osterrieth, 1944; Rey, 1941; scored by the Waber–Holmes method), which are meant to test planning and organization.

2. The total errors score (sum of omission, commission, and late errors) of the Auditory Continuous Performance Test (Weintraub & Mesulam, 1985), which is intended to measure auditory sustained attention, vigilance, and impulsivity.

3. Perseverative errors and loss of set of the computerized Wisconsin Card Sorting Test (WCST; Heaton, Chelune, Talley, Kay, & Curtiss, 1993), which measures reasoning ability, concept formation, and cognitive flexibility.

4. The percentage of words learned (number of words recalled across all trials divided by total number of words) of the Wide Range Achievement of Memory and Learning test for children less than 17 years of age (Adams & Sheslow, 1990) or the California Verbal Learning Test in children greater than or equal to 17 years of age (Delis, Kramer, Kaplan, & Ober, 1987), which is intended to be an index of left prefrontal systems and a measure of verbal learning and working memory.

5. The color–word raw score of the Stroop test (Golden, 1978), which is meant to measure response inhibition. Impairments on this scale could be due to inhibitory difficulties and/or problems with reading and rapid naming. We consider rapid naming rele-

vant to ADHD, given that such difficulties are also found in participants with ADHD, even in the absence of learning disability (LD; Rucklidge & Tannock, 2002). In addition, by correcting for LD in some analyses, we have concluded that the impairments associated with poor test performance were not simply due to comorbid LD in the sample.

6. The Freedom from Distractibility Index (Wechsler, 1974, 1981), which gauges attention and working memory.

To justify our analytical decision to treat the amalgamation of these variables as a measure of EFD, we subjected them to a factor analysis. The first factor attained an eigenvalue of 2.66, whereas the second factor had an eigenvalue of only 0.26, well below the commonly accepted cutoff of unity for factor retention. This analysis supports the notion that these variables are all measuring a single latent construct. Thus, although we recognize that in general EF is considered to be comprised of several factors, the subtests from these measures used in our battery measure a single factor, which supports our analytical approach.

Statistical Analysis

In defining EFDs, we were compelled to attend to conceptual and methodological issues. First, we wanted our definition to be clinically applicable, such that practitioners could readily apply our algorithm without excessive and cumbersome computation. Second, we recognized that performance on tests of EF improves with age (Denckla, 1996); thus, our method needed to take the age of the participants into account.

To address age differences in test scores, we divided the control sample into four groups on the basis of age: 9 years of age or less ($n = 29$), 10–13 years of age ($n = 81$), 14–17 years of age ($n = 78$), and 18 years of age or above ($n = 34$). These age categories were chosen to reflect maturational growth and development as well as the distribution of control participants across age in years. For each EF variable within each age group, we defined a threshold by using the control data that indicated poor performance if the score was 1.5 standard deviations from the mean of normally distributed variables or within the poorest 7th percentile of performance for nonnormally distributed variables.

We then created binary impairment indicators for the EF variables within age group for all participants (ADHD and control). Thus, we could sum the number of variables for which any given participant performed poorly, on the basis of cutoffs derived from the distribution of his or her age cohort. We defined a participant to have EFDs if the number of tests defined as impaired was less than two. Three issues contributed to this choice of cutoff. First, in our previous report (Doyle, Biederman, Seidman, Weber, & Faraone, 2000), we found that less than two tests impaired showed the best discrimination between ADHD and non-ADHD participants. Second, whereas one impaired test may be due to chance, two or more impaired tests would be likely to be interpreted as a deficit by a clinician. Third, we felt that it was inappropriate to place individuals with two abnormal test scores in the nonimpaired group.

To validate our decision to create a binary measure of EFD, we correlated the factor score derived from the factor analysis described earlier to the number of tests impaired, as defined earlier. We found a modest sized, significant correlation ($r = -.59$, $p < .01$), supporting our approach.

After applying our EFD algorithm, we were able to define four groups: (a) control participants without EFD (control participants − EFD, $N = 196$), (b) control participants with EFD (control participants + EFD, $N = 26$), (c) ADHD without EFD (ADHD − EFD, $N = 173$), and (d) ADHD with EFD (ADHD + EFD, $N = 86$). To provide a meaningful illustration of our definition of impairment, we present the means of the EF variables across the four groups stratified by age in years in Table 1.

First, we compared the four groups on demographic factors. To address our hypothesis regarding the effect of EFDs, we modeled the outcomes as a function of group status and any confounding variables. Statistical models

Table 1

Executive Function Measures in Attention-Deficit/Hyperactivity Disorder (ADHD) Children and Controls, Stratified by Executive Function Deficits (EFDs)

Executive function measure	Control − EFD	Control + EFD	ADHD − EFD	ADHD + EFD
Ages 6–9 (years)				
Stroop color–word	23.6 ± 7.2	13.3 ± 6.4	18.0 ± 4.7	14.3 ± 6.6
WCST perseverative errors	18.4 ± 10.9	22.3 ± 15.6	22.3 ± 13.8	33.2 ± 11.8
WCST failure to maintain set	1.9 ± 1.6	0.5 ± 0.6	1.3 ± 1.5	1.3 ± 0.9
Rey delay organization	6.3 ± 3.8	3.3 ± 4.5	4.8 ± 3.1	3.2 ± 2.4
Rey copy organization	6.5 ± 3.4	4.8 ± 2.8	5.8 ± 3.1	3.7 ± 2.7
Auditory CPT mistakes	9.9 ± 5.2	14.0 ± 9.3	10.7 ± 5.3	12.6 ± 6.1
CVLT–WRAML words learned	0.6 ± 0.1	0.6 ± 0.1	0.5 ± 0.1	0.4 ± 0.2
Freedom from Distractibility Index	23.0 ± 4.2	22.8 ± 7.1	21.1 ± 3.9	14.1 ± 3.1
Ages 10–13 (years)				
Stroop color–word	31.9 ± 8.5	25.1 ± 8.9	28.0 ± 6.5	23.2 ± 11.4
WCST perseverative errors	13.7 ± 9.2	19.7 ± 13.9	14.2 ± 9.0	23.0 ± 16.1
WCST failure to maintain set	1.0 ± 1.1	2.3 ± 1.5	1.0 ± 1.1	1.3 ± 1.3
Rey delay organization	7.4 ± 4.2	5.0 ± 3.3	7.2 ± 4.1	3.9 ± 2.6
Rey copy organization	9.3 ± 3.2	5.6 ± 3.5	8.8 ± 3.3	5.4 ± 3.6
Auditory CPT mistakes	4.9 ± 3.3	8.1 ± 4.7	4.8 ± 3.0	9.1 ± 5.1
CVLT–WRAML words learned	0.6 ± 0.1	0.6 ± 0.2	0.6 ± 0.1	0.5 ± 0.2
Freedom from Distractibility Index	23.7 ± 5.3	19.4 ± 5.9	20.9 ± 4.4	16.1 ± 4.6
Ages 14–17 (years)				
Stroop color–word	42.2 ± 7.6	44.5 ± 8.8	40.8 ± 9.1	27.3 ± 8.1
WCST perseverative errors	10.5 ± 6.8	22.7 ± 15.7	12.3 ± 8.5	21.4 ± 14.1
WCST failure to maintain set	0.7 ± 1.1	1.4 ± 1.3	0.8 ± 1.0	1.8 ± 1.7
Rey delay organization	9.5 ± 4.0	8.0 ± 5.3	9.9 ± 3.3	6.7 ± 4.2
Rey copy organization	11.0 ± 2.8	10.9 ± 2.9	11.1 ± 2.5	8.4 ± 4.3
Auditory CPT mistakes	2.0 ± 1.7	2.7 ± 2.5	2.7 ± 2.7	5.3 ± 5.6
CVLT–WRAML words learned	0.7 ± 0.1	0.6 ± 0.2	0.7 ± 0.1	0.5 ± 0.1
Freedom from Distractibility Index	23.9 ± 4.5	21.5 ± 6.5	21.0 ± 4.8	15.8 ± 4.2
Ages ≥ 18 (years)				
Stroop color–word	45.1 ± 8.5	38.5 ± 15.2	43.3 ± 8.5	34.2 ± 8.5
WCST perseverative errors	6.8 ± 4.0	15.8 ± 10.2	7.5 ± 3.2	18.5 ± 8.5
WCST failure to maintain set	0.4 ± 0.9	2.3 ± 3.2	0.9 ± 1.2	1.9 ± 1.2
Rey delay organization	10.5 ± 3.6	11.0 ± 2.3	10.1 ± 3.6	7.6 ± 4.1
Rey copy organization	12.1 ± 1.8	10.3 ± 3.4	10.8 ± 3.3	8.0 ± 3.0
Auditory CPT mistakes	1.2 ± 1.2	2.3 ± 2.6	1.5 ± 1.8	4.4 ± 3.3
CVLT–WRAML words learned	0.7 ± 0.1	0.6 ± 0.0	0.7 ± 0.1	0.6 ± 0.2
Freedom from Distractibility Index	24.4 ± 5.6	21.8 ± 2.6	23.4 ± 4.3	15.1 ± 4.6

Note. Values in the table represent means plus or minus the standard deviations. WCST = Wisconsin Card Sorting Test; CPT = Continuous Performance Test; CVLT = California Verbal Learning Test; WRAML = Wide Range Achievement of Memory and Learning.

were fit with the statistical software package STATA (Stata Corporation, 1997). We used generalized estimating equation models with the appropriate link and family specification depending on the distribution of the outcome variable (i.e., binary or continuous). All statistical tests were two-tailed. The statistical significance of each covariate in these regression models was determined by Wald's test. To assess normality, we used the Shapiro–Wilk test. We used an alpha level of .05 to assert statistical significance.

Results

We found that 86 (33%) of the participants with ADHD were classified as having EFDs, whereas only 26 (12%) of the control participants were classified as having EFDs, $\chi^2(1, N = 481) = 30.9, p < .01$. This association between ADHD and EFD remained after statistical adjustment for gender, age, IQ, LD, and SES. As shown in Table 2, there were small but statistically significant differences across the groups in years of age. Control participants − EFD were on average 1.3 years older than ADHD + EFD participants. In addition, children and adolescents without EFDs (ADHD and control participants) had a significantly lower mean

SES score (indicating higher social class status) as compared with the ADHD + EFD group. No differences were noted in gender across the four groups, and the two ADHD groups did not differ in the rate of current medication status. Because the key comparison was between the two ADHD groups and the age difference noted earlier between the control − EFD and ADHD + EFD groups was not substantial from a developmental perspective, all subsequent analyses were statistically adjusted for SES but not for years of age.

Clinical Features of ADHD

We first investigated whether EFD was associated with the clinical features of ADHD. There were no differences between proband participants with ADHD with and without EFDs in the age at onset of ADHD (3.1 ± 2.2 vs. 3.2 ± 2.4, respectively), $t(257) = -0.59, p = .55$. Only 2 of 14 *DSM–III–R* symptoms were more common in ADHD + EFD proband participants relative to ADHD − EFD proband participants. There were no differences between proband participants with ADHD with and without EFDs

Table 2

Demographic Characteristics in Attention-Deficit/Hyperactivity Disorder (ADHD) Children and Controls, Stratified by Executive Functioning Deficits (EFDs)

Demographic feature	Control − EFD (N = 196)			Control + EFD (N = 26)			ADHD − EFD (N = 173)			ADHD + EFD (N = 86)			Omnibus analyses		
	M	SD	n (%)	M	SD	n (%)	M	SD	n (%)	M	SD	n (%)	df	F	p
Age (years)	13.7	3.7[a]		13.4	4.4		13.1	3.5		12.3	3.7		3, 477	3.3	.019
SES	1.6	0.8[a]**		1.7	0.9		1.7	0.8[a]**		2.2	1.1		3, 476	11.1	< .001
													df	χ^2	p
Gender													3	0.8	.862
Girls			106 (54)			16 (62)			91 (53)			47 (55)			
Boys			90 (46)			10 (38)			82 (47)			39 (45)			
Medication status			0 (0)			0 (0)			104 (60)			51 (59)		3	196.0	< .001

Note. SES = socioecomonic status; Medication status = any psychotropic medication at the time of neuropsychological assessment.
[a] Versus ADHD + EFD.
* p < .05. ** p < .01.

on the number of hyperactive–impulsive symptoms (6.2 ± 1.7 vs. 6.0 ± 1.7, respectively), $t(258) = 0.89$, $p = .38$, or total symptoms (11.1 ± 2.7 vs. 10.2 ± 3.4, respectively), $t(258) = 1.87$, $p = .06$. However, there were more inattentive symptoms among ADHD + EFD children and adolescents compared with ADHD − EFD children and adolescents (5.6 ± 0.7 vs. 5.2 ± 1.1, respectively), $t(258) = 2.79$, $p = .01$.

EFDs and Academic Functioning

As shown in Table 3, there were several differences across groups in achievement and school functioning. Children and adolescents with ADHD with and without EFDs performed worse than control participants on achievement scores and measures of school functioning, and ADHD + EFD children and adolescents demonstrated significantly poorer performance on every academic outcome assessed relative to the ADHD − EFD group. In contrast, school performance did not differ meaningfully in control participants irrespective of the presence or absence of EFDs.

To further test the effect of EFDs within ADHD, we ran additional analyses on the academic outcomes including the participants with ADHD only. We found that ADHD + EFD participants were over 2 times more likely to repeat a grade compared with ADHD − EFD participants, even after controlling for SES, LD, and IQ. Children and adolescents with ADHD + EFD were almost 3 times more likely to have a LD relative to ADHD − EFD children and adolescents, controlling for SES and IQ. In addition, among children and adolescents with ADHD, EFDs were associated with a statistically significant average decrease of over 10 points on the IQ score, controlling for LD and SES, and 4 points on each WRAT–R score, controlling for SES, LD, and IQ. To further show the robustness of the effect of EFDs among children and adolescents with ADHD, we repeated these analyses using a continuous measure of EF. We standardized the EF measures (within age strata) and created a linear combination by summing over these z scores. We then standardized this sum and used the resulting z score as an independent variable in models predicting the academic outcomes, with the same statistical adjustments used earlier. As in the analysis that used a binary measure of EFD, the continuous measure of EF showed that poorer EF functioning significantly predicted worsening academic performance as measured by repeating a grade, LD, IQ, WRAT–R arithmetic, and WRAT–R reading.

It is possible that, because of our use of two measures from the same test, namely the copy and delay organization score from the Rey–Osterrieth Complex Figure and the perseverative errors and failure to maintain set score from the WCST, participants were designated as having EFD on the basis of a single test. This gave these two tests more influence over the EFD measure than the other tests. To account for this potential problem, we recalculated our EFD measure, excluding any participants who were designated as EFD only because of deficits on the two Rey–Osterrieth Complex Figure variables or the two WCST variables. Only 7 participants (2 from the control group and 5 from the group with ADHD) were dropped from the analysis because of this problem. We repeated the analysis of the academic functioning outcomes without these 7 participants, and the results did not change.

EFDs and Social and Psychiatric Outcomes

Table 4 shows the social and psychiatric outcomes in children with ADHD and in control participants, stratified by EFDs. Although participants with ADHD had significantly more impaired performance on global functioning (Global Assessment of Functioning scores) and interpersonal functioning (SAICA total scores) than control participants, these differences were not associated with EFDs. Similar patterns emerged for findings of psychiatric comorbidity: Proband participants with ADHD had higher rates of comorbid disruptive mood and anxiety disorders than control participants, irrespective of the presence or absence of EFDs, and no differences were identified in comparisons within ADHD and control participants with and without EFDs.

Effect of Development on EFDs and Functional Outcomes

We tested whether the association between EFDs and academic, social, and psychiatric outcomes is influenced by neuropsychological development across childhood. We used age as a proxy for

Table 3
Academic Functioning in Attention-Deficit/Hyperactivity Disorder (ADHD) Children and Controls, Stratified by Executive Functioning Deficits (EFDs)

Academic outcome	Control − EFD (N = 196)			Control + EFD (N = 26)			ADHD − EFD (N = 173)			ADHD + EFD (N = 86)			Omnibus analyses			EFD within ADHD	
	M	SD	n (%)	M	SD	n (%)	M	SD	n (%)	M	SD	n (%)	df	χ^2	p<	OR	95% CI
Repeated grade			15 (8)[a]***b**			3 (12)[b]*			32 (19)[b]**			36 (42)	3	34.8	.001	**2.2**	**1.1, 4.4**$_a$
Extra help			49 (25)[a]***b**			9 (35)[a]***b**			122 (71)[b]**			74 (86)	3	108.2	.001	1.4	0.6, 3.2$_a$
Special class			4 (2)[a]***b**			0 (0)			41 (24)[b]**			39 (45)	3	40.5	.001	1.3	0.7, 2.6$_a$
Any LD			18 (9)[a]***b**			3 (12)[b]*			33 (20)[b]**			37 (44)	3	32.5	.001	**2.9**	**1.5, 5.7**$_b$
													df	F	p<	β	95% CI
Full scale IQ	113.9	11.1***b**		106.9	11.6b**		109.2	11.9b**		97.5	11.6		3, 456	28.4	.001	**−10.5**	**−13.6, −7.3**$_c$
WRAT arithmetic	108.5	15.4***b**		103.6	15.9b**		99.4	13.5b**		84.8	15.0		3, 457	41.4	.001	**−4.5**	**−7.9, −1.2**$_a$
WRAT reading	111.1	10.3***b**		107.7	10.4b**		105.6	13.3b**		91.5	15.7		3, 455	37.6	.001	−4.4	−7.9, −0.9$_a$

Note. All omnibus analyses were adjusted for socioeconomic status (SES). Confidence interval (CI) values with the subscript *a* were adjusted for SES, learning disability (LD), and IQ; CI values with the subscript *b* were adjusted for SES and IQ; CI values with the subscript *c* were adjusted for SES and LD. The numbers in bold indicate significance. OR = odds ratio; WRAT = Wide Range Achievement Test.
[a] Versus ADHD − EFD.   [b] Versus ADHD + EFD.
* p < .05.   ** p < .01.

neuropsychological development and created a binary indicator, with participants less than 12 years of age in one group and participants greater than or equal to 12 years of age in the other. We tested the Age Group × EFD interaction in a series of regression models with the functional measures as the dependent variables to see whether the association between EFDs and the outcomes differed by developmental stage. None of the interaction effects were significant with the exception of mood disorders, which showed a trend toward significance ($p = .06$). In the younger group, the rates of mood disorders in those with (13%) and without (15%) EFDs were very similar. However, in the older group, the rate of mood disorders was higher in those with EFDs (45%) compared with those without EFDs (27%).

Discussion

In a large sample of children and adolescents of both genders with and without ADHD, EFDs were significantly more common in proband participants with ADHD relative to control participants. Among participants with ADHD, EFDs increased the risk for grade retention, LD, and lower academic achievement, even after stringent statistical controls. These effects could not be accounted for by differences in the clinical features of ADHD or medication status. In contrast, we did not find statistical evidence that EFDs affected the functional outcome of control participants. These findings provide support for the use of an empirically derived definition of EFD and the selective detrimental effect of EFDs on the academic functioning of children and adolescents with ADHD. These results also suggest that the frequently reported association between ADHD and academic deficits could be particularly strong in those with associated EFDs.

Our finding of a higher rate of EFDs in children with ADHD relative to control participants is consistent with a wide body of work documenting EFDs and other neuropsychological deficits in ADHD samples (Barkley, 1997; Douglas, 1972; Seidman et al., 1997, 2000). Across the 18 studies reviewed by Pennington and Ozonoff (1996), 15 documented significant differences between participants with ADHD and control participants on EF measures. Furthermore, among studies that controlled for confounding factors (i.e., age, gender, IQ, SES) this effect was demonstrated. In our data, EFDs were still significantly associated with ADHD even after statistical adjustment for gender, age, IQ, SES, and LD. Taken together, these results provide strong evidence that ADHD is independently associated with significant EFDs. However, despite this association, the majority of children with ADHD in our sample did not have EFDs. We recognize that contemporary theories about the extensive overlap between ADHD and EFDs, especially when the latter is measured behaviorally, may be at odds with this finding. Although the ADHD − EFD group did not show executive dysfunction by our definition, when compared with control participants, it was still significantly impaired on all academic and psychosocial outcomes and most psychiatric outcomes. In addition, there were no differences between the ADHD − EFD and ADHD + EFD groups on the clinical features of ADHD, such as age of ADHD onset and number of ADHD symptoms. This suggests that EFD among children and adolescents with ADHD is not simply an expression of a subset of these clinical features. Nevertheless, the true association between ADHD and EFDs, as

Table 4

Social and Psychiatric Outcomes in Attention-Deficit/Hyperactivity Disorder (ADHD) Children and Controls, Stratified by Executive Functioning Deficits (EFDs)

Outcome measure	Control − EFD (N = 196)			Control + EFD (N = 26)			ADHD − EFD (N = 173)			ADHD + EFD (N = 86)			Omnibus analyses		
	M	SD	n (%)	M	SD	n (%)	M	SD	n (%)	M	SD	n (%)	df	F	p
Psychosocial functioning															
GAF score	67.5	6.7[a**b**]		66.6	6.6[a**b**]		54.2	7.2		53.6	7.2		3, 475	140.5	< .001
SAICA total	17.8	4.8[a**b**]		15.8	3.8[a**b**]		22.9	5.7		22.6	5.8		3, 392	31.2	< .001
													df	χ^2	p
Psychiatric comorbidity															
Mood			9 (5)[a**b**]			0 (0)			58 (34)			32 (37)	2	41.4	< .001
Multiple anxiety			11 (6)[a**b**]			3 (12)[b*]			55 (32)			34 (40)	3	43.7	< .001
Speech–language			14 (7)[ab**]			4 (15)			30 (17)			25 (29)	3	17.2	< .001
Disruptive			18 (9)[a**b**]			2 (8)[a**b**]			92 (53)			47 (55)	3	83.4	< .001
Tics–Tourettes			8 (9)[a**b**]			0 (0)			30 (36)			14 (35)	2	17.3	< .001
Substance use			13 (7)			2 (8)			17 (10)			8 (9)	3	1.5	.691
Smoking			12 (6)			1 (4)			22 (13)			11 (13)	3	5.7	.125

Note. All omnibus analyses were adjusted for socioeconomic status (SES). GAF = Global Assessment of Functioning; SAICA = Social Adjustment Inventory for Children and Adolescents.
[a] Versus ADHD − EFD.   [b] Versus ADHD + EFD.
* p < .05.   ** p < .01.

defined in various ways, is an empirical question that awaits further research.

Although we and others have documented that ADHD is associated with significant academic deficits (Barkley, Anastopoulos, Guevremont, & Fletcher, 1991; Cunningham & Barkley, 1978; Faraone et al., 1993; Fischer, Barkley, Edelbrock, & Smallish, 1990), our results document that children with ADHD and comorbid EFDs have significantly worse academic deficits compared with children and adolescents with ADHD without EFDs. These results suggest that children with ADHD plus EFDs suffer from the detrimental synergism of the two conditions such that their academic performance is severely compromised. However, it should be noted that control participants with EFD consistently demonstrated small, albeit nonsignificant deficits in academic outcomes relative to control participants without EFD. Although it is possible that we lacked adequate statistical power to detect meaningful differences associated with EFDs in non-ADHD comparison children and adolescents, the negative educational outcomes observed in our control participants with EFDs were clearly smaller than those observed between the groups with ADHD. If replicated, these results suggest that EFDs compound the already compromised educational functioning of a child with ADHD but may have a much more limited impact in children without ADHD, because these children do not reach the dysfunction threshold that triggers grade retention, extra help, and placement in special classes. More work is needed to further evaluate these hypotheses and to ascertain whether the effect of EFDs on school performance is realized only when it overlaps with ADHD.

Our hypothesis that EFDs are associated with broad deficits in multiple domains of functioning was not confirmed. Neither psychiatric comorbidity nor social functioning were associated with EFDs, regardless of ADHD status. Although the reasons for these unexpected findings are not clear, several possible explanations should be considered. Our definition of EFDs may not be sensitive enough to detect deficits outside the educational domain, or the instruments we used may not have been sensitive enough to distinguish between differences in various domains of functioning. However, another explanation is that the effect of EFDs in children may be manifested only in academic performance and not in psychiatric comorbidity or social functioning.

Another possibility is that our participants have not passed through the age of risk for the detrimental impact of EFDs in nonacademic domains. It is possible that such deficits are not manifested until adulthood. Such a possibility is consistent with recent work by Barkley (2001) in which he considered EF from an evolutionary perspective and theorized that environmental pressures may have existed in our species' history that could have selected for the evolution of EF. Barkley argued that the EF system evolved as a tool used by early humans to successfully negotiate an increasingly competitive social environment. In this context, it seems plausible that the deficits associated with EFDs may become more pervasive and impairing as a person enters adulthood. Only through the increasingly complex social interactions that adults need to navigate could EFDs result in psychiatric comorbidity, social, occupational, financial, or even global functioning impairments. Thus, from this perspective, academic deficits could reasonably be the only forum in which EFD could cause problems for children.

Our analysis of age as a modifying factor of the EFD–functional outcome association largely did not provide evidence that the developmental trajectories of neuropsychological functioning influence academic or psychiatric outcomes. The exception to this was mood disorders, with older children exhibiting a vulnerability to this outcome associated with EFDs that younger children did

not. However, there are several caveats that compel a cautious interpretation of these data. Chronological age is merely a proxy for neuropsychological development across childhood, and its use introduced a degree of measurement error into our analysis. In addition, our models were underpowered to detect small- to medium-sized interaction effects.

The validity of our definition of EFDs is partially supported by our findings. If we consider EFDs to exist in the general population, we would expect the rate of EFDs to be relatively uncommon in a control sample drawn from that population, because the notion of disorder implies a phenotype in the extremes of a distribution. This is what we found: The rate of EFDs in our control participants was only 11%. In addition, our cutoff for EFDs, two or more tests 1.5 standard deviations from the mean of the control participants, has face validity as a clinically relevant standard of EFDs. However, we recognize that our method of dichotomizing the number of tests impaired to create a binary EFD measure may result in a loss of some information. Although the strong correlation between the factor score and the number of tests impaired somewhat assuages this concern, we consider this loss of information to be a reasonable trade-off for the applicability and clinical relevance of our method.

The finding that ADHD + EFD children were from families with a lower SES than ADHD − EFD families is intriguing. There are several possible explanations for this finding. First, if EFs are at least partially genetically determined, it could be that the parents of the ADHD + EFD sample also have EFDs to some extent, and this impairment has led to lower educational and occupational achievement. Another possibility is that exposure to low SES environments may have impeded the neuropsychological development of the ADHD + EFD group. Unfortunately, our study design cannot tease apart the direction of the effect.

These results suggest several avenues for further research. Future efforts should investigate the impact that EFD has on several domains of functioning in adult samples with and without ADHD because an improved understanding of EFDs in adulthood could lead to more developmentally sensitive diagnostic criteria (Faraone, 2000; Faraone, Biederman, Feighner, & Monuteaux, 2000; Faraone, Biederman, Spencer, et al., 2000). In addition, studies of children and adolescent samples should specifically address the question of developmental influences on the association between EFDs and functional outcomes to confirm our findings. Furthermore, validation of our proposed definition of EFDs by others could lead to a useful tool for identifying children with EFDs in clinical, epidemiological, and research samples. If so, it would be valuable to examine the use of an EFD screening measure that identifies young children who are at high risk for future academic difficulties for intervention purposes. Finally, the association between EFDs and low SES should be explored in future samples of adults and children to tease apart the direction of the effect.

Our results should be considered in the light of methodological limitations. The sample of participants with ADHD was clinically referred; thus, we do not know to what degree our findings will generalize to nonreferred children with ADHD in the community. This is a concern also noted by Pennington and Ozonoff (1996), who stated that only one of their reviewed studies used a population sample of children with ADHD, and no association with EF was reported. Thus, our results should be confirmed in community samples. In addition, because the vast majority of our participants were Caucasians, our results may not generalize to other ethnic groups. Another methodological shortcoming pertains to our assessments of psychopathology in children younger than 12 years of age. Although children and adolescents older than 12 years of age were directly interviewed, the lack of direct psychiatric interviews with children younger than 12 years of age may have decreased the sensitivity of some diagnoses, particularly with internalizing disorders. However, because children younger than 12 years of age have limited expressive and receptive language abilities, there is a question about whether their lifetime history of psychopathology and behavior can be reliably assessed through self-report (Loeber, Green, Lahey, & Stouthamer-Loeber, 1991). Although limited, studies of interview techniques for children under the age of 12 years suggest that their responses are unreliable (Achenbach, McConaughy, & Howell, 1987).

In addition, as noted earlier, we may have had limited power to detect differences between control participants with and without EFDs, although the mean differences were of limited clinical significance nonetheless. Likewise, we relied on our own data to generate the cutoffs for defining poor performance for each EF measure within specific age groups. Although there are published norms for some of the tests in our battery, the quality of these norms varies widely in terms of the numbers of participants in the normative group and its representativeness of the general population. The use of norms could introduce errors because the size, age range, and gender distribution of the samples used to generate the norms vary from test to test. Children may be more likely to be categorized as impaired on some EF measures than others because of differences in their normative samples. Using our own sample, we provided a valid case-control comparison and a meaningful estimate of the prevalence of EFD among our group with ADHD. Because that estimate cannot be generalized to the population, improved norms or studies of population samples are needed to clarify this issue.

In a similar vein, the use of our own control participants to define the cutoffs for impairment may have led to an unusual cutpoint because outlying observations could have a disproportionately large influence on the result. However, our control samples were most likely large enough (ns = 29, 81, 78, and 34) to minimize the effect of extreme observations. Finally, the mean IQ of our control sample was higher than average. These high scores are consistent with our exclusion criteria. We excluded participants with a full scale IQ of less than 80 and participants from the lowest socioeconomic stratum. In addition, children with ADHD were excluded from our normal control sample. Given that both social class (Matarazzo, 1972) and ADHD are predictive of intellectual functioning, our control group should have higher than average WISC–R scores. We also note that WISC–R IQ scores are approximately 5 points higher than those obtained contemporaneously from its revision, the WISC–III (Wechsler, 1991). As the WISC–III manual indicates, IQ scores are usually inflated when a contemporary standardization sample is not available (Flynn, 1987; Wechsler, 1991). Because IQ is correlated with EF, this may have resulted in a wider definition of impairment than if a truly average control group was used, leading to high rates of EFD in the group with ADHD. Although this prevents us from drawing conclusions about the prevalence of EFD in population samples, it does not compromise our case-control comparisons. Although the children with ADHD in our study also showed high mean IQ scores, the

absolute difference between the groups is still consistent with ADHD-associated IQ deficits. In addition, our finding that only one third of the sample with ADHD classified as having EFD is much less than contemporary theories about the extensive overlap between ADHD and EFD would suggest; a contrast that argues against the idea that control-defined impairment cutoffs classified too many ADHD children as impaired.

Another limitation is that because of continuous neuropsychological development throughout childhood, we cannot say for certain that performance deficits on our battery exhibited by children at one age are due to the same mechanism as that by children of an older age. In either case, the deficit may or may not be due to EFDs. Although age did not surface as a substantial factor in our analyses, future studies should use a developmentally appropriate battery and specifically address these important questions. In addition, our discrepancy measure of learning disabilities, although straightforward and consistent with legal standards, is only one of several methods for defining LDs. Thus, analyses that use other definitions (e.g., phonological decoding problems as an index of reading disability) should be undertaken. However, in another analysis with these same data, researchers who used a more inclusive definition of LDs did not alter the results (Seidman, Biederman, Monuteaux, Doyle, & Faraone, 2001).

Our percentage words learned variable could be criticized for lacking a theoretical relationship to the other variables in our analysis or for having its association with the EF construct only through its correlation with IQ. However, the factor analysis supported the use of the percentage words learned variable as being conceptually related to the other variables, because it loaded heavily on the factor (.58). In addition, after regressing IQ on percentage of words learned, we found the residuals (interpreted as the variability in percentage of words learned remaining after accounting for IQ) to also load on the single EF factor with the rest of the variables. Finally, our use of *DSM–III–R* ADHD criteria precluded the use of formally defined ADHD subtypes. The investigation of our EFD measure and *DSM–IV* ADHD subtypes should be a subject of additional analyses.

Despite these considerations, our results show that the presence of EFDs in children and adolescents with ADHD increases significantly the already compromised educational functioning of these children and that this effect is independent of social class, IQ, or the presence of LD. Because it is not clear how EFDs may or may not respond to standard pharmacological treatments for ADHD, these results suggest that children with ADHD and EFDs may require additional academic intervention to prevent academic failure. Future studies of adults with and without ADHD assessed with adequate neuropsychological measurements are needed to help clarify the full impact of EFDs beyond the educational domain.

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