Validity of the Executive Function Theory of Attention-Deficit/Hyperactivity Disorder: A Meta-Analytic Review

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One of the most prominent neuropsychologic theories of attention-deficit/hyperactivity disorder (ADHD) suggests that its symptoms arise from a primary deficit in executive functions (EF), defined as neurocognitive processes that maintain an appropriate problem-solving set to attain a later goal. To examine the validity of the EF theory, we conducted a meta-analysis of 83 studies that administered EF measures to groups with ADHD (total N = *3734) and without ADHD (*N = *2969). Groups with ADHD exhibited significant impairment on all EF tasks. Effect sizes for all measures fell in the medium range (.46–.69), but the strongest and most consistent effects were obtained on measures of response inhibition, vigilance, working memory, and planning. Weaknesses in EF were significant in both clinic-referred and community samples and were not explained by group differences in intelligence, academic achievement, or symptoms of other disorders. ADHD is associated with significant weaknesses in several key EF domains. However, moderate effect sizes and lack of universality of EF deficits among individuals with ADHD suggest that EF weaknesses are neither necessary nor sufficient to cause all cases of ADHD. Difficulties with EF appear to be one important component of the complex neuropsychology of ADHD.*

Key Words: Attention-deficit/hyperactivity disorder, executive function, meta-analysis, neuropsychology, subtypes

To navigate successfully the ever-changing environmental context, people must continuously evaluate and select from many possible actions. These actions are often directed toward achieving a positive outcome in a simulated future context and must therefore compete with alternative actions that might maximize initial benefits but have larger long-term costs (Pennington 2002). Executive functions (EFs) are neurocognitive processes that maintain an appropriate problem solving set to attain a future goal (Welsh and Pennington 1988). In a simplified model, EFs represent "top-down" cognitive inputs that facilitate decision making by maintaining information about possible choices in working memory and integrating this knowledge with information about the current context to identify the optimal action for the situation. EFs involve multiple distributed neural networks that include the thalamus, basal ganglia, and prefrontal cortex (Casey, in press; Fuster 1997; Middleton and Strick 2001, 2002; Pennington 2002).

Several authors have proposed that symptoms of attention-deficit/hyperactivity disorder (ADHD) arise from a primary deficit in a specific EF domain such as response inhibition or working memory or a more general weakness in executive control (Barkley 1997; Castellanos and Tannock 2002; Pennington and Ozonoff 1996; Schachar et al 2000). This hypothesis is based on the observation that prefrontal lesions sometimes produce behavioral hyperactivity, distractibility, or impulsivity, as well as deficits on EF tasks (Fuster 1997; Stuss and Benson 1986).

Nearly 10 years ago, Pennington and Ozonoff (1996) completed a meta-analytic review that systematically examined 18 studies of the neuropsychologic correlates of ADHD. They concluded that ADHD is associated with specific weaknesses in at least a subset of EF domains. Many of these samples were small, however, and for most EF measures, only a handful of studies were available. In contrast, a literature search conducted in March 2004 found more than 100 papers between 1980 and 2004 that compared EF performance in groups with and without ADHD. This extensive new literature suggests that an updated meta-analysis is warranted to examine the implications of these new data for the EF hypothesis.

Criteria for a Primary Neurocognitive Deficit

This review examines the validity of the hypothesis that ADHD symptoms arise from a primary deficit in executive control that is necessary and sufficient to cause ADHD. At least four criteria must be met for EF weaknesses to be considered the primary deficit in ADHD.

1. Groups with ADHD must consistently exhibit weaknesses on EF measures. In addition, many argue that these weaknesses must remain significant after controlling for potential confounding variables such as age, language, general intelligence, reading ability, and symptoms of other psychopathology, but the importance of this specificity criterion for multifactorial disorders is in considerable dispute in the field (Garber and Hollon 1991; Sergeant et al 2003; Willcutt et al 2005a, 2005b).
2. EF weaknesses must account for a substantial proportion of the variance in ADHD symptoms in the population.
3. EF weaknesses must be present in most individuals with ADHD.
4. EF weaknesses and ADHD symptoms must be attributable to common etiologic influences. Because ADHD is highly heritable, this suggests that EF weaknesses must be coheritable with ADHD.

The first section of this review describes a meta-analysis we conducted to evaluate the extent to which EF weaknesses meet the first two criteria for a primary deficit. We then briefly summarize the conclusions of several accompanying articles in this special issue that are relevant to the third and fourth criteria (Doyle et al 2005; Faraone et al 2005; Nigg et al 2005, all in this issue). In the final section, we consider a plausible alternative to the primary deficit model that suggests that EF weaknesses may be one of several important components in a multifactorial neuropsychologic model of ADHD.

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Methodology of the Meta-Analysis

Selection of EF Measures

Previous theorists have criticized the construct of EFs as weakly defined and overly broad (Pennington and Ozonoff 1996; Sergeant et al 2003). This assertion is supported by factor analyses of batteries of EF measures (Barkley et al 2001; Miyake et al 2000; Robbins et al 1998; Willcutt et al 2001, 2005b). These studies suggest that EF tasks comprise at least four factors: 1) response inhibition and execution, 2) working memory and updating, 3) set-shifting and task-switching, and 4) interference control. In addition, most EF models distinguish between verbal and spatial working memory, and many include additional processes such as planning and organization, vigilance, and visuospatial orienting (Castellanos and Tannock 2002; Huang-Pollock and Nigg 2003; Willcutt et al 2005a).

Because of space constraints, we did not attempt to include all previous neuropsychologic studies of ADHD or all possible EF tasks. We identified 13 measures that were frequently administered in previous studies or highlighted as domains of interest in theoretical models of ADHD. Stroop interference control and visuospatial orienting tasks were not included to avoid redundancy with recent meta-analyses (Homack and Riccio 2004; Huang-Pollock and Nigg 2003; Van Mourik et al 2005), but the conclusions of these reviews are noted.

Table 1 provides a brief description of each task in the meta-analysis and summarizes results from factor analytic, lesion, electrophysiologic, and neuroimaging studies that tested the external validity of the task as an EF measure. Lesion and neuroimaging studies provide direct evidence that most of the tasks activate neural networks that include the prefrontal cortex. Although neurophysiologic studies have not examined the Trail-making test or the two verbal working memory tasks included in the meta-analysis, factor analytic studies indicate that all three of these tasks load on the same factor as other EF measures (e.g., Willcutt et al 2001). Moreover, neuroimaging studies of other working memory measures consistently report prefrontal activation during task performance (e.g., Kubler et al 2003; Wager and Smith 2003).

Identification of Studies

Computerized searches of the PsycINFO and Medline databases were conducted in March 2004. The terms ADHD, ADD, inattention, hyperactivity, hyperkinetic, and attention-deficit disorder were cross-referenced separately with neuropsychology, neurocognitive, cognitive, executive, frontal, inhibition, planning, working memory, and vigilance. The tables of contents of journals that commonly publish articles relevant to this topic were also reviewed (e.g., *Biological Psychiatry, Developmental Neuropsychology, Journal of Abnormal Child Psychology, Journal of the American Academy of Child and Adolescent Psychiatry, Journal of Child Psychology and Psychiatry*). In addition, the reference list of each paper was reviewed for additional studies, and coauthors and several additional researchers in the field screened the final list of studies. Because of the extensive published literature on the relation between EF and ADHD in children and adolescents, unpublished studies and studies of adults were not included.

The literature search yielded 83 studies that provided sufficient information to allow us to calculate effect sizes for the relevant EF measures (total N = 3734 with ADHD and 2969 without ADHD). Most studies used diagnostic criteria from one of the last three editions of the *Diagnostic and Statistical Manual of Mental Disorders* to define groups with and without ADHD (DSM-IV = 29 studies, DSM-III-R = 33 studies, DSM-III = 11 studies). A small subset of studies defined the ADHD group based on ICD-10 criteria (three studies) or clinically extreme scores on standardized parent or teacher rating scales (seven studies). All studies were included in the overall meta-analysis, and we then compared results from studies that defined ADHD based on these various diagnostic criteria.

Data Analysis

The procedure described by Cohen (1988) was used to compute a standardized effect size (d) for the mean difference between groups with and without ADHD on each EF measure administered in each study (Table 2). The mean effect size across all studies that administered each measure and the corresponding 95% confidence interval (CI) were then calculated using the method described by Hedges and Olkin (1985). This procedure weights the effect size from each study by its corresponding sample size to obtain the best estimate of the true population parameter (Cooper 1998). Homogeneity of effect sizes was examined for each measure by calculating the Q_t statistic (Hedges and Olkin 1985). A nonsignificant Q_t value suggests that variance in effect sizes across studies is explained by sampling error, whereas a significant value suggests that systematic differences among the studies may be causing meaningful differences in the effects.

Results of the Meta-Analysis

The meta-analysis found significant differences between groups with and without ADHD on all 13 EF tasks (Table 2). Significant group differences were obtained in 109 of 168 (65%) total comparisons; the weighted mean effect size across all comparisons was .54 (95% CI = .51–.57). Weighted mean effect sizes for all measures fell in the range considered a medium effect (d = .43–.69; Cohen 1988).

Significant group differences were obtained most consistently for stop-signal reaction time (SSRT; 82% of 27 studies) and Continuous Performance Test omission errors (77% of 30 studies), and homogeneity analyses suggest that the variability in effect sizes across studies that used these measures is consistent with what would be expected by chance. The majority of studies also reported significant group differences on the measures of planning (59% of 27 studies), but these results were stronger and more consistent on the Tower of Hanoi and Porteus Mazes than the Tower of London and Rey–Osterreith Complex Figure Test.

Although fewer studies examined the relation between ADHD and working memory, initial results are promising. Six of eight studies found a significant group difference in spatial working memory. Homogeneity analyses revealed significant heterogeneity due to two small effect sizes that are outliers (Geurts et al 2004; Williams et al 2000). The mean effect size increases slightly if these two studies are excluded (d = .75), but the overall interpretation of the result remains the same. Significant group differences were reported by 55% of the 11 studies that included one of the verbal working memory tasks, and three of the nonsignificant results were moderate effects (d = .49–.53) that were not significant due to small sample size. Significant heterogeneity was also detected for the verbal working memory tasks due to one study with a negative effect size (Shue and Douglas 1992; mean weighted d without this study = .59 ± .11).

Wisconsin Card Sorting Test (WCST) perseverative errors were more weakly related to ADHD than many of the other EF tasks. The majority of studies did not detect a significant group

Table 1. Description and External Validity of Executive Function Tasks Included in the Meta-Analysis

Task Name and Description[a]	Putative EF Construct Assessed	Studies[b]					
		Factor	Lesion	ERP	fMRI	sMRI	PET
1. Stop-signal Reaction Time: The duration of the inhibitory process after a tone indicates that a motor response should be terminated.	Response inhibition	20, 21		2, 5	17		
2. Continuous Performance Test commission errors: Responses to a sequence other than the target sequence.	Response inhibition	20, 21		6, 18	19		
3. Continuous Performance Test omission errors: Failure to respond to the target sequence.	Vigilance	20				9	
4. Wisconsin Card Sorting Test perseverative errors: Errors that reflect difficulty shifting to a new rule when provided with feedback indicating that the previous rule is no longer correct.	Set-shifting	11, 20	4	1	10		
5. Trailmaking Test Part B: Timed task that requires the individual to connect a series of letters and numbers in ascending order while alternating between numbers and letters.	Set-shifting	20, 21					
6. Tower of Hanoi/London: The goal is to move a stack of objects from one position to another in the fewest possible moves while adhering to specific rules (i.e., a large ring cannot be placed on a smaller ring).	Planning	11	3		16		12
7. Porteus Mazes: The individual must plan ahead to reach the only exit of a series of mazes without backtracking or crossing any lines.	Planning	8	7				
8. Rey–Osterreith Complex Figure Test: The subject copies an abstract design, and the response is scored based on its organizational quality.	Planning/Organization					9	
9. Working Memory Sentence Span: The participant provides the last word for a set of simple sentences read by the examiner then is required to reproduce each word that he or she provided after all sentences are completed.	Verbal Working Memory[c]	20, 21					
10. Digits Backward: The subject repeats a series of digits in the reverse of the order that they were presented.	Verbal Working Memory[c]	20					
11. Self-ordered pointing: Requires the subject to select a different design on each card in a series without selecting the same design twice.	Spatial Working Memory[c]		14				
12. CANTAB Spatial Working Memory: The subject searches spatial locations to find tokens while remembering not to return to any locations where tokens were previously found.	Spatial Working Memory[c]	15, 20	13				12

[a]References for additional information about each task: Stop-signal reaction time, Logan et al (1997); CPT omission and commission errors, Newcorn et al (1989); Wisconsin Card Sorting Test, Heaton (1981); Trailmaking Test = Reitan and Wolfson (1985); Tower of Hanoi = Borys et al (1982); Tower of London, Shallice (1982); Porteus Mazes, Porteus (1965); Rey–Osterreith Complex Figure Test, Waber and Holmes (1985); Sentence span, Siegel and Ryan (1989); Digits backward, Wechsler (1991); Self-ordered pointing, Petrides and Milner (1982); CANTAB, Owen et al (1996a).

[b]Studies cited support validity of the task as a measure of executive function. Numbers in the column labeled Factor indicate studies that found that the task loaded on the same factor as other EF tasks. Numbers in the column labeled Lesion indicate studies that document that damage to the prefrontal cortex leads to impairment on the task. Numbers in the last four columns indicate studies that demonstrated that the task is associated with individual differences in prefrontal cortex structure or function in studies of event-related potentials (ERP), functional or structural magnetic resonance imaging (fMRI and sMRI), or positron emission tomography (PET). The numbers in each column refer to the following studies: 1, Barceló (1999); 2, Brandeis et al (1998); 3, Carlin et al (2000); 4, Demakis (2003); 5, Dimoska et al (2003); 6, Fallgatter; 7, Levin et al (2001); 8, Mariani and Barkley (1997); 9, Matáro et al (1997); 10, Mentzel et al (1998); 11, Miyake et al (2000); 12, Owen et al (1996a); 13, Owen et al (1996b); 14, Petrides and Milner (1982); 15, Robbins et al (1998); 16, Rowe et al (2001); 17, Rubia (1999); 18, Sartory et al (2002); 19, Vaidya et al (1998); 20, Willcutt et al (2005a); 21, Willcutt et al (2001).

[c]Working memory measures included in the meta-analysis require both storage and manipulation of information in memory. In a recent comprehensive meta-analysis of the relation between ADHD and working memory, Martinussen et al. (under review) also review studies of tasks that require storage of information in memory but do not require this information to be manipulated.

Table 2. Meta-Analysis of Studies That Administered Executive Function Measures to Groups with and Without ADHD

Study	ADHD Criteria[b]	Control/ ADHD (*n*)	Effect Size (*d*) of the Difference Between Groups[a]										
			Stop-Signal RT	CPT Comm. Errors	CPT Omiss. Errors	WCST Persev. Errors	Trails B Time	Tower of Hanoi	Tower of London	Porteus Mazes	ROCFT Copy Organiz.	Verbal Working Memory[c]	Spatial Working Memory[d]
Aman et al (1998)	III-R	22/22	1.20[q]	—	—	—	—	1.29[q]	—	—	—	—	—
August and Garfinkel (1989)	RS	43/50	—	.47[q]	.41[q]	—	—	—	—	—			
Barkley et al (1990)[e]	III	34/90	—	.60[q]	.70[q]	—	—	—	—	—	—	—	—
Barkley et al (1992)[e]	III	12/24	—	—	—	.46	.72	—	—	.40	.55	—	—
Barkley et al (2001)	IV	39/101	—	.07	.66[q]	—	—	—	—	—	—	.22[DB]	—
Barnett et al (2001)	IV	26/27	—	—	—	—	—	—	—	—	—	—	1.45[q,CA]
Bedard et al (2003)	IV	59/59	.57[q]	—	—	—	—	—	—	—	—	—	—
Börger et al (1999)	III-R	16/21	—	—	.65	—	—	—	—	—	—	—	—
Boucugnani (1989)	III	28/28	—	—	—	.78[q]	1.16[q]	—	—	—	—	—	—
Breen (1989)	III	13/26	—	.94	1.26[q]	—	—	—	—	—	—	—	—
Brewer et al (2001)	IV	22/26	—	.38	.75	1.46[q]	—	—	—	—	—	—	—
Cairney et al (2001)	IV	15/13	—	—	—	—	—	—	—	—	—	—	1.10[q,CA]
Carter et al (1995)	III-R	20/20	—	.47	.77[q]	.63	—	—	—	—	—	—	—
Chee et al (1989)	III	36/51	—	.70[q]	.57[q]	—	—	—	—	—	—	—	—
Chelune et al (1986)	III	24/24	—	—	—	.68[q]	—	—	—	—	—	—	—
Collings (2003)	IV	24/46	—	.28	.59[q]	—	—	—	—	—	—	—	—
Culbertson et al (1998)	III-R	56/99	—	—	—	—	—	—	.94[q]	—	—	—	—
Dimoska et al (2003)	IV	13/13	1.31[q]	—	—	—	—	—	—	—	—	—	—
Fallgatter et al (2004)	ICD-10	19/16	—	.00	1.16[q]	—	—	—	—	—	—	—	—
Fischer et al (1990)[f]	III-R	60/100	—	.57[q]	.66[q]	.42	—	—	—	—	—	—	—
Geurts et al (2004)	IV	41/54	.98[q]	—	—	—[g]	—	.41	—	—	—	—	.12[SP]
Gorenstein et al (1989)	III	26/21	—	—	—	.66[q]	1.02[q]	—	—	—	—	—	—
Grodzinsky et al (1992)[f]	III-R	64/66	—	.76[q]	.94[q]	.16	.32	—	—	.65[q]	.35[q]	—	—
Hinshaw et al (2002)	IV	88/100	—	.15	.49[q]	—	—	—	—	.58[q]	.36[q]	—	—
Hooks et al (1994)	III-R	52/40	—	.44[q]	.48[q]	—	—	—	—	—	—	—	—
Horn et al (1989)	III-R	31/54	—	.83[q]	.73[q]	—	—	—	—	—	—	—	—
Houghton et al (1999)	IV	28/94	—	—	—	.31	.56	—	.15	—	—	—	—
Jennings et al (1997)	III-R	26/40	.53[q]	—	—	—	—	—	—	—	—	—	—
Karatekin and Asarnow (1998)	III-R	27/31	—	—	—	—	—	—	—	—	—	.49[DB]	—
Kempton et al (1999)	IV	15/15	—	—	—	—	—	—	.48[q]	—	—	—	1.29[q,CA]
Klorman et al (1999)	IV	28/299	—	—	—	.07	—	No SD[q]	—	—	—	—	—
Konrad et al (2000)	IV	21/16	1.15[q]	—	—	—	—	—	—	—	—	—	—
Kuntsi et al (2001a)	RS	119/51	.22	—	—	—	—	—	—	—	—	.50[q,SS]	—
Loge et al (1990)	III-R	20/20	—	.85[q]	.65[q]	.16	—	—	—	—	—	—	—
Manassis et al (2000)	III-R	16/30	.17	—	—	—	—	—	—	—	—	—	—
Mariani et al (1997)	III-R	30/34	—	.46	.86[q]	—	—	—	—	.71[q]	—	—	—
Matáro et al (1997)	III-R	19/11	—	.39	1.79[q]	.35	.80	.19	—	—	.51	—	—
McGee et al (1989)	III	62/25	—	—	—	.12	.57[q]	—	—	—	.30	—	—
McInnes et al (2003)	IV	19/39	—	—	—	—	—	—	—	—	—	1.73[q,DB]	—
Newcorn et al (1989)	III-R	68/10	—	.60[q]	−.12	—	—	—	—	—	—	—	—
Nigg et al (1998)[h]	III-R	71/100	—	—	—	—	—	—	—	.60[q]	.33[q]	—	—
Nigg et al (2002)[i]	IV	41/64	.80[q]	—	—	—	.53[j,q]	—	.70[q]	—	—	—	—
Oie et al (1999)	III-R	30/20	—	—	—	—	—	—	—	—	—	.50[DB]	—
Oosterlaan and Sergeant (1996)	RS	17/15	.64[q]	—	—	—	—	—	—	—	—	—	—

(Table 2 continued on page 1340)

Table 2. (continued)

			Effect Size (*d*) of the Difference Between Groups[a]										
Study	ADHD Criteria[b]	Control/ ADHD (*n*)	Stop-Signal RT	CPT Comm. Errors	CPT Omiss. Errors	WCST Persev. Errors	Trails B Time	Tower of Hanoi	Tower of London	Porteus Mazes	ROCFT Copy Organiz.	Verbal Working Memory[c]	Spatial Working Memory[d]
Oosterlaan and Sergeant (1998)	RS	21/12	.77[q]	—	—	—	—	—	—	—	—	—	—
Overtoom et al (2002)	III-R	16/16	.92[q]	—	—	—	—	—	—	—	—	—	—
Ozonoff and Jensen (1999)	RS	33/31	—	—	—	—[g]	—	.39	—	—	—	—	—
Pennington et al (1993)	III-R	23/32	—	—	1.16[q]	.60	—	.65[q]	—	—	—	—	—
Pineda et al (1999)	III-R	72/100	—	—	—	.65[q]	—	—	—	—	.44[q]	—	—
Pliszka (1992)	III-R	34/92	—	.87[q]	.39	—	—	—	—	—	—	—	—
Pliszka et al (1997)	III-R	14/13	1.36[q]	—	—	—	—	—	—	—	—	—	—
Pliszka et al (2000)	IV	10/10	.75	—	—	—	—	—	—	—	—	—	—
Purvis and Tannock (2000)	III-R	17/34	.61[q]	.48[q]	1.06[q]	—	—	—	—	—	—	—	—
Reeve and Schandler (2001)	RS	10/10	—	—	—	1.07[q]	—	—	—	—	—	—	—
Rubia et al (1998)	RS	11/11	1.31[q]	—	—	—	—	—	—	—	—	—	—
Rubia et al (2001)	IV	23/16	.50	—	—	—	—	—	—	—	—	—	—
Rucklidge and Tannock (2002)	IV	37/59	.72[q]	—	—	—	—	—	—	—	—	.65[q,DB]	—
Rund et al (1998)	III-R	30/20		.31	.35	—	—	—	—	—	—	—	—
Sartory et al (2002)	III-R	19/20	—	1.01[q]	.77[q]	.10	1.65[q]	—	—	—	—	—	—
Sawyer et al (2001)	ICD-10	24/16	—	1.04[q]	.74[q]	—	—	—	—	—	—	—	—
Schachar et al (1988)	III	15/44	—	.35[q]	.37	—	—	—	—	—	—	—	—
Schachar and Logan (1990)	III	10/27	.72[q]	—	—	—	—	—	—	—	—	—	—
Schachar et al (2000)	IV	119/33	.42[q]	—	—	—	—	—	—	—	—	—	—
Schachar and Tannock (1995a)	III-R	16/40	.64[q]	—	—	—	—	—	—	—	—	—	—
Schachar et al (1995b)	III-R	22/14	.60[q]	—	—	—	—	—	—	—	—	—	—
Scheres et al (2001)	IV	41/51	.18[k]	—	—	—	—	—	—	—	—	—	—
Seidman et al (1997a)	III-R	36/43	—	—[l]	—[l]	.21	—	—	—	—	.34	—	—
Seidman et al (1997b)	III-R	99/118	—	—[l]	—[l]	.53[q]	—	—	—	—	.41[q]	—	—
Semrud-Clikeman et al (2000)	III	11/10	—	—	—	.54	—	—	—	—	—	—	—
Shue and Douglas (1992)	III-R	24/24	—	—	—	.88[q]	.72[q]	—	—	—	—	−.42[DB]	.85[q,SP]
Solanto et al (2001)	IV	29/77	.69[q]	—	—	—	—	—	—	—	—	—	—
Stevens et al (2002)	IV	76/76	.34[q]	—	—	—	—	—	—	—	—	—	—
Toplak et al (2003)	IV	39/59	—	—	—	—	—	—	—	—	—	.70[q,DB]	—
Tripp et al (1999)	IV	19/43	—	.48	−.01	—	.19	—	—	—	—	—	—
Tripp et al (2002)	IV	28/28	—	—[l]	—[l]	.70[q]	.47	—	—	—	—	—	—
van Leeuwen et al (1998)	III-R	11/9	—	1.27[q]	1.45[q]	—	—	—	—	—	—	—	—
Weyandt and Willis (1994)	III-R	45/36	—	—	—	—[g]	—	.69[q]	—	—	—	—	—
Wiers et al (1998)	IV	34/28	—	—	—	—	—	—	.33	—	—	—	.88[q,SP]
Willcutt et al (2001)	III	121/100	.64[q]	.60[q]	—	.44[q]	.34[q]	—	—	—	—	.44[q,SS]	—
Willcutt et al (2005b)[m]	IV	177/151	.57[q]	.49[q]	.60[q]	.43[q]	.53[q]	—	—	—	—	.71[q,n]	.55[q,CA]
Williams et al (2000)	RS	10/20	—	—	—	—	—	—	—	—	—	—	.00[CA]
Wu et al (2002)	IV	29/83	—	—	—	—	—	—	.13	—	—	.53[DB]	—
Yeo et al (2003)	IV	24/23	—	—	.60[q]	—	—	—	—	—	—	—	—
Weighted mean effect size			.61	.51	.64	.46	.55	.69	.51	.58	.43	.55	.63
± 95% confidence interval[o]			±.09	±.08	±.09	±.09	±.11	±.26	±.18	±.19	±.12	±.11	±.16
Number of studies that found a significant group difference ($p < .05$)			22/27 (82%)	17/28 (61%)	23/30 (77%)	11/24 (46%)	8/14 (57%)	4/7 (57%)	3/6 (50%)	4/5 (80%)	5/9 (56%)	6/11 (55%)	6/8 (75%)

Table 2. (continued)

Study	ADHD Criteria[b]	Control/ ADHD (n)	Effect Size (d) of the Difference Between Groups[a]										
			Stop-Signal RT	CPT Comm. Errors	CPT Omiss. Errors	WCST Persev. Errors	Trails B Time	Tower of Hanoi	Tower of London	Porteus Mazes	ROCFT Copy Organiz.	Verbal Working Memory[c]	Spatial Working Memory[d]
Q_t Homogeneity Index[p]			34.2	32.4	39.4	31.3	22.2	4.7	12.2	4.2	2.4	31.0[q]	21.7[q]

CPT, Continuous Performance Test; WCST, Wisconsin Card Sorting Test; ROCFT, Rey-Osterreith Complex Figure Test. Significant difference between ADHD and comparison groups;—indicates measure not included in the study.

[a] Effect size, Cohen's d (Cohen, 1988): (ADHD M − Comparison M/pooled SD). All scores are scaled so that a positive effect size indicates greater impairment in the ADHD group regardless of the original scaling of the measure.

[b] Diagnostic criteria used to define the ADHD group. RS, normative cutoff score on a standardized parent or teacher rating scale.

[c] Superscripts next to effect sizes in this column distinguish between the verbal working memory tasks: DB, Wechsler Digits Backward; SS, Working Memory Sentence Span.

[d] Superscripts next to effect sizes in this column distinguish between the spatial working memory tasks: CA, CANTAB Spatial Working Memory; SP, Self-ordered pointing.

[e] Pooled effect size for groups with DSM-III attention-deficit disorder with and without hyperactivity.

[f] Pooled effect size for younger and older samples.

[g] These studies reported number of perseverative responses rather than perseverative errors (mean $d = .02$).

[h] A subset of these data were described by Carte et al (1996).

[i] A subset of these data were described by Nigg (1999).

[j] Only significant for the DSM-IV inattentive type.

[k] Mean effect size for 2-, 4-, and 8-sec interstimulus intervals.

[l] Administered an auditory CPT (mean $d = .15$ for commission errors, mean $d = .41$ for omission errors).

[m] A subset of these data were presented by Chhabildas et al (2001).

[n] Mean effect size for digits backward ($d = .81$) and sentence span ($d = .61$).

[o] Weighted mean effect size (d) and confidence interval (Cooper 1998) across all studies of each measure.

[p] The Q_t statistic (Hedges and Olin, 1985) is a measure of the probability that the variance in effect sizes across studies is attributable to sampling eror.

[q] Indicates significant heterogeneity of effect sizes for reasons other than sampling error.

difference on the WCST, and the mean effect size was among the lowest of all the tasks ($d = .46$). This pattern of results is similar to the findings of recent meta-analyses of Stroop interference control (mean $d = .35$; van Mourik et al, in press) and covert visuospatial attention (Huang-Pollock and Nigg 2003). These small effects and inconsistent results suggest that weaknesses in set-shifting, Stroop interference control, and visuospatial orienting of attention are poor candidates for a primary neuropsychologic deficit in ADHD.

Potential Moderators of the Relation Between EF and ADHD

The overall results of the meta-analysis indicate that ADHD is associated with weaknesses in several key EF domains; however, interpretation of these results is complicated by differences among the studies in sampling procedures and the diagnostic criteria used to define the groups, as well as the extent to which variables such as intelligence, reading ability, and symptoms of other disorders were controlled. In this section, we briefly examine whether any of these variables influence the relation between EF and ADHD.

Sampling Methods and Diagnostic Algorithms

The single population-based study included in the initial meta-analysis by Pennington and Ozonoff (1996) reported minimal evidence of an association between EF and ADHD (McGee et al 1989), leading the authors to suggest that additional research was needed to test whether the relation between ADHD and EF was significant in the general population or was a sampling artifact restricted to clinic-referred samples. Although nearly 90% of the studies in the review presented here recruited participants from clinics, eight studies used a community-based sampling procedure to identify children who met criteria for ADHD whether or not they had ever been referred for clinic services. The mean effect size on the EF measures in these eight community studies ($d = .49 \pm .06$) is only slightly smaller than the effect size in clinic-referred samples ($d = .56 \pm .04$), and the group difference was significant for 18 of the 28 comparisons in the community samples. These results clearly suggest that the relation between EF and ADHD is not restricted to clinic-referred samples.

Mean effect sizes were similar whether studies defined ADHD based on DSM-III ($d = .56 \pm .08$), DSM-III-R ($d = .57 \pm .06$), or DSM-IV ($d = .51 \pm .05$). Studies that used cutoff scores on standardized rating scales to define the groups yielded significant but smaller effects ($d = .41 \pm .16$). Thus, the conclusions described herein also appear to be robust across various diagnostic criteria for ADHD.

ADHD Subtypes

Several authors proposed that different patterns of EF weaknesses could provide key evidence of discriminant validity between the DSM-IV inattentive and combined subtypes, particularly on measures of response inhibition (e.g., Barkley 1997; Chhabildas et al 2001; Nigg 2001). The studies in the meta-analysis suggest that both subtypes differ significantly from comparison groups without ADHD on SSRT (inattentive type $d = .68 \pm .26$; combined type $d = .86 \pm .25$) and most other EF measures (d across measures $= .58 \pm .14$ for the inattentive type and $.57 \pm .12$ for the combined type), although neither group differed from control subjects on WCST perseverative errors in any study (inattentive type $d = .11 \pm .19$; combined type $d = .13 \pm .21$). In contrast, these studies found few consistent differences between the combined and inattentive types on any EF measure (mean d across measures = .09 ± .10; although see Nigg et al 2002 for a description of a possible gender × subtype interaction on the SSRT).

Only three studies included the DSM-IV hyperactive-impulsive type (Bedard et al 2003; Chhabildas et al 2001; Schmitz et al 2002). These preliminary results suggest that the hyperactive-impulsive type is associated with minimal executive impairment (mean d = .14). Although these results should be interpreted cautiously until larger samples are available, they suggest that the hyperactive–impulsive type may not share the same etiologic mechanism as the combined type. Furthermore, these initial data on the DSM-IV subtypes could indicate that EF weaknesses are primarily associated with DSM-IV inattention symptoms rather than hyperactivity–impulsivity symptoms. This finding is corroborated by two studies that included both symptom dimensions in a regression model predicting executive dysfunction; each study found that inattention was significantly associated with EF weaknesses, whereas hyperactivity–impulsivity was not independently associated with EF performance when the influence of inattention was controlled (Chhabildas et al 2001; Nigg et al, in press).

The Influence of Group Differences in Intelligence, Learning Difficulties, and Comorbid Disorders

Some researchers argue that intelligence, academic achievement, and symptoms of comorbid disorders should always be statistically controlled to ensure that neuropsychologic impairments associated with ADHD cannot be explained more parsimoniously by group differences on these correlated variables (Lahey et al 1998). On the other hand, ADHD symptoms and EF impairments or other neurocognitive weaknesses may directly cause poor performance on standardized tests of intelligence or academic achievement (Barkley 1997), in which case controlling for these variables may remove a portion of the variance that is shared between ADHD symptoms and EF weaknesses.

These issues have not been conclusively resolved, and the optimal approach may vary depending on the specific research question. Therefore, the effect sizes in Table 2 are derived from raw mean scores on each EF measure. We then examined whether the EF weakness remained significant in studies that controlled for these correlated variables (for a discussion of the related issue of specificity of EF weaknesses to ADHD versus other disorders, see Sergeant et al 2002). Several studies found that although the ADHD main effect was weakened, group differences on SSRT, CPT omission errors, planning tasks, and spatial and verbal working memory tasks remained significant after controlling for intelligence, reading achievement, and symptoms of comorbid disorders (Barkley et al 2001; Nigg 2001; Nigg et al 1998, 2002; Rucklidge and Tannock 2002; Willcutt et al 2001, 2005b), suggesting that the significant relation between EF and ADHD is not explained by group differences on these variables.

Comparison to the Previous Meta-Analysis

These results replicate and extend the findings of Pennington and Ozonoff (1996) in a much larger sample of studies. They found that groups with ADHD performed significantly worse than the comparison group in 40 of 60 comparisons (67%) on EF measures. Although their initial meta-analysis included only four of the 13 EF measures (10 studies included the WCST, 5 included the Trailmaking test, 3 included the Tower of Hanoi, and 1 study included the SSRT) and 10% of the total group comparisons (17 of 168) that are included in this review, the overall proportion of significant group differences is extremely similar (109/168; 65%).

The estimated effect size on the WCST was nearly identical in the two reviews (d = .46 vs. .45). In contrast, the weighted mean effect size for the Trailmaking test in this review (d = .55) is somewhat lower than the simple mean of the effect sizes from the six studies included in Pennington and Ozonoff (d = .75). When these six effects are weighted by the corresponding sample size, however, the mean effect (d = .62) is more similar to the estimate obtained in this review.

The lower effect size for the Tower of Hanoi in this review (d = 1.08 vs. .69) also appears to be explained in part by differences in the specific procedures of the meta-analysis. To control for the influence of comorbid reading disability in one study (Pennington et al 1993), Pennington and Ozonoff (1996) excluded the group with both reading disability and ADHD and reported the effect size for the group with ADHD alone (d = 1.24). In contrast, individuals with comorbidity were not excluded from our review. When the effect size for the study by Pennington et al (1993) is calculated based on all individuals with ADHD (d =.65), the weighted mean effect size for the three studies included in Pennington and Ozonoff (d = .81) is much closer to the result obtained in our review.

Summary and Conclusions from the Meta-Analysis

This meta-analysis suggests that ADHD is associated with weaknesses in several key EF domains. Effect sizes for all EF measures fell in the range that is typically considered a medium effect (Cohen 1988), but the strongest and most consistent effects were obtained on measures of response inhibition, vigilance, spatial working memory, and some measures of planning. Executive function weaknesses are present in both clinic-referred and community samples and are not fully explained by group differences in intelligence, academic achievement, or symptoms of other disorders.

Implications for the EF Theory and Comprehensive Neuropsychologic Models of ADHD

Although ADHD is consistently associated with weaknesses in key EF domains, the magnitude of the group difference on EF measures (d = .4–.6) is much smaller than the group difference in ADHD symptoms (d = 2.5–4.0 in the studies included in the meta-analysis). Moreover, fewer than half of children with ADHD exhibit significant impairment on any specific EF task (Nigg et al 2005, this issue), and correlations between ADHD symptoms and scores on EF tasks are typically significant but small in magnitude (r = .15–.35; Nigg et al 1998; Willcutt et al 2001). Family and twin studies suggest that the small to medium correlations between EF and ADHD are primarily attributable to common genetic influences (Chhabildas et al, unpublished data; Doyle et al 2005, this issue), but these studies also indicate that the majority of the genetic and environmental influences on ADHD symptoms are independent from the influences that lead to weaknesses in executive control.

Taken together, these results clearly indicate that EF weaknesses are significantly associated with ADHD, but they do not support the hypothesis that EF deficits are the single necessary and sufficient cause of ADHD in all individuals with the disorder. Instead, EF difficulties appear to be one of several important weaknesses that comprise the overall neuropsychologic etiology of ADHD (other neurocognitive correlates of ADHD are described elsewhere in this issue by Nigg et al 2005, Pennington 2005, Sagvolden et al 2005, Sergeant 2005, and Sonuga-Barke

2005). The failure to find a single primary neuropsychologic cause is almost certainly not unique to ADHD. Indeed, the neuropsychology of most developmental psychopathologies is likely to be complex and multifactorial (Sergeant 2003), suggesting that a single necessary and sufficient cause is improbable for most disorders.

The transition from models positing a single core deficit to multiple-deficit models represents a paradigm shift in the way that the neuropsychology of ADHD is conceptualized (Sonuga-Barke 2003). As part of this shift theoretical models are beginning to emerge that acknowledge and attempt to account for the neuropsychologic heterogeneity of ADHD (e.g., Castellanos and Tannock 2002; Nigg et al 2005, this issue; Sergeant 2005, this issue; Sonuga-Barke 2005, this issue). For example, independent pathway models posit that ADHD is a heterogeneous disorder with discrete neuropsychologic subtypes that arise from dysfunction in distinct pathophysiologic substrates (e.g., Nigg et al 2004a, 2004b; Sonuga-Barke 2005, this issue). In contrast, other multiple deficit models suggest that ADHD is attributable to the additive or interactive effects of dysfunction in multiple neural networks in the same individual (e.g., Sergeant et al 2003; Willcutt et al 2005a).

Additional studies are needed to test these and other competing neuropsychologic models of ADHD. In the final section, we describe three directions for future research that may clarify the relation between EF and ADHD and facilitate the development of a comprehensive neuropsychologic model of ADHD.

Directions for Future Research

Evaluate the Psychometric Characteristics of EF Tasks

Relatively few studies have examined the reliability of EF tasks in children. Although some EF measures appear to have satisfactory psychometric characteristics (Delis et al 2001; Kuntsi et al 2001b), reliability estimates are only moderate (or unknown) for many of the EF measures included in the meta-analysis. Because the reliability of a measure constrains the amount of variance that the measure can explain, measures with weak psychometric characteristics will never yield large effect sizes. Future studies of EF and other neuropsychologic correlates of ADHD should use tasks with existing reliability data or carefully assess the reliability of each task. Consideration should also be given to employing statistical techniques such as latent trait analysis to minimize the impact of the psychometric weaknesses of each individual task by obtaining a measure of the common variance among multiple measures of a specific EF domain.

Approaches to Clarify the Structure of Executive Functions

Interpretation of the meta-analysis is complicated by the fact that many executive tasks involve multiple neurocognitive processes. Thus, it is difficult to be certain that failure on a planning, working memory, or response inhibition task is really due to a weakness in the construct for which the task is named. To clarify the interpretation of different EF tasks, future studies should attempt to develop tasks that are better able to isolate specific parameters of interest. This may be accomplished through careful task analysis or by developing appropriate within-task and between-task controls.

Directly Test Competing Neuropsychologic Theories

As noted previously, most studies have examined the neuropsychologic correlates of ADHD from a single theoretical perspective (e.g., EF, delay aversion, motivational dysfunction). Future studies that pit competing theories directly against one another are needed . This could be accomplished by administering measures of each theory in the same sample of individuals (Solanto et al 2001) or by adding a control task to a key measure of a theory to test whether the ADHD group deficit is better explained by a weakness in another neurocognitive function. It will be essential for studies of competing models to be designed so that each theory makes strong a priori predictions about the outcome of the experiment. These studies will clarify the relations among different EF constructs and other neurocognitive processes, as well as the relative importance of each neurocognitive weakness as part of the overall pathophysiology of ADHD.

Conclusion

Executive dysfunction in domains such as response inhibition, planning, vigilance, and working memory plays an important role in the complex neuropsychology of ADHD. Nonetheless, EF weaknesses are neither necessary nor sufficient to cause all cases of ADHD. Additional research is needed to assess the impact of diagnostic and neuropsychologic heterogeneity and to clarify the relations between various EF dimensions, as well as the relations between EF and other neurocognitive and emotion–motivation domains.

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