Why Children with ADHD Do Not Have Low IQs

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Abstract

The major cognitive deficit of children with attention-deficit/hyperactivity disorder (ADHD) is impaired executive function (EF), a cognitive component that some theorists believe to be the primary substrate for the general intelligence (*g*) factor. We review the constructs of *g* and EF and the relevant research findings on ADHD. We then analyze the results of a battery of diverse tests, including measures of EF, administered to 123 boys with ADHD. The correlations among the EF measures, two well-accepted measures of IQ, and the *g* factor extracted from the entire battery are trivial at best. These results are discussed in the context of collateral evidence supporting the independence of *g* and EF and its clinical and theoretical implications.

We wish to apologize for the poetic license taken in the title of this article. A more correct statement of the problem would be, "Why distributions of IQ scores for children with attention-deficit/hyperactivity disorder (ADHD) are not appreciably different from IQ distributions observed in the population at large." The belief that children with ADHD do not necessarily have low IQs can be traced back to a nodal article by Clements (1964) and has rarely since been called into question. In this article, we consider three premises that would seemingly dictate an alternative conclusion:

1. Child psychopathologists view ADHD as a disorder of executive function (EF; e.g., Barkley, 1995, 1997).
2. Cognitive theorists hold that individual differences in general intelligence (*g*) can be accounted for by individual differences in EF (e.g., Sternberg, 1985; Sternberg & Gardner, 1982).
3. Psychometric theorists believe that IQ is a reasonably good approximation of *g* (e.g., Carroll, 1988; Jensen, 1998).

Accepting the validity of these premises (the investigators cited arguably represent the mainstream in their respective areas of concentration), the inescapable conclusion would be that children with ADHD should tend to have lower IQs than children in the general population. The purpose of this article is to briefly consider the empirical evidence that fails to support this seemingly reasonable expectation and to discuss these findings in the context of clinical and experimental evidence that may explain the phenomenon.

Empirical Evidence

Do children with ADHD have lower IQ scores than children without ADHD? A few studies (e.g., Barkley, 1990; Doyle, Biederman, Seidman, Weber, & Faroane, 2000; Faroane et al., 1993; Goldstein, 1987; Werry, Reeves, & Elkind, 1987) might seem to offer limited support for this conclusion, but there have been equally many reports of independence between IQ and the ADHD diagnosis (e.g., Douglas, 1972; Prifitera & Dersh, 1993; Reader, Harris, Schuerholz, & Denckla, 1994; Schwean, Saklofske, Yackulic, & Quinn, 1993; Wechsler, 1991).

Most recently, the MTA Cooperative Group (1999) reported findings from an ongoing National Institute of Mental Health (NIMH)–funded multisite study of ADHD (MTA study) in which the mean Full Scale IQ (FSIQ) derived from the *Wechsler Intelligence Scale for Children–Third Edition* (WISC-III), administered to 579 well-characterized children with ADHD was 100.9, with a standard deviation of 14.8. When corrected for the fact that children with IQ < 80 were screened from the study (presumably about 8% based on the normal distribution), the mean FSIQ estimate would still be 98.45, arguably not different from the WISC-III test standardization population mean of 100. Because this is the largest and most carefully screened population of children with ADHD yet studied, these results would support the conclusion that the FSIQ levels of children with ADHD, as a group, do not differ appreciably from those of the general population.

Doyle et al. (2000), reporting test results of 113 well-characterized boys with ADHD, found a mean FSIQ of 107.1 (SD = 16.7). This finding was

based on evaluation of a cohort of children with FSIQ ≥ 80 from which children with very low socioeconomic status (SES) were excluded (to reduce etiologic heterogeneity). In view of the constrained IQ distribution of this sample, the obtained mean FSIQ score of 107 might very well, after adjustment, be close to the mean of the general population.

IQ Subtest Profiles

Among investigators who have found IQ to be depressed in children with ADHD, most will concede that the lower overall score is usually the result of low performance on just a few of the measures used to estimate IQ—a point originally advanced by Clements and Peters (1962). Depressed ACID (Arithmetic, Coding, Information, and Digit Span) or ACIDS (Arithmetic, Coding, Information, Digit Span, and Symbol Search) profiles are most commonly ascribed to children with ADHD (Dykman, Ackerman, & Oglesby, 1980; Loge, Staton, & Beatty, 1990; Prifitera & Dersh, 1993). Other profile-based schemes involve the use of group subtest relationships, such as contrasting the WISC-III Freedom from Distractibility Index (FDI) or Processing Speed Index (PSI) with the Performance IQ (Naglieri, Das, & Jarman, 1990; Schwean et al., 1993).

There appears to be a consensus that children with ADHD are less successful on the ACID(S) subtests and that the relative performance decrement on these subtests may reflect EF limitations. However, the correlations of some of the ACID(S) subtests (e.g., Digit Span, Coding) with FSIQ are among the lowest found for WISC-III subtests (Jensen, 1980; Schwean et al., 1993; Wechsler, 1991). In fact, Wechsler (1958), in describing the rationale for including Digit Span among the WISC subtests, noted, "The ability involved contains little of *g* [i.e., general intelligence] and, as Spearman has shown, is more or less independent of this general factor" (pp. 70–71). Digit Span was included among the subtests primarily for "diagnostic significance" (Wechsler, 1958, p. 71), and is now considered supplementary (i.e., does not enter into the calculation of IQ unless another verbal subtest has been invalidated). Arithmetic, though having moderately high correlations with *g*, is nevertheless more adversely influenced by noncognitive factors than other subtests, as is Coding (Siegman, 1956).

Finally, in a recent large study in which test batteries containing both high *g*-loaded and EF tests were used to differentiate the performance of children with ADHD from controls, the diagnostic efficiency of the test battery was not appreciably enhanced by correction for FSIQ (Doyle et al., 2000). Before presenting additional empirical evidence, we briefly consider the constructs of general intelligence and executive function.

General Intelligence

People who are proficient at solving one problem tend to be proficient at solving others; those less capable of solving one problem tend to be less capable of solving others. More than a century ago, Francis Galton conducted the first empirical demonstrations of this phenomenon. Using a correlation coefficient, Galton's successor, Karl Pearson, was able to mathematically characterize the degree of correspondence between performance on one test and performance on another. Pearson further demonstrated that whenever a battery of mental tests was administered to a representative sample of participants, the correlations among all the tests would be positive. Pearson's successor, Charles Spearman, interpreted this phenomenon as indicating an underlying trait, best evidenced by the fact that a robust first principal component, positively correlated with all tests, emerged whenever a battery of diverse mental tests was factor analyzed. He called this trait *general intelligence,* or *g* (Spearman, 1904, 1927; see Jensen, 1998, for a comprehensive review).

For Spearman (1927), the degree to which a particular test is "*g*-loaded" could be estimated from the correlation of that test with the first principal component. The success with which one solved any mental test depended on *g* plus other abilities that might be required for solving that particular test—Spearman's well-known *two-factor theory*. The degree to which a test was uncorrelated with *g*, the *residual variance,* reflected the presence of specific, non-*g* abilities, as depicted in Figure 1 (Jensen & Weng, 1994). Later, Spearman and others (e.g., Thurstone, 1938) came to identify "group" factors, which appeared to reflect commonality among certain groups of tests (e.g., verbal tests, spatial reasoning tests). Identification of the intermediate strata between individual test items and *g* became the basis for the *hierarchical model* subscribed to by most modern psychometric theorists. In such a model, the most basic level consists of performance on specific tests, with each succeeding level of the hierarchy reflecting tests or factors composed of measures that are more highly correlated among themselves than they are with other measures or other factors. At the apex of this hierarchy is *g*, which might be a third-, fourth-, or fifth-order factor, depending on the uniqueness of the test items sampled at the basic level (see Figure 2; Jensen & Weng, 1991).

For almost a century, there have been no serious challenges to Spearman's view that *g* represents the major source of individual differences in mental test performance, although there has been extensive discourse as to the underlying cause of the phenomenon (e.g., Detterman, 1987; Guilford, 1967; Thomson, 1951; Thurstone, 1938). For purposes of the current discussion, it is important to note that Sternberg (e.g., 1985) and other cognitive theorists have asserted that *g,* at the apex of this hierarchy of cognitive abilities, reflects

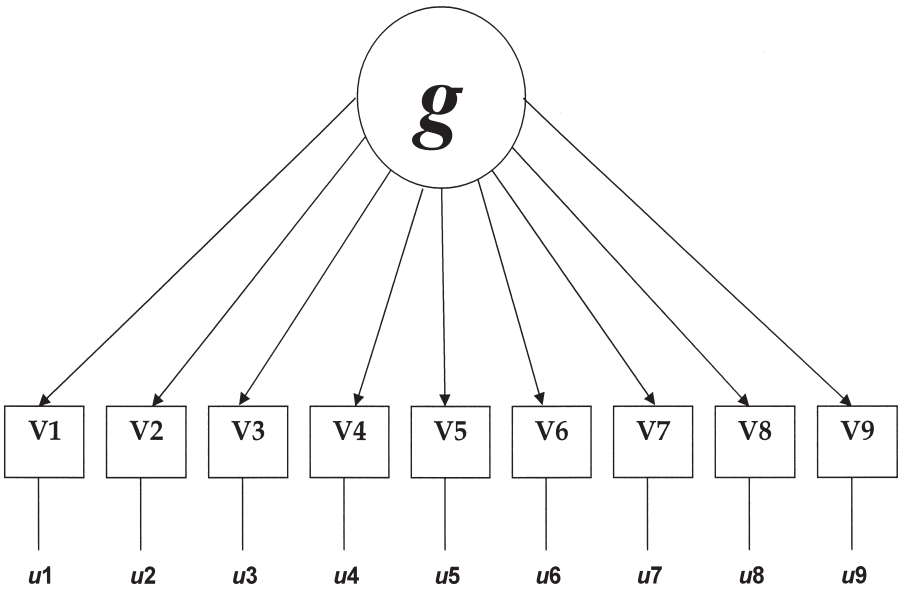

FIGURE 1. Schematic representation of Spearman's two-factor theory of cognitive abilities. Every test measures a general factor (*g*), common to all of the variables (V), and the factors specific to each variable, termed specificity (*s*). Each variable's uniqueness (*u*) comprises *s* and measurement error. (Reprinted from *Intelligence,* Vol. 18, A. Jensen and L.-J. Weng, What is a good *g*?, pp. 231–258, Copyright 1994 by Elsevier. Reprinted with permission from Elsevier.)

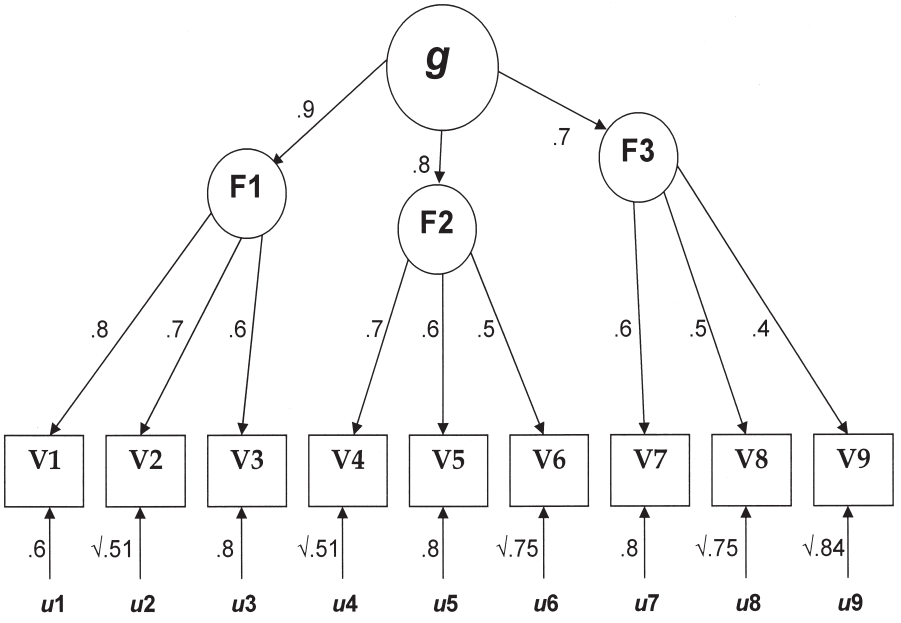

FIGURE 2. The hierarchical relationship among factors that emerges from a typical factor analysis of a number of diverse mental tests. The first-order factors (F) are correlated, giving rise to a second-order factor (*g*). Variables (V) are correlated with *g* only via their correlation with the three first-order factors. (Reprinted from *Intelligence,* Vol. 18, A. Jensen and L.-J. Weng, What is a good *g*?, pp. 231–258, Copyright 1994 by Elsevier. Reprinted with permission from Elsevier.)

the fact that all mental test performance relies heavily on EF.

Executive Function

With the advent of artificial intelligence and cognitive science (e.g., Ashby, 1952; Newell & Simon, 1972), the debate on the nature of psychometric *g* has been eclipsed by investigations of *components*, or computational subroutines that operate during mental activity, such as problem solving. Within this framework, *g* has been said to represent the operation of a particular set of components (variously termed "executive processes," "executive routines," "control processes," "metacomponents," or "executive functions") that exert superordinate control over the brain's computational programs (Sternberg, 1985). In this article, we use the term *executive function* (EF), in the singular, to denote this set of processes.

It is beyond the scope of this article to review the many hypothesized EF components. In the interest of brevity, we assume that most of these metacomponents will be found among Sternberg's (1984) list of EF processes: (a) decision as to just what the problem is that needs to be solved, (b) selection of lower order components, (c) selection of one or more representations or organizations of information, (d) selection of a strategy for combining lower order components, (e) decision regarding tradeoffs in the speed and accuracies with which various components are executed, and (f) solution monitoring. Other lists of EF components abound (e.g., Fletcher, 1996; Welsh & Pennington, 1988). Perhaps the most illuminating and succinct characterization of EF is that of Barkley (2000), who stated that EF involves "the 'where' or 'whether' aspects of behavior, while non-executive functions involve the 'what' and 'how' " (p. 1065).

Sternberg and Gardner (1982) reasoned that as EF is "common to all of the tasks in a given task universe" (p. 232), the *g* extracted by factor analysis largely represents individual differences in EF. Those differences are "in

large part responsible for the appearance of a general factor in mental ability tests" (Sternberg, 1985, p. 13). Any act of problem solving requires EF, irrespective of the number, type, or computational power of the discrete subroutines executed within the brain's neural networks. As EF is "highly overlapping across tasks of a widely differing nature" (Sternberg, 1985, p. 119), much like Spearman's conception of *g*, there seemed to be ample rationale for concluding that psychometric *g* is a manifestation of the ubiquity of EF. Not only is the apparent logic of this theory seductive, but empirical findings from studies conducted by Sternberg and colleagues (e.g., Sternberg & Gardner, 1983) can be construed as support for this proposition.

EF and Attention Theory

Posner has written extensively on the role of EF in the larger context of attention theory (e.g., Posner & Raichle, 1994; Posner, Peterson, Fox, & Raichle, 1988). For Posner, attention operates by changing the relative activity in anatomical areas that perform computations necessary for adaptation. Based on human brain imaging studies, anatomical networks subserving three subcomponents of attention have been described: (a) arousal, (b) orienting, and (c) executive. The executive network is said to coordinate multiple specialized neural processes that direct behavior toward a goal—a view not inconsistent with Sternberg's (1984), Barkley's (1997), or Welsh and Pennington's (1988) view of EF.

ADHD as an EF Disorder

Because the executive network does not mediate the totality of identified attention processes, children diagnosed with ADHD may have attention deficits that are not exclusively EF related. For example, some theorists have emphasized the similarity of ADHD to "neglect" syndromes—in Posner's scheme, a defect of the orienting rather than the executive network (Voeller, 1986; Voeller & Heilman, 1988). However, a preponderance of child psychopathologists now consider an EF deficit to be the core feature of ADHD (e.g., Barkley, 1997; Denckla, 1995; Douglas, 1988; Seidman, Biederman, Faroane, Weber, & Oulelette, 1997; Sergeant & van der Meere, 1988). EF is absolutely necessary for goal attainment, because it is composed of cognitive subcomponents such as prolongation of action, separation of affect, guidance by inner language, and analysis/synthesis of action (Barkley, 1997). The net result of the integrated activity of these EF subcomponents is the resolution of conflicts among the multiple information-processing networks that are continuously competing for expression by selecting, sequencing, and monitoring their actions (Swanson et al., 1997).

Neurobiology of ADHD

Anatomical Evidence. Posner has shown that the anatomical network that apparently subserves EF is located in the anterior cingulate gyri, left lateral frontal lobe, and basal ganglia (Posner & Raichle, 1994). It is not surprising, then, that recent brain imaging studies of children with ADHD have identified dysmorphologies in these and anatomically contiguous neural structures. These findings, summarized in Figure 3, are of interest because most investigators have reported anatomical differences between children with ADHD and controls that involve the nigrostriatal or frontostriatal networks and closely interconnected structures (e.g., Denckla & Reiss, 1997) —what we will term hereafter the *EF system.*

The neural structures that underlie the EF system are located in the brain's dopaminergic pathways, in particular the mesolimbic and nigrostriatal dopaminergic systems. The mesolimbic system, composed of the A10 dopaminergic neurons emanating from the ventral tegmental area (adjacent to the zona compacta of the sub-

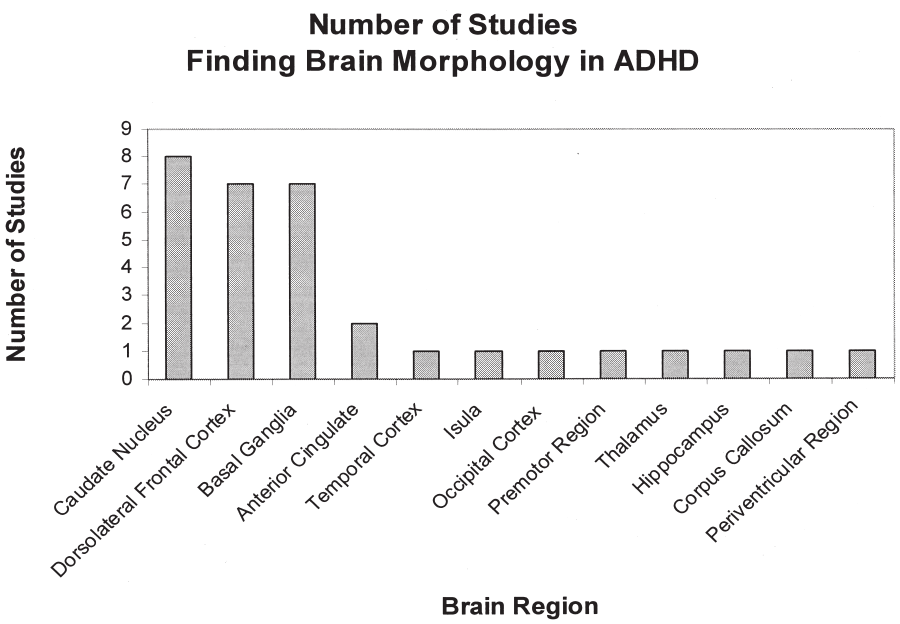

FIGURE 3. Summary of imaging findings showing atypical brain morphology in ADHD (based on Aylward et al., 1996; Casey, Giedd, Vauss, Vaituzis, & Rapoport, 1992; Casey et al., 1997; Castellanos et al., 1994; Castellanos et al., 1996; Filipek et al., 1997; G. S. Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990; G. W. Hynd et al., 1991; G. W. Hynd et al., 1993; Lou, Henricksen, & Bruhn, 1990; Lou, Henriksen, Bruhn, Borner, & Nielsen, 1989).

stantia nigra) and projecting to the nucleus accumbens and olfactory tubercle, has reciprocal connections with the anterior caudate and prefrontal cortex (Middleton & Strick, 1994; Ungerstedt, 1971). The role of this set of structures within the EF system may be to channel interoceptive information that underlies decisions and the formation of sets (Graybiel, Aosaki, Flaherty, & Kimura, 1994; Iversen, 1984).

The nigrostriatal dopaminergic system emanates from the A8 and A9 dopaminergic neurons in the substantia nigra. These neurons, containing about 75% of all brain dopamine, terminate in the caudate nucleus and putamen, with rich interconnections to frontal and cingulate cortices. The complexity of the nigrostriatal dopaminergic system, including its lateralization, has been discussed at length by Iversen (1977, 1984). A set of investigations conducted by Schultz and associates (e.g., Schultz, 1998) have convincingly shown that this system is involved in processing reward information by labeling environmental stimuli with appetitive value, predicting and detecting rewards, and signaling alerting and motivating events. Obviously, a perturbation of a system that mediates these functions could result in the disorders of goal-directed behavior that are commonly found among children with ADHD.

Pharmacological Evidence. ADHD has been successfully treated for more than 60 years with dopamine (DA) agonists (Bradley, 1937; Wender, 1971; see Swanson et al., 1993, for a "review of reviews"), and the main site of action of DA agonists is the EF system (Levy, 1991; Malone, Kershner, & Swanson, 1994; McCracken, 1991; Rapoport & Zametkin, 1989; Swanson, Sergeant, et al., 1998). Recently, the degree of EF system dysmorphology found among children with their ADHD has been correlated with response to stimulant medication (Filipek et al., 1997).

Treatment with stimulant medication is usually found to have a positive benefit for children with ADHD (MTA Cooperative Group, 1999), particularly in improving EF (Barnett et al., 2001; Douglas, Barr, Amin, O'Neill, & Britton, 1988; Whalen & Henker, 1991). However, Doyle et al. (2000) contrasted the performance of 71 medicated and 51 nonmedicated boys with ADHD and failed to demonstrate that psychopharmacological treatment was associated with better performance on a neuropsychological test battery that included several EF measures. A plausible explanation for this finding is that the participants were not randomly assigned to the medication condition (i.e., the medicated group may have consisted of children with greater initial EF deficits, biasing the decision to medicate).

On the other hand, several studies have shown that IQ is not affected by stimulant treatment. Schwean et al. (1993) concluded that IQ may be too coarse a measure to reflect the effects of stimulants, but that tests that assess quite specific cognitive processes (e.g., planning, attention, simultaneous and sequential processing; in other words, EF) are indeed sensitive to stimulants. Indeed, if IQ were only modestly EF dependent, it would be unlikely that drugs specific for EF dysfunction would have a sufficiently broad influence on the multiple neural networks that are invoked when *g*-loaded problems are being solved (Anderson, 2001; Crinella & Yu, 1995).

Genetic Evidence. The familial basis of ADHD has been convincingly demonstrated over the past 3 decades by adoption, family, and twin studies, with heritability estimates ranging as high as .9 (e.g., Levy, Hay, McStephan, Wood, & Waldman, 1997). Because of the well-established effectiveness of DA agonists in treating the disorder, molecular genetic studies have focused on DA transporter and DA receptor genes (LaHoste et al. 1996; Swanson et al., 2000; Swanson, Sunohara, et al., 1998). Swanson et al. have shown that a particular polymorphism in the DA receptor D4 (D4R4) gene, located on chromosome 11p15.5, is associated with blunted sensitivity of the D4 receptor, which is prominently found in brain regions that play a role in attention (e.g., anterior cingulate gyrus). This finding has since been replicated (e.g., Rowe et al., 1998; Smalley et al., 1998). There is an equally impressive body of work on genes that control other facets of the DA system, including the DRD2 receptor gene (Comings et al., 1991); the DA transporter gene, DAT1 (Gill, Daly, Heron, Hawi, & Fitzgerald, 1997); and the DA β-hydroxylase gene, DBH (Kuperman, Kramer, & Loney, 1988), not to mention genes related to other neurotransmitter systems (see, Comings, 2001, for a recent review). Hence, children will manifest symptoms of ADHD because of an accumulation of polygenic influences that evoke behaviors that are sometimes sufficient to meet diagnostic criteria for ADHD, the most noteworthy being alleles affecting dopaminergic systems.

Neuropsychology of ADHD

Long before data from imaging and genetic studies were available, frontal lobe dysfunction was presumed to be the neural substrate for ADHD. Indeed, the many imaging studies associating structural or functional differences in the frontal lobes and closely interrelated structures with ADHD are consistent with the finding that many of the neurocognitive deficits found in frontal lobe patients (see, e.g., Fuster, 1989, 2002; Luria, 1966; Milner, 1982, 1995; Pribram, 1971; Shallice, 1982) have also been reported in ADHD (see, e.g., Barkley, 1994; Benton, 1991; Chelune, Ferguson, Koob, & Dickey, 1986; Douglas, 1980, 1983; Douglas & Peters, 1979; Elliott, 2003; Evans, Gualtieri, & Hicks, 1986; Mattes, 1980; McBurnett, et al., 1993; Sneidman, Biederman, Faraone, Weber, & Oullette, 1997; Van der Meere & Sergeant, 1988). Preferences differ as to the kinds of clinical or laboratory tests that are used to specifically target frontal lobe deficits (Fuster, 1989), and it is clear that children with

ADHD may not exhibit deficits on each and every one of these "frontal lobe" tests (Barkley, Grodzinsky, & DuPaul, 1992; Loge et al., 1990; Pennington, Bennetto, McAleer, & Roberts, 1996). In this regard, it is well to consider Fletcher's (1996) admonition that "it is wrong to ascribe cognitive functions subserved under 'executive' functions to the frontal lobes, particularly *at the level of specific tasks*" (p. 1; emphasis added). Nevertheless, the "frontal lobe syndrome," as broadly conceived, would encompass most of the EF deficits that typify ADHD.

In a recent theoretical formulation of ADHD, Barkley (1997) integrated much of the evidence on the relationship of frontal lobe dysfunction with ADHD into a comprehensive view that emphasizes the role of behavioral inhibition (i.e., inhibition of prepotent responses, stopping prepotent responses, and controlling interference) required for four components of EF: (a) working memory; (b) self-regulation of affect, motivation, and arousal; (c) internalization of speech; and (d) analysis and synthesis of behavior. In Barkley's conceptualization, virtually all of the classical ADHD symptoms (e.g., impulsivity, distractibility, perseveration, diminished persistence, diminished sensitivity to feedback, lack of planning and judgment, and poor modulation of affect) can be accounted for by disinhibition.

Barkley's (e.g., 1997) and Sternberg's (e.g., 1985) arguments converge with those of Fuster (e.g., 1981, 1989), who has summarized the role of the frontal lobes as that of "prevailing" on other brain systems "to ensure the orderly execution of actions" (1989, p. 167). Much as Sternberg (1985) spoke of the nonspecific role of EF in information processing, Fuster emphasized the finding that the frontal lobes are not essential for the execution of any particular action, but for the orderly and purposive execution of all complex behaviors—what Lashley (1951) described as the "syntax of action" (p. 122). The syntax of action unfolds through the formation of cross-temporal connections, necessary for preserving the continuity of purpose over time. In much the same vein, Barkley (1997) believed that inhibition—the ability to stop or delay responding for periods of time—enables cross-temporal connections to become established, perhaps contributing to the psychological sense of time. In summary, there are remarkable similarities between the behavioral deficits of children with ADHD and those deficits found in frontal lobe syndromes. Such deficits could be explained by invoking the overarching construct of disinhibition, which has been shown to compromise most EF components.

Dissociation of IQ and EF in ADHD

The evidence discussed in the preceding section demonstrates that there is a set of brain structures or neural networks that could justifiably be labeled the EF system and that this system is atypical in structure or in function among children with ADHD. There remains the question whether *g* and EF are simultaneously affected by perturbations of this system.

The independence of *g* and EF was considered in a nodal study by Welsh, Pennington, and Grossier (1991), in which a battery of EF tests was administered to children without ADHD, with subsequent analysis yielding three factors: (a) response speed, (b) set maintenance, and (c) planning—arguably EF factors. These EF factors were either inversely correlated or not correlated with IQ—a complete dissociation of *g* and EF. In a later study, Pennington, Grossier, and Welsh (1993) found moderate correlations between FSIQ and measures of EF (e.g., perseverative errors on the *Wisconsin Card Sorting Test*, $r = -.38$; percentage correct on the *Continuous Performance Test*, $r = .38$). However, it should be noted that these coefficients were derived from a combined clinical–control population, yielding a somewhat platykurtic distribution of participants with respect to EF functioning, favoring higher intercorrelations. Reader et al. (1994), in a study of children with ADHD with above-average IQ (*Mdn* = 117), found significant deficits (in relation to normative data) on two measures of EF, the *Wisconsin Card Sorting Test* and the *Continuous Performance Test*—a finding that suggests that significant EF deficits are not necessarily accompanied by lower overall intelligence. Unfortunately, whereas intercorrelations among the EF measures were measured, IQ was not included. In a study of nonhandicapped schoolchildren, Hutton, Wilding, and Hudson (1997) found that a number of measures from the *Tests of Everyday Attention* (Robertson, Ward, Ridgeway, & Nimmo-Smith, 1994), which included a test of attention switching that correlates with perseverative errors on the *Wisconsin Card Sorting Test,* did not have significant correlations with IQ.

Recently, Crinella and Yu (2000), extending the work of Pennington et al. (1993), used a sample consisting of children both with and without ADHD. When the test battery was factor analyzed, the first principal component met the criteria for psychometric *g*, whereas the second component appeared to be an EF factor. The correlation between the *g* and EF factors was a modest +.24. Hence, EF accounted for a statistically significant but rather modest amount of the variance in *g*, a finding consistent with that of Pennington et al. (1993). These studies help to explain the fact that the IQs of children with ADHD are relatively unaffected by their EF disorder. The following study was undertaken to replicate and further extend the aforementioned findings.

Method

Participants

Participants were recruited from an initial pool of 150 consecutive male patients presenting for neuropsychological evaluation at the University of California, Irvine, Child Development

Center (CDC), a university-based clinic specializing in the diagnosis and treatment of children with ADHD. Children who met the criteria for ADHD of the *Diagnostic and Statistical Manual of Mental Disorders–Fourth Edition* (*DSM-IV*; American Psychiatric Association, 1994), were offered enrollment in the study, and legal informed consent for use of their test data was obtained. Children with FSIQs < 80 were excluded based on a rationale similar to that of Doyle et al. (2000). Also excluded were those children who were unable or unwilling to discontinue medication for at least 24 hours before the evaluations took place. The residual cohort consisted of 123 boys, ages 7 through 13 (M = 9.86, SD = 2.10). The CDC is located in the relatively affluent Newport Beach–Irvine area of Orange County, California, and the majority of participants were middle class and above.

Procedure

All participants completed the *Wechsler Intelligence Scale for Children–Third Edition* (WISC-III; Wechsler, 1991), *Raven Progressive Matrice*s (RPM; Raven, Court, & Raven, 1988), *Continuous Performance Test* (CPT; Conners, 1992), and *Wisconsin Card Sorting Test* (WCST; Heaton, 1981). The tests were administered in two sessions, each beginning at 10:00 a.m. The RPM and WISC-III were administered, in that order, on the first day of testing, and the CPT and WCST were administered, in that order, on the second day of testing (after which additional tests were usually given).

The RPM was included because it is a test that has been invariably found to have *g* loadings in the .80 vicinity and because it is now favored in investigations of "fluid intelligence" (e.g., Duncan, Burgess, & Emslie, 1995; Duncan, Emslie, Williams, Johnson, & Freer, 1996). The CPT and WCST are commonly used to investigate EF components (e.g., Pennington et al., 1993; Reader et al., 1994). For purposes of the current analysis, the CPT was used specifically to probe the capacity to control impulsive errors (IMPULS), and the WCST was used specifically to assess two other aspects of EF, the ability to shift cognitive sets (via inhibition of perseverative tendencies; PERSEV) and the ability to maintain set (FAILSET).

Both the the CPT and the WCST were computer administered. For the CPT, the child was seated before the computer display screen and instructed to press the space bar as quickly as possible when any letter, except *X*, flashed on the screen. When the target letter *X* appeared (twice, randomly, within each set of 20 trials), the response was to be withheld. For all letters, the display time was 250 ms, with each appearance of a letter separated by one of three interstimulus intervals (ISIs): 1, 2, or 4 seconds. A total of 360 letter presentations occurred, divided into six blocks of 60. Within each block, 20 consecutive trials were given for each of the three ISIs, with the within-block sequence of ISIs counterbalanced. The task required a total of 15 minutes. The dependent variable of interest was *impulsive errors* (IMPULS; i.e., key presses when the nontarget *X* appeared).

For the WCST, the participant was again seated before the computer display screen and instructed to match a series of 128 single-stimulus cards to one of four sample cards that remained stationary on the screen. The sample cards could be categorized by color (red, green, blue, or yellow), form (crosses, circles, squares, or triangles), and numbers (one, two, three, or four). The participants were instructed to match each stimulus card with one of the four sample cards. The participant was not informed of the relevant category, only that the computer would signal "right" (a bell) or "wrong" (a buzzer) after each response. The test proceeded through unannounced shifts among three possible sorting categories (color, form, and number), with each shift occurring only after 10 of 10 consecutive correct matching responses. The dependent variables of interest were *perseverative errors* (PERSEV; i.e., matching a card using the previously correct but now incorrect category) and *failure to maintain set* (FAILSET; i.e., failure to persist with the criterion of 10 of 10 consecutive correct responses after 4 consecutive correct responses had been achieved).

Results

Table 1 shows means and standard deviations for all measures, including composite scores, as well as their correlations with age. As would be expected for standard scores already age-normed, none of the cognitive measures was significantly age-correlated. Raw scores on the three EF measures (IMPULS, PERSEV, FAILSET) were also clearly unrelated to age in this sample.

FSIQ

As indicated in Table 1, the mean FSIQ on the WISC-III for this cohort was 105.62, somewhat higher than the mean for the WISC-III test standardization population. The obtained mean IQ was also higher than the mean IQ of 98.45 reported in the MTA study. The discrepancy between the MTA and the current population was not an unanticipated finding, in view of the fact that the MTA study was more representative of all SES strata, whereas this study was constrained to higher SES. The constricted range of scores is further exemplified by the somewhat lower *SD* (14.43) for this sample, in contrast to the test standardization population *SD* of 15. With respect to our measure of "fluid" intelligence, the RPM, the mean IQ of 106.40 was not significantly different from the WISC-III FSIQ.

FSIQ and EF

Table 2 is an intercorrelation matrix for all variables, including (for informational purposes only) the correlations of the (non-independent) composite scores—Verbal IQ (VIQ), Performance

IQ (PIQ), FSIQ, FDI, and ACID—among themselves, and other independent measures. Most salient for the question posed in the preceding section, the correlations between FSIQ and the three EF measures were as follows: IMPULS, $r = .07$; PERSEV, $r = -.22$; and FAILSET, $r = .19$. For IMPULS, there was clearly no relationship with FSIQ. For FAILSET, the relationship was statistically significant ($p = .025$) but in the unanticipated direction. For PERSEV, the correlation with FSIQ was in the anticipated direction and statistically significant ($p = .01$). However, a correlation of this magnitude accounts for less than 5% of the variance.

TABLE 1
Means, Standard Deviations, and Age Correlations for All Measures

Variable	*M*	*SD*	r_{age}
Age	9.86	2.09	
Information	11.16	2.34	–.008
Similarities	11.90	2.78	–.043
Arithmetic	10.28	2.62	–.051
Vocabulary	11.83	2.39	–.179*
Comprehension	12.41	3.09	.071
Digit Span	10.26	1.98	.010
Picture Completion	11.05	2.04	–.085
Coding	9.17	2.93	–.050
Picture Arrangement	10.97	2.54	–.225**
Block Design	11.23	2.51	–.035
Object Assembly	10.10	2.88	.040
RPM IQ	106.88	11.79	–.242**
CPT Errors (IMPULS)	20.04	9.63	–.066
WCST Perseverations (PERSEV)	23.68	13.73	–.05
WCST Set Failures (FAILSET)	2.12	1.72	–.022
Verbal IQ	108.75	18.41	–.015
Performance IQ	105.25	18.23	–.072
Full Scale IQ	107.49	14.43	–.066
FDI (scaled score)	10.11	2.12	–.025
ACID (scaled score)	10.09	1.77	–.072

Note. RPM = *Raven Progressive Matrices* (Raven, Court, & Raven, 1988); CPT = *Continuous Performance Test* (Conners, 1992); WCST = *Wisconsin Card Sorting Test* (Heaton, 1981); FDI = Freedom from Distractibility Index; ACID = Arithmetic, Coding, Information, and Digit Span composite.
*$p \leq .05$ (two-tailed), **$p \leq .01$ (two-tailed).

Raven IQ and EF

The correlation of the three EF measures with RPM IQ were as follows: IMPULS, $r = -.04$; PERSEV, $r = -.25$; and FAILSET, $r = .07$. Obviously, the correlations of the RPM IQ with IMPULS and FAILSET were nonsignificant. The correlation of PERSEV with the RPM IQ was statistically significant ($p = .005$) and of the same order of magnitude as the correlation of PERSEV with FSIQ, accounting for about 6% of the variance. Thus, none of the three EF measures showed more than a modest correlation with either FSIQ or "fluid" intelligence as measured by the RPM.

ACID and FDI

The correlations of FSIQ with the WISC-III composite scores that are thought to be sensitive to the neurocognitive deficits found in ADHD (FDI and ACID) were highly significant: FDI, $r = .78$, $p \leq .000$, and ACID, $r = .70$, $p \leq .000$—not a surprising finding, as these are not independent measures. It is of interest to note that for this sample, consisting of boys with ADHD who might be expected to have at least some degree of dissociation between FSIQ and FDI or ACID, the associations among the three indices remained quite strong. By way of comparison, in the WISC-III standardization population, stratified by age groups, the extent to which FSIQ could be estimated from the FDI score ranged from .61 to .74. Thus, for the current ADHD sample, the correlation is actually higher. For the independently derived RPM IQ, our "fluid" intelligence measure, we also found moderately strong associations with ACID ($r = .51$) and FDI ($r = .49$).

If ACID, FDI, IMPULS, PERSEV, and FAILSET are all sensitive to deficits found in children with ADHD, then significant intercorrelations among these five measures might be expected. In fact, statistically significant correlations were found between IMPULS and ACID ($r = -.18$) and between PERSEV and FDI ($r = -.32$), although these were not nearly of the magnitude of the correlations of ACID or FDI with FSIQ. With respect to the interrelationships among the three EF measures themselves, the correlations between IMPULS and PERSEV ($r = -.09$) and IMPULS and FAILSET ($r = .05$) were not significant, whereas the correlation of PERSEV with FAILSET ($r = -.17$) was significant but in the unanticipated direction (i.e., because both are error scores, a positive correlation would be predicted).

Principal Components Analysis

In order to consider these results in the larger context of general intelligence (*g*), the non-independent composite scores (VIQ, PIQ, FSIQ, FDI, and ACID) were removed from the data set, and the scaled scores from the WISC-III individual subtests, the RPM IQ, and IMPULS, PERSEV, and FAILSET were factor analyzed, using an unrotated principal components solution. The first component, accounting for 35% of the variance, clearly met the criteria for psychometric *g* (Jensen, 1998). The factor loading matrix, shown in Table 3, indicates that the three variables with the three highest *g* loadings were the RPM IQ, Comprehension,

TABLE 2
Correlation Matrix for All Dependent Variables, Including Composite Scores

	INF	SIM	ARI	VOC	COM	DSP	PCO	COD	PAR	BLO	OBJ	RIQ	IMP	PER	FAIL	VIQ	PIQ	FSIQ	FDI
INF	—																		
SIM	.53	—																	
ARI	.51	.34	—																
VOC	.60	.56	.51	—															
COM	.59	.62	.52	.53	—														
DSP	.17	.18	.56	.25	.38	—													
PCO	.41	.50	.26	.48	.45	.11	—												
COD	.19	.19	.22	.22	.17	−.02	.17	—											
PAR	.33	.44	.39	.46	.35	.24	.37	.31	—										
BLO	.25	.32	.19	.31	.28	.34	.45	.16	.32	—									
OBJ	.22	.31	.18	.23	.31	.29	.42	.17	.31	.67	—								
RIQ	.58	.37	.54	.51	.50	.33	.46	.15	.57	.51	.48	—							
IMP	−.07	.07	−.07	.06	.05	−.23	.12	−.09	.18	−.06	−.02	−.04	—						
PER	−.08	.00	−.17	−.04	−.12	−.30	−.16	−.02	−.07	−.25	−.26	−.25	−.09	—					
FAIL	.35	.03	.06	.07	.19	−.07	−.02	.13	−.06	.06	.11	.07	.05	−.17	—				
VIQ	.79	.62	.72	.75	.85	.39	.49	.26	.51	.33	.25	.35	.03	−.02	.23	—			
PIQ	.31	.46	.29	.40	.45	.20	.54	.56	.46	.70	.60	.63	.08	−.02	.15	.37	—		
FSIQ	.70	.73	.63	.71	.74	.37	.64	.46	.64	.57	.56	.66	.08	−.22	.19	.72	.73	—	
FDI	.50	.47	.79	.44	.77	.77	.34	.18	.47	.38	.35	.49	−.14	−.32	.08	.75	.38	.78	—
ACID	.63	.43	.84	.55	.58	.59	.32	.57	.48	.33	.28	.51	−.18	−.16	.13	.75	.48	.70	.81

Note. INF = Information; SIM = Similarities; ARI = Arithmetic; VOC = Vocabulary; COM = Comprehension; DSP = Digit Span; PCO = Picture Completion; COD = Coding; PAR = Picture Arrangement; BLO = Block Design; OBJ = Object Assembly; RIQ = Raven IQ; IMP = IMPULS; PER = PERSEV; FAIL = FAILSET; VIQ = Verbal IQ; PIQ = Performance IQ; FSIQ = Full Scale IQ; FDI = Freedom from Distractibility Index; ACID = Arithmetic, Coding, Information, and Digit Span.

TABLE 3
First Principal Component (*g*) Loadings of All Independent Measures for All Participants

Variable	Loading	Communality
Information	.723	.761
Similarities	.696	.621
Arithmetic	.677	.745
Vocabulary	.743	.673
Comprehension	.755	.664
Digit Span	.481	.811
Picture Completion	.661	.602
Coding	.321	.524
Picture Arrangement	.643	.555
Block Design	.609	.769
Object Assembly	.581	.756
Raven IQ	.794	.650
IMPULS	.009	.808
PERSEV	−.269	.657
FAILSET	.164	.861

Note. *N* = 123. IMPULS = *Continuous Performance Test* (Conners, 1992) impulsive errors; PERSEV = *Wisconsin Card Sorting Test* (Heaton, 1981) perseverative errors; FAILSET = *Wisconsin Card Sorting Test,* failures to maintain set.

and Vocabulary, consistent with most other findings (Jensen, 1998). Note that the loadings of the three EF measures on the first principal component, or *g*, were trivial. Of the three measures, only the loading of PERSEV was correlated with *g* in the predicted direction.

From inspection of the factor loading matrix, it appeared that the *g* factor scores of the participants might be reproducible with a high degree of fidelity without using any of the EF measures. To test this hypothesis, we entered the WISC-III subtests and the Raven IQ into a number of stepwise multiple correlation analysis models. As shown in Table 4, the *g* factor scores derived from the entire battery could be predicted with a high level of confidence using only four measures: Raven IQ, Comprehension, Vocabulary, and Similarities ($R = .900$; $F = 125.62$; $p < .000$). Forcing IMPULS, PERSEV, and FAILSET to the model resulted in the inclusion of only PERSEV, which increased R from .900 to .912, consistent with the modest *g* factor loading of this measure.

Discussion

Given that the IQ obtained from standardized tests is highly *g*-loaded, its measured level will be a reflection of the participation of numerous neural information-processing networks (Anderson, 2001; Crinella & Yu, 1995;

Jensen, 1998). Hence, the proportionate influence of a deficit in an EF component, or even in several EF components (as typically found in ADHD), would have a minimal influence in determining the ability to perform on highly *g*-loaded tasks (e.g., RPM), because so many diverse non-EF components are also required for performance on a such a task. This is clearly illustrated by the diversity of brain locales shown on functional magnetic resonance imaging (fMRI) studies of individuals who are in the process of solving RPM problems (Prabhakaran, Smith, Desmond, Glover, & Gabrieli, 1997).

TABLE 4
Stepwise Multiple Regression Prediction of First Principal Component (*g*) Factor Scores

Model	*R*	*F*	*p*
1. Raven IQ	.762	167.91	.000
2. Raven IQ Comprehension	.866	180.39	.000
3. Raven IQ Comprehension Vocabulary	.887	146.58	.000
4. Raven IQ Comprehension Vocabulary Similarities	.900	125.62	.000
5. Raven IQ Comprehension Vocabulary Similarities Perseveration	.912	115.27	.000

Composition of Individual EF Measures

As Table 2 shows, PERSEV had a number of significant but small correlations with other measures, most notably with FDI (–.32) and Digit Span (–.30), which are fairly collinear, and, as noted earlier, Digit Span is relatively unsaturated with *g* (Wechsler, 1958). However, PERSEV also had modest correlations with Object Assembly (–.26), Block Design (–.25), and especially FSIQ (–.22), which have relatively high *g* saturation (Jensen, 1998). The significant correlation of PERSEV with *g*-loaded subtests and with FSIQ is consistent with the report of Pennington et al. (1993) who found a –.38 correlation between FSIQ and perseverative errors. The magnitude of the PERSEV–FSIQ correlation in the present study is somewhat smaller, perhaps because we did not have control participants, who would no doubt have increased the range of PERSEV. In Pennington et al.'s (1993) study, the mean number of errors for the ADHD group was 37 (*SD* = 23.6), whereas the mean number of errors for the control group was 25 (*SD* = 18.0). As Table 1 shows, the mean number of perseverative errors for our all-ADHD population was 23.68 (*SD* = 13.73), so that a more modest correlation would be expected due to the constricted range of scores. Chelune et al. (1986) also found that children with ADHD and controls differed significantly on perseverative errors. On the other hand, Seidman et al. (1997) and Loge et al. (1990) did not find the PERSEV measure to have great diagnostic value in discriminating ADHD from non-ADHD groups. The vast difference between mean error scores for the two groups (Seidman et al., ADHD = 15.4, control = 9.1; vs. Loge et al., ADHD = 27.1, control = 24.0) points to the existence of other factors that are obviously not encompassed by the ADHD diagnosis. Taking these discrepancies into account, it is probably fair to say that the correlation of perseverative errors with IQ will tend to be statistically significant but of a magnitude that will account for only 10% to 15% of the variance.

As Table 2 also shows, the highest correlation for IMPULS was with Digit Span (r = –.23). IMPULS was not significantly correlated with any of the independent *g*-saturated measures. This finding represents a departure from the results reported by Pennington et al. (1993), who found a significant correlation between CPT errors and FSIQ. It is clear, however, that the version of the CPT used in our laboratory was much less complex in terms of the executive functions that were tapped by the version used in Pennington's laboratory, which required the implementation of a complex rule over an extended period of time, thus incorporating strategy comprehension and working memory elements. As noted by Plomin and Spinath (2002), "This could mean that working memory is the Factor X that explains *g*. . . . However, it seems more likely that working memory is just another name for *g*—tests of working memory look suspiciously like psychometric tests of *g*" (p. 5). The relatively low correlation of IMPULS with FSIQ no doubt reflects the fact that the CPT version used in our laboratory was a more or less "pure" test of impulse control, having very little to do with understanding complex rules or retaining significant amounts of information in working memory.

Finally, it is clear from inspection of Table 2 that FAILSET has several significant correlations with *g*-saturated measures like Information (r = +.35) and Verbal IQ (r = +.23), *but in the unanticipated direction.* That is, for this population of 123 boys with ADHD, a higher number of set failures was associated with better performance on *g*-loaded tasks. This perplexing finding

was verified after hand-rescoring the WCST. It is true that the distribution of scores for FAILSET was skewed, which could account for a smaller correlation, but not for the direction of the obtained relationship. Although the number of set failures we found was rather small (M = 2.12; SD = 1.72), our mean was higher than those reported by Loge et al. (1990; M = 1.2), Doyle et al. (2000; M = 1.8), and Pennington et al (1993; M = 0.9), who also found that the measure did not discriminate between ADHD and controls. Pennington et al. (1996) noted that the equivocal and often contradictory results of studies with the WCST may be due to the fact that a WCST error is "a bit like a pathognomonic sign: a good indicator of pathology but a poor measure of normal variation" (p. 335). Of course, such a pathognomonic measure need not have a significant *g* loading to be of importance when found.

Additional Evidence

There is additional evidence to be considered in concluding that *g* and EF are independent dimensions of cognitive performance. For example, in recruiting participants for clinical studies, it is usually found that among children who meet the criteria for the diagnosis of ADHD, only a few will be excluded because of low IQ (typically < 80)—certainly no more than the 8% predicted by the normal distribution of IQ scores. For example, Pennington et al. (1993) reported that no participants were excluded because of IQ < 80 (of course, this may simply have been due to unreported prescreening). More important, if EF deficits were sufficient to depress *g* to any great extent, a somewhat greater proportion of children with ADHD would have mental retardation, which is clearly not the case. Children with mental retardation unquestionably have more EF deficits than children without mental retardation (Gadow & Poling, 1988; Zeaman & House, 1963), but they also have deficits in sensory acuity, abstract concept formation, memory storage and retrieval, social perception, reaction speed, and many other neurocognitive components (Spitz, 1986).

An increasingly popular view of mental retardation is that a low IQ represents deficits in test-taking strategies—the result of EF dysfunction. In fact, a number of investigators have found that training in strategies is of benefit to persons with intellectual disabilities, helping them to learn a number of useful (but not particularly *g*-loaded) adaptive activities (e.g., Brown & DeLoache, 1978; Butterfield & Belmont, 1977). Jensen (1998) considered the proposition that individual differences in "strategic" tendencies (i.e., EF), could explain the significant correlation between *g* and measures of simple reaction speed, but he concluded that individual differences in both *g* and EF reflect individual differences in a more basic physiological property such as "neural efficiency." Jensen cited as evidence the nodal experiment by Alderton and Larsen (1994), wherein cross-task correlations in strategies were artificially constrained but intertask correlations (i.e., *g*-based differences) still remained. Thus, intelligent people, according to Jensen, do not "optimize strategies, but optimize performance" (p. 239).

It has never been convincingly demonstrated that IQ can be changed very much or for very long by interventions such as drugs, behavior modification, special education programs, or early infant stimulation (Baumeister & Bacharach, 2000; Carroll, 1993; Lipsey & Wilson, 1993; Schwean et al., 1993; Spitz, 1986). On the other hand, treatment with stimulant medication alone is usually found to have a positive benefit for children with ADHD (MTA Cooperative Group, 1999), particularly in improving EF (Barnett et al., 2001; Douglas et al., 1988; Whalen & Henker, 1991). For example, Barnett et al. (2001) showed that medicated children with ADHD scored higher than nonmedicated children with ADHD on spatial working memory, considered to be an EF-saturated task. Pharmacological intervention enhances the child's ability to use intellectual resources by optimizing EF.

The Special Case of Frontal Lobe Patients

As discussed in preceding sections, neuroanatomical, neuropsychological, and psychopharmacological evidence converges on the finding that the EF system, which is dysfunctional in children with ADHD, includes the frontal cortex and interconnected subcortical structures. The frontal cortex is involved with subcortical structures in several distinctive circuits supporting feedback control (Alexander, DeLong, & Strick, 1986). Of particular relevance is the connective loop formed by projections from the frontal cortex to the caudate nucleus and anterior putamen (Villablanca & Olmstead, 1982), from these structures to the substantia nigra, and from there back to the frontal cortex (Parent, Bouchard, & Smith, 1984; Porrino & Goldman-Rakic, 1982). Fuster (1989) reported that this loop is involved in "global, abstract, and schematic aspects of behavioral action" (p. 25) and presented a wealth of evidence to support the theory that the frontal lobes participate in one or more of the closed loop circuits that mediate human EF.

At the turn of the 20th century, it was generally believed that human intelligence emerged in conjunction with the phylogenetic appearance of the frontal lobes. Flechsig's (1901) dictum that complex mental functions paralleled the course of myelination, coupled with the finding that the frontal lobes are among the last structures in the brain to myelinate, was usually given as a rationale for this belief. Hebb (1949) summed up much of this speculation as "an enormous effort to show that the frontal lobe is the seat of some higher function or other. There are few mental processes that have not been ascribed to it" (p. 286).

Since Hebb's (1945, 1949) nodal studies, there has been a growing appreciation of the fact that frontal lobe lesions do not affect intelligence as

measured by IQ tests (Mettler, 1949; Teuber, 1964; Weinstein & Teuber, 1957). However, the erroneous conclusion that the frontal lobes are "silent" brain areas was soon dispelled by evidence that frontal lobe deficits could be easily demonstrated using less complex tests (i.e., those with relatively low *g* loadings). Thus, there is a long-standing body of evidence that lesions to a very critical structure in the human EF system, the frontal cortex, do not cause significant deficits in psychometric *g*, as represented by IQ.

Recently, Denckla and Reiss (1997) made a case for the preeminence of the basal ganglia dysfunction in ADHD, suggesting at the same time that IQ is mediated by the prefrontal cortex. This idea coincides with emerging views of a "fluid" intelligence mediated by the prefrontal cortex (e.g., Duncan et al., 1995; Pennington, 1997; see the next section). In this view, because children with ADHD can have above-average IQs, frontal lobe dysfunction cannot be the source of EF deficits. Furthermore, the "overgrowing" of the prefrontal cortex leads it to "dominate its subcortical partners," a process that may underlie the observation that a significant proportion of children with ADHD cease manifesting ADHD-like symptoms in adult life. As noted by Plomin (1999), individuals who outgrow ADHD may do so because they are able to "seek out and create environments correlated with their genetic propensities" (p. C25). Unsaid is the likelihood that they will seek out environments that do not expose their EF weaknesses.

Other "Intelligences"

In the current cultural context, the assertion that IQ, as measured by standardized psychological tests, may be a reasonable estimate of general intelligence is often met with harsh argument, and it has become fashionable to speak of non–IQ-based forms of intelligence (Gardner, 1983; Goleman, 1995; Sternberg et al., 2000). This modern trend is reminiscent of similar views of half a century ago, spurred by the fact that IQ deficits had not generally been found in frontal lobe patients. The obvious disorganization of personality seen in the frontal lobe patient became the impetus for developing measures of human intelligence with more adaptive merit than psychometric IQ. Halstead's (1951) distinction between biological and psychometric intelligence was perhaps the earliest and most enduring example of this movement. Halstead explained that the intelligence associated with frontal lobe deficits is reflected in impaired survival skills ("controlled adaptability") rather than in decrements of IQ. As we shall see, Halstead was absolutely correct in theorizing a form of cognitive activity that accounts for "controlled adaptability," which could easily be relabeled as EF, and for pointing out that biological intelligence and *g* were not identical processes.

Duncan and colleagues (e.g., Duncan et al., 1995; Duncan et al., 1996; Duncan et al., 2000) have used measures of "fluid intelligence" to demonstrate intellectual deficits associated with frontal lobe lesions. Duncan asserted the following:

1. Measures of fluid intelligence (e.g., *Raven Progressive Matrices*) have higher correlations with *g* than do conventional IQ scores.
2. Fluid *g* is primarily a manifestation of EF, whereas crystallized *g* (as measured by conventional IQ tests) is not.
3. When more appropriately measured by tests of fluid intelligence, *g* is affected by frontal lobe lesions.

Duncan et al. have conducted a number of experiments with frontal lobe patients that would seem to support their contention. Also, Pennington (1997) has reported that at least one EF component, "set-shifting," has a significant correlation with fluid *g*, as represented by the RPM IQ.

However, we have previously shown that it is possible for at least some individuals with frontal lobe injuries to show very high RPM IQs while at the same time manifesting serious set-shifting errors on the WCST (Crinella & Yu, 2000). Moreover, Prabhakaran et al. (1997), using fMRI scanning of healthy volunteers solving RPM problems, showed that complex problem-solving activity activated several nonfrontal areas of the brain, including parietal, occipital, and temporal regions. Furthermore, Van Rooy, Stough, Pipingas, Hocking, and Silberstein (2001), using steady-state probe topography to monitor average- and high-IQ participants on a working memory task, found greater amplitude and latency differences in the high-IQ group, particularly in posterior areas of the brain (e.g., occipital lobes). Studies of this nature are consistent with Anderson's (2001) conclusion that IQ tests are "complex and depend for their successful performance on the coordinated, efficient function of distributed areas of neurons in the brain" (p. 368).

In the present study, we included the RPM IQ as an indicator of fluid intelligence. There are several noteworthy findings. First, the correlations between the RPM IQ and two of the three measures of EF (IMPULS and FAILSET) were not significant. Consistent with Pennington et al.'s (1993) finding, our measure of set shifting (PERSEV), was significantly correlated with the RPM IQ ($r = -.25$) but would account for very little of the variance in the latter. Thus, EF does not predict the "fluid" RPM IQ any more than it predicts the relatively more "crystallized" WISC-III FSIQ, or, for that matter, the *g* factor score derived from the entire battery. Finally, the loading of the RPM IQ on the *g* factor derived from this battery was .794, of a magnitude similar to the *g* loadings for Comprehension and Vocabulary, suggesting that fluid and crystallized intelligence are far from independent entities.

Animal Models of the Dissociation of g *and EF*

In animal studies, we have attempted to identify discrete brain areas that

might constitute the neural substrate for EF and to determine whether this "rodent EF system" became more critical for performance as the *g* loadings of laboratory tasks escalated. Our findings were that (a) lesions in some brain areas depressed *g* factor scores more than lesions in other areas, irrespective of lesion size; (b) a problem's *g* loading tended to be of similar magnitude, irrespective of the test battery in which it was imbedded; and (c) a task's *g* loading tended to be an essentially one-to-one correlate of the number of brain areas that were critical for the task's performance. We also reported that bilateral lesions to any one of eight structures (substantia nigra, caudatoputamen, ventral tegmentum, pontine reticular formation, globus pallidus, ventrolateral thalamus, median raphe, and superior colliculus) significantly affected performance on a wide variety of problem-solving tasks (Crinella & Yu, 2000; Thompson, Crinella, & Yu, 1990). These structures are of particular interest because they mediate EF components such as (a) Shifting cognitive sets (Thompson, 1982a, 1982b; Thompson & Yang, 1982); (b) response flexibility (Thompson, Harmon & Yu, 1984); (c) response inhibition (Thompson, Bjelajac, Huestis, Crinella, & Yu, 1989; Thompson, Harmon, & Yu, 1985); (d) transfer strategies (Thompson, Bjelejac, Fukui, et al., 1989); and (5) working memory (Thompson, Huestis, Bjelejac, Crinella, & Yu, 1989). On the basis of this evidence, we felt justified in considering these eight brain structures to be the biological substrate for EF, or the "EF system."

If *g* were largely dependent on EF, then lesions to the EF system should cause a proportionately greater depression of *g* factor scores than lesions to other brain areas. However, our attempts to validate this hypothesis resulted in a contrary finding—namely, that lesions causing the greatest decrement in performance on highly *g*-loaded laboratory tests were more often *not* to structures comprising the EF system. In fact, there was relatively little overlap between the EF system and the brain structures that were most strongly associated with psychometric *g* (Crinella & Yu, 1995, 2000). The non-EF brain structures important for solving *g*-loaded problems were extensive, including, in particular, structures that have been shown to perform computations on complex information, both external (e.g., parietal cortex, occipitotemporal cortex, dorsal hippocampus) and internal (e.g., posterolateral hypothalamus, subthalamus, anterior thalamus).

A careful analysis of the behavior of animals with EF system lesions showed a pattern of difficulty in selecting adaptive behaviors from among their existing repertoires and in initiating behavioral subroutines that would have enabled them to make progress toward a goal. They were unable to execute the neural programs necessary to obtain their goal, a behavior that Luria (1966) described as a disorganization of "system-selective behavior" (p. 302).

Our rodent evidence for subcortical involvement in EF would seem to converge with the clinical observations of children with ADHD (Denckla & Reiss, 1997), the imaging evidence showing that basal ganglia structures are at least as probable as the prefrontal cortex as neuroanatomical substrates for ADHD (see Figure 3), and the correlative neuroanatomical evidence demonstrating basal ganglia involvement in EF (Folstein, Brandt, & Folstein, 1990; Middleton & Strick, 1994; Phillips & Carr, 1987; Saint-Cyr, Taylor, & Lang, 1988; Schultz, 1998; Stern & Mayeux, 1986). Our work with the laboratory rat has shown that the EF system must be intact for the adequate performance of a wide variety of laboratory problem-solving tasks. However, the number of additional, non-EF brain structures necessary for unimpaired performance increases in proportion to the *g* loading of the task (Crinella & Yu, 1995). These findings support a view that the neuroanatomical EF system in isolation is not sufficient to act as the biological substrate for *g*.

Limitations

The present study has a number of limitations. First, the participants were all referred male patients, and their SES was constrained by the geographical location of the clinic. This selection bias does have the virtue of eliminating possible sources of confounds, such as well-known gender differences in ADHD and the presence of ADHD-like behavioral deficits that might be reactions to environmental disadvantages. However, the upward skewing of the mean IQ of the population tends to restrict the range and dispersion of scores, leading to somewhat lower correlation coefficients, favoring the null hypothesis of zero association between IQ (or *g*) and EF. It is doubtful, however, that expanding the range of IQs would have led to a substantive difference in the strength of the EF–IQ (or *g*) correlations observed, which were either trivial or quite modest.

We are well aware (see our introductory discussion of EF) that there exists a long list of candidate EF components and that many tests could have been used to represent them. We administered only two (the CPT and WCST), from which we selected only three measures for analysis. Both tests yield a number of additional scores, but it is important for correlational studies that the scoring categories be conceivably independent, and many of the scoring categories on the CPT and WCST are interdependent. As shown in Table 2, they were in fact quite independent of each other. Had we used a more extensive battery, with a larger number of putative EF components represented (e.g., Loge et al., 1990; Reader et al., 1994), we might have converged on an EF factor that had a stronger association with IQ (or *g*). However, we might also have run the risk of forcing the collective EF battery closer to *g* by a concatenation of elementary processes, which, in our view, is the basis of *g* (Crinella & Yu, 1995), or by the introduction of EF tasks with greater complexity than we have chosen here. With respect to the latter, it

has been shown that when a test of an EF component is made more complex (e.g., by introducing arithmetic operations into a working memory task), it will, in fact, have a higher correlation with IQ (Plomin & Spinath, 2002). In the current study, the three EF components used were specifically selected because they lacked complexity but still represented commonly accepted EF components. Deficits in these particular components (impulse control, set shifting, and set maintenance), if not exhaustive of EF, are arguably among the core features of ADHD.

It is also possible to look more exhaustively at a single elementary process, thereby building redundancy into the observation and leading to a more reliable measure of that process. For example, in another study, we used eight different laboratory tests to investigate the construct of inhibition (Thompson, Bjelajac, Huestis, et al., 1989). However, factor analysis of the battery showed that two distinct factors were involved.

We also recognize that there was considerable phenotypic variation among our participants, even though all were diagnosed with ADHD using *DSM-IV* criteria. These diagnostic criteria can be satisfied by individuals with substantial behavioral heterogeneity—a continuing source of error in studies of ADHD. In the simplest instance, the criterion for the diagnostic classification of ADHD, predominantly hyperactive–impulsive type (*DSM–IV* 314.01)—at least six of nine listed symptoms present—may be met by any one of 130 combinations of symptoms. The recent work of Swanson et al. (1998), using a phenotype refined beyond *DSM-IV* criteria, provides an example of the reduction of behavioral heterogeneity to produce more reliable findings. There now exists the added possibility of using genotypic similarities in examining the neurocognitive status of children with ADHD (e.g., Swanson et al., 2000).

Because these data were gathered from a referred population, we did not have a nonclinical control group. It would have been of interest to investigate the structure of performance on this battery for a population with greater heterogeneity or to determine whether the *g* versus EF distinction would hold true for a large random sample of the population. Recall, however, that Welsh et al. (1991), using a somewhat similar approach, did a normative study with a nonclinical population in which the *g* versus EF distinction clearly held.

Finally, the analyses performed on these data were limited in scope to an exploration of the relationship between EF and IQ, or the first principal component in our test battery (*g*), and not the broader dimensional structure of the test battery. Our emphasis on the *g* factor necessitated that we ignore other factors, each of which would have merited attention in a study of the overall structure of cognition.

Implications

Clinical. In describing their child with ADHD, parents are invariably quick to emphasize that he or she is "bright." They worry that their child will be mistakenly labeled as less intelligent than others because of poor productivity and, thereby, suffer irreversible academic harm because of lowered teacher expectations. A comprehensive review of the child's performance in the neuropsychological laboratory will usually show parents that under conditions that minimize the influence of inattentiveness (e.g., reduced distractions, high-interest stimuli), the child can solve complex (i.e., *g*-loaded) problems at least as well—and usually better—than he or she performs on simple laboratory tests of EF. The present study may offer clinicians and, indirectly, parents and teachers some added insight into why the child with ADHD is capable of such performances in the testing laboratory but not in school—namely, that the less structured school environment will increase the risk of sabotage by a fragile EF system.

In truth, the most worrisome deficits of children with ADHD are not the product of low IQ, but rather of the instability of control processes that govern everyday adaptations to the environment (i.e., EF). The vast majority of the adaptations required would be considered minimally *g*-loaded tasks, but they are nevertheless essential for optimal productivity. Most parents are acquainted with the fact that their child's IQ will not change appreciably over time. Parents of children with ADHD harbor the fear that the same will hold true for their child's level of self-sufficiency. In fact, the potential of the child with ADHD can be harnessed effectively with behavioral and pharmacological interventions that are known to improve EF. Even for the child with a lower than average IQ, interventions that promote the establishment of a wider repertoire of minimally *g*-loaded abilities could conceivably make the difference between success and failure in both academic and social arenas. Although it is necessary to recognize that a broad repertoire of EF-mediated adaptive behaviors will not substitute for IQ when it comes to the likelihood of success in calculus or physics, it is also important to convey to parents that a high IQ is no substitute for predominantly EF-based "street smarts." In this regard, a fascinating study of children with ADHD, conducted by Lawrence et al. (2002), demonstrated that they were significantly different from children without ADHD when required to take a prescribed route through a real zoo (e.g., "from the Reptile House via the Penguin Plunge to the Crocodile House"), especially when more complex routes were assigned. Interesting enough, although the instructions that they needed to follow were given orally, Verbal IQ did not correlate with performance on the assigned tasks.

Theoretical. Studying the unique clinical disorder that is ADHD provides us with an opportunity to examine the distinction between elementary problem-solving components, such as

those composing EF, and global problem-solving capacity (*g*), as only the former is affected by the disorder. In doing so, we revisit old controversies among intelligence theorists and students of the functional organization of the higher central nervous system. Most of our conclusions have, in some guise, been stated before—in some instances as far back as the "frontal lobe" literature of more than half a century ago (e.g., Halstead, 1951; Hebb, 1949). Sadly, the views of these early investigators are periodically rediscovered with scarce acknowledgment of their origins. The fact that the application of new technologies often results in the re-emergence of analogous theories is an indication of the prescience of these early investigators and of the timelessness and impenetrability of the issues they identified.

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