Review of Educational Research
Spring 2002, Vol. 72, No. 1, pp. 61–99

A Review of the Research on Interventions for Attention Deficit Hyperactivity Disorder: What Works Best?

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This meta-analysis examined 74 studies in which there had been an intervention that aimed to improve the behavioral, cognitive, and/or social functioning of people with attention deficit hyperactivity disorder (ADHD), or attention deficit disorder (ADD). Overall, there were larger effects of the various interventions on behavioral than on educational outcomes. These overall effects were larger for medical interventions than for educational, psychosocial, or parent training interventions, but there was little support for flow-over effects, from the reduction in behavior problems to enhanced educational outcomes. The effects on educational outcomes were greater for educational interventions than for other types of intervention.

KEYWORDS: *attention deficit hyperactivity disorder, educational interventions, medical interventions, meta-analysis, psychological interventions.*

In the 1990s, there was a remarkable increase in the number of students diagnosed as possessing attention deficit disorder with or without hyperactivity. This increase has been accompanied by an upsurge in the administration of drugs to "assist" these students, particularly in classroom activities, and this remedy has led to many controversies. The efficacy of a range of interventions for attention deficit disorder is still a matter of lively debate, and there is growing concern over the increase in diagnosis, the rate of prescription of drugs, and the use of medication as the sole method of treatment. Juxtaposed with the increase in diagnosis is a phenomenal growth in the literature on this topic. For instance, in searches of Medline, PsycLIT and ERIC from 1990 to 1999, we found 1,379, 2,546, and 436 citations, respectively, in which *attention deficit hyperactivity disorder* (ADHD) was a key phrase. In a similar period of time, one decade earlier, there were 40, 317, and 35 citations, respectively, in the same electronic databases. Even when the key phrase *attention deficit disorder* (ADD) was used (to account for the previously more commonly used term), the citations in this earlier period were 450 in Medline, 833 in PsycLIT, and 82 in ERIC.

The implications of these figures are twofold. First, if the volume of literature is so vast and growing rapidly, why would we wish to present yet another review on attention deficit disorder? Much of the literature reports findings with little attention to the size or magnitude of the effect. Hence, this study uses meta-analytic methods to provide an assessment of the magnitude of the effects and evaluates these effects relative to a number of possible moderators. Although there have been several meta-analytic reviews since the 1990s that have examined the effectiveness of interventions for students with attention deficit disorders (e.g., Baer & Nietzel, 1991; DuPaul & Eckert, 1997; Swanson et al., 1993), none of these compare the full range of interventions; instead, they focus on either pharmacological or behavioral treatments. In addition, the Swanson et al. review was a review of three previous meta-analyses that were conducted more than a decade earlier and focused on hyperactivity only.

Second, most of the literature has been conducted from medical and psychological perspectives rather than from an educational one. It appears at first glance in reviewing the literature that programs for students with attention deficits are medical rather than educational, although most make claims that the program ultimately enhances educational outcomes, such as achievement. A focus of this article is the effects of the interventions on educational outcomes.

Thus, our paper aims to contribute in a unique way to the educational literature by synthesizing the past decade of research on the full range of interventions for students with attention deficit disorders. In this study, we report the results of our meta-analytic study, synthesizing the findings of 74 studies that investigated the effects of a range of interventions (pharmacological, school-based psychological, non-school-based psychological, parent training, and multimodal) on the cognitive, social, emotional, and physical development of students classified as having attention deficit disorder with or without hyperactivity (ADHD/ADD, hereafter abbreviated ADHD primarily because the latter is the more inclusive term and is used in almost all articles). We also examine how effects are moderated by various attributes of the studies, such as the source of publication, the design of the study, the characteristics of the subjects (age, gender, comorbidity), the type of outcome measure used, the outcome type, and intervention setting.

Most of the hundreds of articles on ADHD summarize the current state of affairs with respect to the disorder in strikingly similar ways. First, there is usually a summary of the epidemiology of the disorder that includes the quoting of prevalence statistics that show (a) a rapid rise in prevalence over the last decade or so; (b) variation both within and across countries and cultural groups; and (c) greater frequency of the disorder in males than in females. Second, in terms of etiology, we are generally told that the causes of ADHD are unknown or mixed (psychosocial, biological, hereditary). A third set of attributes of the disorder concern clinical diagnostic criteria, and typically noted are high rates of comorbidity with other disruptive behavior disorders. Finally, review articles generally include a consideration of the various methods of management of the disorder. Because the focus of the current meta-analysis is on interventions, we consider only briefly epidemiology, etiology, and diagnostic criteria of the disorder and provide a more detailed review of the literature on interventions for students with ADHD.

Epidemiology

The generally accepted prevalence figure is 3% to 5%, although much higher rates are frequently reported, and the variability is large. Current prevalence rates

obtained in various countries, however, generally exceed the 3% to 5% reported by the DSM-IV (American Psychiatric Association [APA], 1994). For instance, Whalen (1989) reported prevalence rates for ADHD among school-aged children as being between 20% and 24%, whereas Zentall, Harper, and Stormont-Spurgin (1993) reported rates of 3% to 15%. Searight and McLaren (1998) suggest several possible reasons for the variability in prevalence rates, including changes in diagnostic standards, overlap between ADHD and other externalizing disorders, and economic factors that have led to reductions in mental health, education, and managed care services, thereby promoting the "medicalization" of ADHD.

In the last decade, there has been a rapid rise in the reported prevalence rates of ADHD. Robinson, Sclar, Skaer, and Galin (1999) examined the National Ambulatory Medical Care Survey data for the years 1990 through 1995. They found that the number of office-based visits documenting a diagnosis of ADHD increased from 947,208 in 1990 to 2,357,833 in 1995. This increase in diagnosis was matched with a 2.9-fold increase in the number of ADHD individuals prescribed stimulant medication.

Males are more likely to be affected with ADHD than females. According to Sagvolden and Archer (1989), 90% of all children diagnosed with disorders of attention are boys. Williams, Wright, and Partridge (1999) claim that boys are 5 to 9 times more likely to be affected with ADHD than girls. Gaub and Carlson (1997) reviewed 18 studies examining gender differences in ADHD and found that the ratio of male to female prevalence of ADHD is 3:1 in community samples and between 6:1 and 9:1 in clinic-referred samples. They found that ADHD girls relative to ADHD boys showed lower levels of hyperactivity, fewer conduct disorder diagnoses, and lower rates of other externalizing behavior but greater intellectual impairment. Regardless of gender, there is now an increasing recognition that ADHD persists into adolescence in at least 50% to 70% of cases (Barkley, 1990; MacLeod & Prior, 1996; Weiss & Hechtman, 1992).

Etiology

A number of genetic, neurochemical, neurobehavioral, and neuroimaging studies have reported a biological predisposition to ADHD (e.g., Barkley, 1990; Goodman & Stevenson, 1989; Hynd, Hern, Voeller, & Marshall, 1991), whereas others claim that the etiology is unknown (e.g., Cantwell, 1996). Goodman and Stevenson estimated that hereditary factors in ADHD account for 30% to 50% of variance. Neurochemical research has pointed to the importance to attention, behavioral inhibition, and motor activity of the neurotransmitters dopamine and norepinephrine (Zametkin & Rapoport, 1987). Neuroanatomical research has demonstrated the importance of specific areas of the brain (e.g., frontal lobe, caudate nucleus in the basal ganglia, and right hemisphere) in the regulation of attention, impulsivity, and hyperactivity (McMullen, Painter, & Casey, 1994).

The other most frequently cited etiological association relates to parenting and parental mental health. Critical parenting and limited parenting skills have been associated with hyperactivity (Taylor, Sandberg, Thorley, & Giles, 1991; Williams et al., 1999). Research findings suggest that children who have experienced a chaotic home environment are more likely to display attentional difficulties at school (Cantwell, 1996); that marital disharmony, family dysfunction, and overt hostility between child and parent are associated with hyperactivity (Brandon, 1971; Gillberg,

Carlstrom, & Rasmussen, 1983; Tallmadge & Barkley, 1983); and that there is an association between maternal depression and ADHD (Biederman, Faraone, Keenan, & Tsuang, 1991; Lahey, Piacentini & McBurnett, 1988). Barkley (1985), however, cautions that parental reactions may be responses to the behaviors exhibited by the children rather than causes of the behaviors. Williams et al. (1999) list the following as other possible etiological factors: thyroid dysfunction, birth experiences, early life experiences, structural brain damage, and adverse social factors. Numerous other causes have been proposed, including various food additives, sugar intoxication, lead poisoning, and nutritional deficiencies, but there has not been substantial empirical support (Arnold & Jensen, 1995; Barkley, 1990).

Models of ADHD

Barkley (1997a, 1997b) summarized the history of ADHD in terms of the explanations that have been proposed for the behavior of people with ADHD (although that term was not used in early descriptions of the disorder). Initially, poor volitional inhibition and defective moral regulation of behavior were proposed as being central to the understanding of ADHD. Later explanations emphasized hyperactivity (e.g., Chess, 1960), the inability to sustain attention, and poor impulse control (Douglas, 1972). Douglas (1983) expanded her description of the key features of ADHD to include four major deficits: poor investment and maintenance of effort, deficient modulation of arousal to meet situational demands, strong inclination to seek immediate reinforcement, and deficient impulse control. These four deficits were seen to arise from an overarching impairment in self-regulation. Defective motivational processes (Glow & Glow, 1979), deficient stimulus control, a diminished sensitivity to reinforcement, and deficient rule-governed behavior (Barkley, 1981, 1989; Haenlein & Caul, 1987) all have been proposed as central to an understanding of ADHD.

The most recent explanations of ADHD, however, have emphasized behavioral inhibition as the central impairment of the disorder, and it is on the basis of this notion that Barkley (1997a, 1997b) has attempted to develop a unifying theory of ADHD that incorporates theories of the neuropsychological functions of the brain's prefrontal lobes. In contrast to the current clinical view that ADHD is primarily an attention deficit, Barkley's theory holds that ADHD comprises a deficit in behavioral inhibition. Inhibition is linked to four executive neuropsychological abilities. *Working memory* allows for the holding of events in mind and the manipulation of, or acting on the basis of, those events; imitation of complex behavior sequences; forethought and hindsight; and having a sense of time. *Internalization of speech* makes it possible to describe and reflect on one's own behavior; to engage in moral reasoning, self-questioning, and problem solving; to self-instruct; and to generate rules. *Self-regulation of affect, motivation, and arousal* enables emotional self-control; perspective taking; the self-regulation of drive and motivation; and the engagement in goal-directed action through the regulation of arousal. *Reconstitution* involves the analysis and synthesis of one's own behavior through the accurate and efficient communication of information. Impairment in these executive abilities is associated with impairments in motor control; impairments in fluency and syntax, as exhibited by the lack of inhibition of task-irrelevant responses; insensitivity to response feedback; inability to execute goal-directed responses or responses that are novel or involve complexity; and impairment in the ability to re-engage in a task after having been disrupted.

Diagnostic Criteria

McBurnett, Lahey, and Pfiffner (1993) noted that every new version of the DSM has included a major revision of ADHD criteria. Currently, three subtypes have been identified in the most recent DSM-IV (APA, 1994), namely predominantly hyperactive-impulsive type, predominantly inattentive type, and combined type (see McBurnett et al., 1993, for a detailed review of issues related to the diagnosis of ADHD). Diagnosis of ADHD is complicated and often subjective in nature. At present there is no single acceptable measure to diagnose ADHD. Identification of the disorder is usually based on a clinical diagnosis that is dependent on direct observation, parent interviews, teacher interviews, behavior rating scales, situation questionnaires, psychoeducational testing, and medical evaluation (McMullen et al., 1994). Because of the difficulties often associated with diagnosis, multidisciplinary and multiagency teams are frequently assembled to facilitate the process (Williams et al., 1999). Pediatricians, psychiatrists, psychologists, teachers, parents, therapists, paraprofessionals, and people with ADHD themselves are often involved in the classification procedure.

In general, the literature supports considerable comorbidity of ADHD with conduct disorder, oppositional defiant disorder, mood disorders, anxiety disorders, learning disabilities, and other disorders, such as mental retardation, Tourette's syndrome, and borderline personality disorder (Biederman, Newcorn, & Sprich, 1991). Pelham, Wheeler, and Chronis (1998) noted 50% to 75% comorbidity with other disruptive behavior disorders.

The Current Context: The Social Construction of ADHD

Recent discussions of the nature of disability have included strong arguments for the notion that disability is not an essential quality but rather that it is a social construction (e.g., Barnes, Mercer, & Shakespeare, 1999; Christensen, 1996; Marks, 1999). The notion of who fits into the category of "disabled" changes depending on a wide range of technological, organizational, and cultural factors (Marks, 1999). To this, some would add geographic location as a deciding force in the construction of disability. For instance, with respect to ADHD, Slee (1996) argues that "geographical disproportionality in diagnosis seems to indicate the prevalence of diagnostic predisposition as a causal factor" (p. 107).

A medical model of disability assumes that there is a readily identifiable norm of behavior. The basic cause of an individual's diversion from this norm is an underlying pathology or disease, which requires appropriate diagnosis so that symptoms can be effectively treated. In an education context, symptoms are usually based on the failure of a child to function appropriately in the classroom. But the line between acceptable and unacceptable classroom behavior and performance is extremely blurred (Christensen, 1996). This means that notions of what constitutes normal classroom behavior have led to the application of the label ADHD to some children who simply move around too much, who do not pay proper attention to the task in hand (usually one imposed by the teacher), or who blurt out answers without stopping to think about what they are saying. For these children, the educative role of the school can become subordinate to the medical role that is forced upon it by the medicalizing trends at work in the wider society. The graphic descriptions of lunchtime lines of children receiving their medication for ADHD (e.g., Stein, 1999) are a stark reminder of this trend.

Recent arguments against the disease concept for attentional disorders (e.g., Breggin, 1998; Breggin & Breggin, 1995; Kendall & Braswell, 1993; Stein, 1999) have led to the proposition that many children diagnosed as having ADHD do not pay attention to what they are doing because they are not motivated to behave and perform well. Others argue that typical ADHD behaviors are often within the realm of normal behavior for children but are interpreted differently by different people. "Attention Deficit Disorder is not a disease, it's part of the spectrum of children's behaviour. The issue is to find the line where abnormality stops and normality begins, . . . and the line moves according to who's drawing it" (*Speed for Breakfast*, 1995).

Interventions

Supporters have little difficulty in finding literature to justify the use of a favored method of treatment. Despite claims to the contrary (Stevenson & Wolraich, 1989), there is not a consensus as to which intervention for students with ADHD is the most effective. The categorization of types of intervention varies in the literature but generally includes the broad groupings of pharmacological, behavioral, and educational interventions and various combinations of these (Pelham et al., 1998). This is a deceptively simple categorization of the types of intervention that have been used to modify the behavior and learning of children (and adults) with ADHD; the reality is that the nomenclature used to describe the various approaches to treatment is vast, sometimes overlapping, and frequently confusing. Within each of the broad categories of intervention, there is considerable variation in terms of the type and reported effectiveness.

Pharmacological Interventions

Medication is the most commonly reported form of intervention for children with ADHD. Swanson et al. (1993) published a "review of reviews" on the effects of stimulant medication on children with ADHD. Swanson's team of researchers compared three types of reviews published in the late 1970s and 1980s, selected from meta-analyses (Kavale, 1982; Ottenbacher & Cooper, 1983; Thurber & Walker, 1983), traditional narration descriptive reviews (Adelman & Compas, 1977; Barkley, 1977; Whalen & Henker, 1976), and general public reviews (Kohn, 1989; McGuiness, 1989; Schrag & Divoky, 1975). These three types of reviews found that stimulants have an effect on attention, concentration, and motivation but no clear effect on academic performance or learning; that stimulants may be used as a "crutch" when implemented in the short term; and that medication treatments may postpone the use of nonpharmacological intervention that may be more effective in the long term. Kohn (1989) concluded similarly and claimed that the drugs "may have much greater relevance for stress reduction in caregivers than intrinsic value to the child" (p. 98).

Some studies of medical treatments have highlighted the notion that drugs do not "cure" the disorder and that the positive effects are limited in scope (i.e., there is not complete "normalization," as some symptoms persist or are only partially alleviated), are short term (they typically dissipate after about four hours after ingestion of the medication), and do not generalize to situations in which treatment is absent (Whalen & Henker, 1991). In addition, a number of side effects of medical treatments (such as weight loss, shakiness, dry mouth, appetite loss, and somatic effects of treatment) have been highlighted (e.g., Klorman, Brumaghim, Fitzpatrick, & Borgstedt, 1990). There are other reported limitations of medication.

For instance, although 70% to 80% of ADHD children show improvements in some aspects of their behavior, such improvements generally do not bring them "even close to being normalized—often remaining one standard deviation above the norm" (Pelham et al., 1998, p. 191) on impulsivity and related behavior outcomes.

Although studies of the efficacy of pharmacological treatments have shown positive effects on some target behaviors such as on-task classroom behavior, disruptive social behavior, and negative peer interaction, Pelham et al. (1998) report that there is no evidence showing long-term academic gains for medicated students or meaningful changes in negative peer nominations. Certainly, pharmacological treatment is financially cheaper than psychosocial intervention and for this reason is often the preferred mode of intervention.

Behavioral Interventions

Behavioral interventions (variously referred to as "behavior therapy," "behavior management," "contingency management," and "response cost") use principles of reinforcement and punishment to reduce problematic behaviors and increase desirable behaviors (Damico & Armstrong, 1996). Fiore, Becker, and Nero (1993) reviewed behavioral interventions and concluded that the three most common approaches—positive reinforcement, punishment, and response cost—can be effective in establishing behaviors conducive to classroom learning. There are many studies demonstrating that use of behavior management techniques in combination with stimulant medication can be even more effective than stimulant medication by itself (Gittelman-Klein, Abikoff, Pollack, Klein, Katz, & Mattes, 1980; Pelham, Carlson, Sams, & Vallano, 1993). Off-task behaviors are the most common targets for behavioral interventions, although Barkley (1990) noted that greater on-task behavior does not guarantee improved academic performance. Consequences or contingencies for ADHD children generally need to be more immediate, powerful, tangible, and frequent than those that teachers often use in their everyday work with other children in the classroom.

Cognitive Behavioral Interventions

Cognitive Behavioral Therapy (CBT) has evolved from the seminal work of Meichenbaum and Goodman (1971). In CBT, children are taught to use self-talk, self-instruction, self-monitoring, and self-reinforcement as problem solving and motivational strategies to develop self-control of their attention and impulse behavior problems. Ervin, Bankert, and DuPaul (1996) differentiated between several types of cognitive-behavioral interventions: those that are cognitively based, such as self-instruction and social problem solving; and those that are contingency-based, such as self-monitoring, self-evaluation, self-reinforcement, and correspondence training. Following a review of the different types of cognitive-behavioral interventions, these researchers concluded that such interventions are most effective when they are combined with behavioral contingencies in the natural environment and when they focus on specific training that matches the desired performance as closely as possible. However, despite noting some short-term successes in achieving behavior change, Ervin et al. found little evidence to suggest that the effects of cognitive-behavioral interventions generalize to times and settings in which the intervention is absent. Ervin et al. account for these disappointing results in terms of what they perceive to be the faulty premise on which many cognitive-behavioral

interventions for ADHD are based—that ADHD children lack the cognitive strategies needed to complete tasks and to interact successfully with other children.

Ervin et al. (1996) proposed that Barkley's (1994, 1997a, 1997b) theory of impaired delayed responding holds greater promise for the design and implementation of cognitive-behavioral interventions for ADHD students. To overcome the deficits in response inhibition, treatment must focus on encouraging children to delay their responses; it must take place in the setting and at the time that the problem behavior occurs (in the classroom or home rather than the clinic); and contingencies must be used to encourage ADHD children to delay responding and to apply cognitive strategies.

Research on the effects of CBT on children with ADHD has generally been conducted in clinical settings or research centers rather than in classrooms. In her review of three recent meta-analyses of CBT interventions, Poley (1995) highlights the generally poorer success rate of classroom-based CBT when compared with clinic-based CBT interventions. In the former, an effect size of only .11 was found by Dush, Hirt, and Schroeder (1989), whereas effect sizes ranging from .38 to .55 were found for the clinic-based CBT interventions (Durlak, Fuhrman, & Lampman, 1991; Lipsey & Wilson, 1993).

Parental Interventions

Parent training programs have been developed to assist parents in the development of appropriate skills to manage ADHD-type behaviors. Barkley (1990) recommends that programs should be customized to the age of the ADHD child, the severity of the ADHD symptoms, and the specific background and needs of all members of the family. Studies involving preschool children with ADHD and their families have shown that parent training can lead to increased child compliance and improvements in observed parenting skills (Anastopoulous, DuPaul, & Barkley, 1991; Pisterman, McGrath, Firestone, & Goodman, 1989). However, reviews by Fischer (1990), Mash and Johnson (1990), and Pelham and Lang (1993) indicate that families of ADHD children are often dysfunctional in a range of areas (e.g., maternal stress and depression, paternal alcohol abuse, and inappropriate parental discipline), and there is little to suggest that the clinical gains shown in structured parent-child interactions will carry over in such situations.

Educational Interventions

Educational interventions consist primarily of classroom academic management or the arrangement of learning environments in particular ways, such as reducing noise levels, structuring classrooms formally as opposed to informally, seating ADHD children in front seats, and providing frequent breaks between learning tasks. McMullen et al. (1994) provide an excellent summary of school-based interventions and services for children with ADHD. They discuss general school and teacher procedures, such as inclusive practices, team approaches, service plans, family involvement, and continuous assessment. They also discuss specific school-based methods that focus on behavior management, self-instruction, social skills training, cooperative learning, and peer tutoring.

A narrow range of educational interventions has been studied to date. In a review of educational interventions for students with ADHD, DuPaul and Eckert (1997) concluded that it was unclear to what degree these interventions enhanced

the academic achievement of students with ADHD because many studies did not include academic outcome measures. DuPaul and Eckert did find that educational interventions were at least as effective as contingency management strategies in terms of improving ADHD-related behaviors, but such improvements did not necessarily translate into improved academic performance.

With respect to educational interventions, Burcham, Carlson, and Milich (1993) noted that it is important to determine whether implementing a change in the educational design is likely to have a positive effect before taking action. Further, they suggested that it is necessary to decide whether a strategy that may effect positive change in a clinical or experimental setting will generalize to the school or home setting. Unless one of the three major components of ADHD (inattention, impulsivity, and hyperactivity) is addressed, there are unlikely to be any gains in educational performance because appropriate behaviors in these components are essential prerequisites for successful learning.

Multimodal Interventions

Following a review of a range of approaches to the treatment of ADHD, there is often a conclusion that the profusion of problems surrounding ADHD—both those unique to the individual and those associated with context (e.g., home, school) or significant others (e.g., parents, teachers)—mandates multimodal approaches to optimize therapeutic impact (Batsche & Knoff, 1994). Contradictory claims have been made about the effectiveness of multimodal interventions. For instance, Ervin et al. (1996) concluded that when treatment strategies are combined (in particular, stimulant medication and cognitive behavioral interventions), the outcomes are likely to be better than when one treatment strategy is used in isolation. Other studies, however, found that treatment combinations are not superior to stimulant medication alone (Abikoff & Gittelman, 1985; Cohen, Sullivan, Minde, Novak, & Helwig, 1981). Whalen and Henker (1991) reported a scarcity of research on the benefits of multimodal approaches. They concluded that documented evidence of the efficacy of multimodal treatments is difficult to find and that almost nothing is known about how treatments interact with each other. Moreover, Ervin et al. (1996) reported several methodological limitations of research into the effectiveness of multimodal approaches that relate to possible ceiling effects of medication, the suitability of measures used to detect change, and length of duration of interventions.

Other Meta-Analyses

Table 1 summarizes the results of seven meta-analyses that have reported effect sizes for pharmacological and behavioral/cognitive interventions on ADHD, although none of the meta-analyses reported effects for both types of intervention. Other meta-analyses of the treatment of ADHD do not report effect sizes (e.g., Jadad et al., 1999), and they have not been included in Table 1.

DuPaul and Eckert's (1997) meta-analysis of interventions focused only on nonpharmacological interventions. They used 63 articles published between 1966 and 1995 reporting studies that had used participants diagnosed as having ADD, ADHD, hyperkinetic impulse disorder, hyperactive deficits, or attention deficits. The intervention strategies included educational interventions, contingency management interventions, and cognitive behavioral interventions. They differentiated between within-subjects, between-subjects, and single-subject designs, although only the

TABLE 1
Summary of results from seven meta-analyses of ADHD interventions

Study	Publication date	Years of articles	No. of studies	No. of people	Average *n*	Type of intervention	Effect	
							Behavior	Academic
Baer & Nietzel	1991	1968–1989	36	1550	43	Cognitive & behavioral	.74	
DuPaul & Eckert	1997	1980–1995	63	637	10	School-based	.45	.31
Kavale	1982	<1982	135	5300	39	Stimulant medication	.80	.49
Losier, McGrath, & Klein	1996	1975–1992	15	479	30	Stimulant medication	1.59, .80	
Ottenbacher & Cooper	1983	Average 1975	61	1972	32	Pharmacological (mostly stimulant medication)	.96	.47
Silva, Munoz, & Alpert	1996	1972–1992	10	242	24	Carbamazepine	1.01	
Thurber & Walker	1983	<1983	20	1219	61	Stimulant medication	.75	.23

within-subjects designs were sufficiently rigorous and had sufficient data to be useful. The overall effect size was .64. The effects for educational interventions were .69, contingency management .94, and cognitive-behavioral .19. There were no differences between interventions in public (.83) and non-public schools (.77). Interventions implemented in special education classrooms were much greater (1.24) than those in general education classrooms (.49). The mean effect for academic measures was .31, and these were moderated by type of intervention: cognitive-behavioral (.46), educational (.20), and contingency management (.11). The authors do not specify the nature of the academic outcomes, and thus it is not clear whether the outcomes are related more to memory or to specific subject achievement tasks. DuPaul and Eckert concluded that school-based interventions for children with ADHD lead to significant behavioral effects regardless of the type of experimental design employed and that contingency management and educational interventions are more effective than cognitive-behavioral procedures in improving classroom behavior. Thus, although the meta-analysis by DuPaul and Eckert (1997) was comprehensive in its consideration of a range of treatments, it did not include a comparison of medically based and non–medically based treatments.

Kavale (1982) conducted a meta-analysis based on 135 stimulant medication outcome studies and reported an overall effect size of .58 for the effects of stimulant medication. The effects on behavioral outcomes were much greater (.80) than on cognitive outcomes (.50) and physiological outcomes (−.25). The cognitive effects were slightly greater for memory and copying (.41) and for drawing and copying (.47) than for intelligence (.39) and achievement (.38). The effect size for reading was .32 and for arithmetic .09. Kavale noted that the effects were larger when conducted by medical investigators, suggesting "possible vested interest in the outcomes" (p. 286).

The meta-analysis by Ottenbacher and Cooper (1983) also focused on the behavior and performance of hyperactive children who were given drug treatment (mostly methylphenidate). They reported large effects for behavior (.90) from their meta-analyses based on 61 studies. Drug therapy was found to reduce hyperactive behavior (.96) and to increase attention span (.84), but there was a smaller direct effect on improving overt academic performance (.47). Ottenbacher and Cooper noted major differences in the overall effects related to the research design. Drug-versus-control studies had higher effects (1.21) than drug-versus-placebo studies (.84). Ottenbacher and Cooper concluded that approximately 30% of the effect found in the drug-versus-control conditions could be attributed to the placebo phenomenon itself.

A meta-analysis of the effects of medication on hyperactivity by Thurber and Walker (1983) showed that the strongest drug-related improvements were on measures concerned with attention and distractibility (.75). There was a smaller improvement in school achievement (.23). They concluded that the stimulant medications mainly affected attentional control outcomes and were not sufficient for the amelioration of hyperactivity symptoms.

Silva, Munoz, and Alpert (1996) specifically evaluated the effects of Carbamazphione, a newer antidepressant medication, on ADHD children. In the 7 uncontrolled clinical studies reviewed, they reported only the range of effect sizes (.60 to 2.2) on behavior, and in the 3 double-blind, placebo-controlled studies they reported an average of 1.01. All but 1 study used observations rather than dependable scales to evaluate the behavior changes. Silva, Munoz, and Alpert note

that sedation and rash were the most common side effects, leading some children to drop out of the studies. They also report on 1 study in which the side effects were impulsivity, irritability, and aggression, thereby making it difficult "to differentiate medication effects from the treatment target symptoms when treating aggressive disorders" (p. 356).

Baer and Nietzel (1991) reported a meta-analysis of 36 cognitive and behavioral intervention studies designed to reduce impulsivity in children. Subjects all were described as impulsive, but variations in clinical diagnosis included ADHD, conduct disorder, behavior disorder, and learning disability. An overall effect on impulsivity of .77 was reported.

Losier, McGrath, and Klein (1996) systematically reviewed 15 studies in which the Continuous Performance Test (CPT) had been used to compare the omission and commission errors of ADHD children under placebo and methylphenidate conditions. A significant reduction in both types of errors was shown for the methylphenidate condition. For errors of omission, the effect was 1.59, and for errors of commission the effect was .80. Losier et al. noted the experimental inconsistency (such as interstimuli intervals, number of trials, time spent on task) associated with the use of the CPT, thereby calling into question the validity of the CPT as a tool to assess cognitive deficits in children with ADHD.

In a "review of reviews" that included meta-analyses spanning both pharmacological and nonpharmacological interventions, Jadad et al. (1999) did not report effect sizes, but they did conclude that "most published systematic reviews and meta-analyses on the treatment of ADHD have limited value for guiding clinical, policy, and research decisions" (p. 1025). They found only 2 studies that did not have extensive methodological flaws related to poor description of the methods used by the authors to identify, select, assess, and synthesize information; most researchers in the area had not kept abreast of recent methodological developments. The two reviews with minimal flaws indicated that methylphenidate significantly reduced the number of errors of commission and omission in children with ADHD and that school-based interventions led to behavioral gains but had less impact on academic and clinical test performance.

Summary

There have been many different interventions for the treatment of ADHD, but most have involved either the administration of drugs; a focus on changing the behavior or thought processes of the child, parent, and/or teacher; or some combination of these. It seems that the etiology of ADHD is confusing, with so many possible influences; thus, it is not surprising that there are many methods of diagnosis, many treatments, and much confusion over the use of the term ADHD. Once a child has been "labeled," however, drug therapy is the most common approach to intervention. Despite evidence of improvement in ADHD-related behaviors following medication, there is ongoing concern about the possible side effects, the lack of long-term benefit, and philosophical and logistical concerns that make these treatments unacceptable to some people (Ervin et al., 1996). Too often, the target behaviors are achievement, whereas the focus behaviors are attention or some form of behavioral control, which has led to many false claims that improvement in the latter behaviors leads to improvement in the former. The argument is that on-task, in-seat behaviors are necessary for achievement to occur, and once these behav-

iors are realized then achievement does occur. A major focus of this article is to assess the effects of the various treatments on both behavior and achievement.

Despite the abundance of research, debate persists over the comparative efficacy of the various treatments. In part, this is because of the lack of a recent meta-analysis that considers the complete range of interventions. Thus, the aim of the current meta-analysis is to bridge that gap and provide an overview of the full range of interventions for people with ADHD conducted in the 1990s.

Method

A search was made of the PsycLIT, Medline, Dissertation Abstracts, and ERIC databases. The search was limited to work in the English language, although no restriction was imposed with regard to publication type. Because of the large volume of literature on the topic, we limited the search to articles published from 1990 to 1998 (the year in which the meta-analysis was commenced). Key terms in the search included "ADD," "ADHD," "Attention Deficit Disorder," and "Attention Deficit Hyperactivity Disorder." Secondary sources were examined and citations were checked against studies located in the search of the databases. Some authors were contacted directly and unpublished work obtained. Criteria for including studies in the sample were that they reported the results of an intervention with people diagnosed with ADD or ADHD as the *primary* disorder. Diagnosis was judged to be acceptable if performed by a physician or psychologist. Three studies also were included in which diagnosis by a teacher was specified because it was reported either that subjects met DSM-III-R criteria for ADHD and that reports from teachers and parents had been considered, or that the Connors' Teacher Rating Scale-Revised had been used. Seven additional studies were included because, although the source of diagnosis was not specified, the method of diagnosis described was either the DSM-III-R or the DSM-IV. Additional criteria for selection of studies were that the type of intervention and outcome measures be sufficiently well described to enable classification and that it be possible to calculate an effect size. We were able to locate 74 unique studies (denoted by asterisks in the reference list) that met these criteria. Sixty-eight (92%) of the studies were published journal articles, and the remaining 6 were theses. Although the number of theses is small, it exceeds the number included in the seven meta-analyses summarized in Table 1 (five of these included no theses, one included 9 theses out of a total of 63 studies, and there was no information about the sources of publication in the other study).

Meta-analysis is a procedure for synthesizing findings across many studies, assessing the effects of various moderators, and ascertaining the major potential sources of variability in the program effects. Glass, McGaw, and Smith (1981) and Hedges and Olkin (1985), among many others, have presented standard texts on the methodology. The fundamental unit of analysis is the effect size, which is the difference between the mean of outcome measures at the end of the program and the mean prior to commencing the intervention (post–pre means) or the mean of the outcome measure administered in a treatment group compared to the mean of the outcome measure administered to a control group (treatment–control means). In all cases, this difference between the mean is divided by the appropriate pooled group standard deviation, and the sign of the difference is positive when the treatment has a positive effect. Becker (1988) provides details for estimating effect sizes from pretest and post-test data collection designs, using fixed, random, or mixed models, and

her methods are used in this study. We corrected the effect sizes for bias (as they overestimated the population effect size, particularly in small samples) by using Hedges' correction (Hedges & Olkin, 1985). Because the effect sizes within studies are correlated with each other, we used the weighted least squares approach. That is, the mean weighted effect size within each study was used as the unit of analysis (the weight is inversely proportional to the estimated sampling error in the effect size). The interpretation of effect sizes is based on Cohen's (1988) benchmark, where .20 is small, .50 is medium, and .80 is large (see also Hattie, 1992). Effect sizes of .20, .50, and .80 suggest that the score of the average person in the experimental group exceeds the scores, respectively, of 58%, 69%, and 79% of the control group.

To determine whether the overall effect sizes shared a common effect size (i.e., were consistent across the studies), a homogeneity statistic, Q_T, was calculated, which has an approximate chi-square distribution with k-1 degrees of freedom, where k is the number of studies (Hedges & Olkin, 1985). Given the large number of effect sizes that are combined into the various categories and the sensitivity of the chi-square statistic to this number, it is not surprising that nearly all homogeneity statistics are statistically significant. As with most meta-analyses, the interactions with other variables are often more informative than any overall mean.

Results

There were 1,497 effect sizes (n_e) derived from 74 studies (n_s), based on 2,193 persons (ranging from $n = 4$ to 161). Despite the major arguments about the distinctions between ADD and ADHD, the major classification (97%) was ADHD, primarily because ADHD is the current label preferred by DSM-IV. As expected, the majority of persons were males (85%). The number of persons per study was small ($x = 29$, range = 4 to 161), and thus this meta-analysis is based on an overall number of people far smaller than is typical in synthesizing studies. We note that Kavale (1982), in an earlier meta-analysis, reported an average of 39 participants per study. There was no correlation between the number of persons in the study and the study effect size ($r = .04$), and the correlation between the study effect size and year of publication (1990–1998) was .004. This indicates that the effects of interventions are not related to the sample size, and the effects have not changed over the past 10 years.

In these 74 studies we also located 160 effect sizes from 13 studies relating to control groups—that is, ADD/ADHD students who did *not* receive any medication or treatment. The mean effect size for these studies was –.007, indicating that they were truly control groups. There were 84 additional effects (not included in any of the following analyses) in which the participants were given a placebo (another form of control) rather than the treatment. The mean was –.11, indicating that the placebo actually decreased the performance that was assessed. There were 156 effects from 8 studies that related to follow-up investigations. The typical follow-up was 196 days (minimum = 14 days; maximum = 1 year). The average follow-up effect size was .01 ($SE = .001$). There were 88 effects for follow-up for the control groups with a mean effect size of –.01 ($SE = .002$). Clearly, whatever the effects of the treatments on these people, it was not maintained over time. (These effects were not included in the subsequent analyses.)

The overall weighted mean for the various interventions across the 74 studies relating to various outcomes was .48 ($SE = .07$). Figure 1 presents a frequency graph for all the effect sizes across the three groups. There were 248 effects from

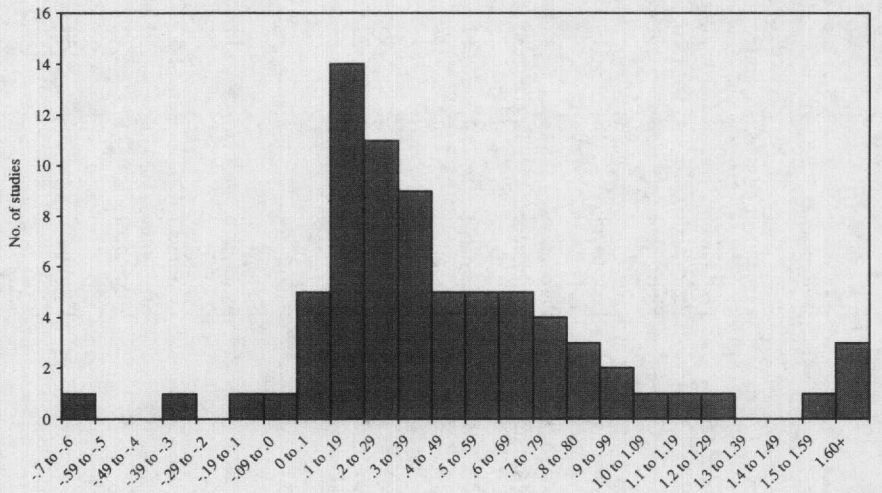

FIGURE 1. *Frequency graph of 1,497 effect sizes across normal, Tourette's, and "mentally retarded" samples.*

5 studies for people classified as being "mentally retarded" with an average weighted effect size of .46, and 108 effects from 4 studies for people with Tourette's syndrome with a mean of .82. Because these are special populations of students, they were excluded from all subsequent analyses. The remaining 1,141 effects were from 65 studies, with a mean of .42 (median = .30, $Q_T = 113$, $df = 64$, $p < .01$, $SE = .06$). Table 2 presents a summary of all articles, and it is noted that across all the studies, 5% were negative, indicating a 95% positive response to the interventions—a figure close to that reported by Kavale (1982) in a meta-analysis based on articles earlier than 1980.

Attributes of the Studies

The affiliation of the author appears not to be related to the effect sizes. Studies written by physicians ($n_s = 26$, $n_e = 568$) had an effect size of .48; for educators the effect size was .49 ($n_s = 4$, $n_e = 56$); for physicians and psychologists publishing together, .35 ($n_s = 17$, $n_e = 597$); for psychologists, .31 ($n_s = 9$, $n_e = 93$). The effect sizes also were not related to where the article was published. Articles published in medical journals ($n_s = 35$, $n_e = 912$) had an effect size of .47; in educational journals the effect size was .47 ($n_s = 5$, $n_e = 52$); in psychological journals it was .30 ($n_s = 17$, $n_e = 380$); in psycho-educational journals it was .36 ($n_s = 8$, $n_e = 153$).

Effect sizes relating to the major thrust of the article were similar, except for 1 study in which there was a combined neurological and behavioral thrust (.11, $n_s = 1$, $n_e = 27$). Articles with a social skills emphasis had an effect size of .42 ($n_s = 4$, $n_e = 71$); with a neurological emphasis, .42 ($n_s = 43$, $n_e = 1179$); with a behavioral emphasis, .36 ($n_s = 7$, $n_e = 99$); with a self-regulation emphasis, .32 ($n_s = 2$, $n_e = 55$); with a psycho-educational emphasis, .35 ($n_s = 6$, $n_e = 66$).

The design of the studies led to major differences. When the design was ABA, the effect was .45 ($n_s = 31$, $n_e = 383$), and for crossover it was .38 ($n_s = 30$, $n_e = 733$),

TABLE 2
Summary of studies included in the meta-analysis

Authors	Year	Affiliation	No.	% Male	Age	Secondary classif.	Design	Days	Outcome*	Treatment	No. effects	Hedges' effect
Alston & Romney	92	Psych	60	100	Elem	Normal	Tr-Ctl	1	5	Pharmacol	8	−.11
Aman & Turbott	91	Med	26	85	Elem	Normal	ABA	21	2,3,4,5	Pharmacol	4	1.16
Aman et al.	93a	Med & Psych	28	71	Elem	Mretard	Xover		3	Pharmacol	58	.31
Aman et al.	93b	Med & Psych	28	71	Elem	Mretard	Xover	28	1,3	Pharmacol	18	.10
Anastapoulos et al.	93	Other	34	74	Elem	Normal	ABA	9	1,3,4,5	Parent	11	.18
Balthazor et al.	91	Med & Psych	19	100	Elem	Normal	Xover	4	3	Pharmacol	12	.30
Barrickman et al.	91	Med	19	84	Mixed	Normal	ABA	42	1,2,3,5	Pharmacol	9	.17
Bloomquist et al.	91	Med & Psych	52	69	Elem	Normal	ABA	10	3,4,5	School	36	.22
Brewer	96	Med	18	94	Elem	Normal	ABA		1	Pharmacol	1	.13
Buhrmester et al.	92	Other	32	100	Elem	Normal	Xover	21	2,3,4	Pharmacol	8	−.33
Carlson et al.	91	Med & Psych	13	100	Elem	Normal	Xover	2	1,3	Pharmacol	19	.55
Carlson et al.	92	Med & Psych	24	100	Elem	Normal	Xover	24	1,3	Pharmacol + Behavioral	27	.11
Carlson et al.	93	Med & Psych	26	100	Elem	Normal	Xover		1,3,5	Pharmacol	30	.17
Chappell et al.	95	Other	10	80	Mixed	Tourette's	ABA	140	1,3,5	Pharmacol	5	1.22
Cocciarella et al.	95	Psych	7	71	Mixed	Normal	ABA	7	3	Non-school	8	.29
Conners et al.	96	Med & Psych	109	92	Elem	Normal	Tr-Ctl	35	3	Pharmacol	1	.12
Douglas et al.	95	Med & Psych	17	94	Elem	Normal	Xover	8	1,3	Pharmacol	54	.39
Dunson et al.	94	Psych	20	65	Elem	Normal	ABA	25	3,4	School	6	.62

DuPaul & Rapport	93	Psych	56	82	Elem	Normal	Xover		3,5	Pharmacol	12	.05
DuPaul et al.	96	Other	24	79	Mixed	Normal	Tr-Ctl	28	2,3,5	Pharmacol	57	.22
Evans & Pelham	91	Med	9	100	Middle	Normal	Xover	30	1,3	Pharmacol	18	.52
Evans et al.	95	Med	16	na	Mixed	Normal	ABA	40	1,3	School	21	.25
Fehlings et al.	91	Other	25	100	Elem	Normal	ABA	120	1,3,5	Non-school	8	.23
Fischer & Newby	91	Med	161	88	Mixed	Normal	Xover	21	1,3	Pharmacol	44	.38
Fitzpatrick et al.	92	Med & Psych	19	89	Elem	Normal	Xover	56	2,3	Pharmacol	51	1.05
Folk	94	Psych	26	46	Elem	Normal	ABA		3	Non-school	3	.59
Forness et al.	92	Med & Psych	42	100	Elem	Normal	Tr-Ctl		1	Pharmacol	14	.16
Gadow, Paolicelli, et al.	92	Med	43	na	Elem	Normal	Xover		3,4	Pharmacol	20	.01
Gadow et al.	95	Med & Psych	34	91	Elem	Tourette's	Xover	56	3,4,5	Pharmacol	39	.54
Gadow et al.	90	Med & Psych	11	100	Elem	Normal	Xover	42	3,4	Pharmacol	32	.45
Gadow, Nolan, et al.	92	Med	11	na	Elem	Tourette's	Xover	56	3,4,5	Pharmacol	63	.75
Gammon & Brown	93	Med & Psych	32	94	Mixed	Normal	ABA	84	1,2,3,5	Pharmacol	9	.15
Goldhaber	91	Med	8	na	Mixed	Normal	ABA	40	3,4	Non-school	5	1.57
Gordon et al.	91	Med & Psych	6	50	Elem	Normal	ABA	13	3	Pharmacol + Behavioral	1	.38
Grizenko	93	Med	30	77	Elem	Normal	ABA		1,3,4,5	Pharmacol + Behavioral	13	.23
Gualtieri et al.	91	Med	12	92	Elem	Normal	Xover		2,3	Pharmacol	27	.99
Handen et al.	92	Med & Psych	14	71	Elem	Mretard	Xover	6	1,3,4	Pharmacol	94	.65
Handen et al.	90	Med	12	92	Elem	Mretard	Xover	21	1,3,4	Pharmacol	58	.84
Handen et al.	96	Med & Psych	44	61	Elem	Mretard	ABA	21	1,3	Pharmacol	20	.61

(*continued*)

TABLE 2
Summary of studies included in the meta-analysis (Continued)

Authors	Year	Affiliation	No.	% Male	Age	Secondary classif.	Design	Days	Outcome*	Treatment	No. effects	Hedges' effect
Harbeitner	96	Educat	9	67	Elem	Normal	ABA	75	1,2,3,45	Non-school	27	.30
Hedges et al.	95	Med	18	67	Adult	Normal	ABA	56	1,2,3,5	Pharmacol	12	.20
Hinshaw et al.	92	Other	44	100	Elem	Normal	Xover	2	3	Pharmacol	3	.21
Horn et al.	90	Other	60	70	Elem	Normal	ABA	84	1,3,5	Parent	42	.11
Horrigan & Barnhill	95	Med	15	100	Mixed	Normal	ABA	50	3	Pharmacol	3	2.18
Kaduson & Finnerty	95	Educat	63	92	Elem	Normal	ABA	10	3,4	Non-school	13	.57
Kaplan et al.	90	Med & Psych	6	100	Middle	Normal	Xover	98	3	Pharmacol	6	.31
Keith & Engineer	91	Other	20	85	Elem	Normal	ABA	1	1,3	Pharmacol	28	.61
Kent et al.	95	Med	12	92	Elem	Normal	Xover	12	2,3,4	Pharmacol	20	.39
Klein	91	Med	77	na	Elem	Normal	ABA	84	1,2,3	Pharmacol	26	.12
Klorman et al.	90	Med & Psych	48	88	Mixed	Normal	Xover	21	2,3,4,5	Pharmacol	23	.31
Kolko et al.	90	Med	56	68	Mixed	Normal	ABA	15	3,4	Non-school	13	.44
Long et al.	93	Med	32	81	Elem	Normal	Tr-Ctl	60	1,3	Parent	7	.87
Malhotra & Santosh	98	Med	12	83	Elem	Normal	ABA	42	3	Pharmacol	6	.26
Malone & Swanson	93	Med	25	na	Elem	Normal	Tr-Ctl	2	3	Pharmacol	7	.32
Matier et al.	92	Med & Psych	38	87	Elem	Normal	ABA	1	3	Pharmacol	8	.28
Mazius	90	Educat	4	75	Elem	Normal	ABA	12	1,3,5	School	10	.77
Musten et al.	97	Med & Psych	31	84	Pre-school	Normal	Xover	30	1,3	Pharmacol	28	.40

Pelham et al.	91	Med	34	100	Middle	Normal	Xover	22	1,3	Pharmacol + Behavioral	32	.47
Pelham et al.	90	Med	22	100	Elem	Normal	Tr-Ctl	45	1,3,4	Pharmacol	52	.47
Pelham et al.	93	Psych	31	100	Elem	Normal	Xover	30	3,4	Pharmacol	40	.92
Pelham et al.	95	Med & Psych	28	79	Elem	Normal	Xover	49	1,3	Pharmacol	16	.78
Pfiffner & McBurnett	97	Med	27	70	Elem	Normal	ABA	8	3,4	Non-school	26	.19
Pisterman et al.	92	Psych	55	91	Mixed	Normal	ABA		3	Parent	6	.04
Poley	95	Psych	26	88	Elem	Normal	Tr-Ctl	8	3	Non-school	6	.27
Rapport et al.	93	Med	16	100	Elem	Normal	ABA	112	1,3	Pharmacol	6	.83
Risser & Bowers	93	Psych	20	75	Mixed	Normal	Tr-Ctl		1	Pharmacol	4	−.69
Solanto et al.	97	Med	22	86	Elem	Normal	Xover	4	3	School	40	.18
Spencer, Beiderman, Kerman, et al.	93	Med	33	100	Mixed	Tourette's	ABA	480	3	Pharmacol	1	2.62
Spencer, Beiderman, Wilens, et al.	93	Med & Psych	12	91	Mixed	Tourette's	ABA	570	3	Pharmacol	1	3.11
Stein et al.	96	Med	25	100	Elem	Normal	Xover		1,3,4,5	Pharmacol	33	.62
Trommer et al.	91	Other	25	84	Middle	Normal	Xover		3	Pharmacol	1	.76
Weber et al.	92	Other	44	68	Mixed	Normal	ABA	730	1	Pharmacol	20	−.02
Wender & Reimherr	90	Med	14	na	Adult	Normal	ABA	56	3	Pharmacol	1	.28
Williams	90	Educat	12	75	Elem	Normal	Xover	9	1,3	Non-school	6	.03

Note. Psych = Psychology; Med = Medicine; Educat = Education; Elem = Elementary School; Middle = Middle School; Mretard = Mentally Retarded; Xover = Crossover; Tr-Ctl = Treatment-Control.

*1 = cognitive, 2 = physical, 3 = behavioral, 4 = social, 5 = personal.

whereas for treatment–control it was .09 ($n_s = 4$, $n_e = 25$). Carlson and Schmidt (1999) posited two possible reasons for these differences. First, treatment–control designs tend to underestimate effect sizes when post-treatment standard deviations are used. This is because post-treatment standard deviations are often larger than pre-test standard deviations, primarily because post-treatment standard deviations may be inflated by interactions of the subjects with the treatment. That is, students may be influenced by the ADHD treatments in variable ways, with the result of "more variable scores on the posttraining dependent variable measure than would have been observed during the pretraining assessment" (p. 853). In the current study such variability was not observed, as the pre-test standard deviation was 9.55 and the post-test standard deviation was 9.12 (a 2% change). Second, pre–post test designs have been criticized because they lack controls for various extraneous effects, whereas the use of control groups permits any nontraining effects on the dependent variable to be captured and removed during the calculation of effect sizes (leading to the hypothesis that the control effect sizes will be positive). In the current study, the post–pretest effect size for the control groups was –.11 (as noted above) and thus cannot be a reason for the larger effects from the various designs. We note that the mean effect for the ABA studies (which involved over-time comparisons) was similar to the mean effect for the cross-over designs (which involved comparison groups); only one type of comparative design (the control-experimental) led to lower mean effects. Furthermore, we tested the homogeneity of these effect-sizes, and the between-group homogeneity statistic indicated that there were no statistically significant differences between the three means ($H_B = 1.64$, $df = 2$, $p > .05$).

A more plausible reason for the lower treatment–control effect relates to specific attributes of the studies. For instance, one of the 4 relevant studies (Risser & Bowers, 1993), was concerned with neuropsychological functioning as assessed by an EEG, and the effect size was –.69. This was in contrast to the average effect of .35 for the other 3 studies in that group. Thus, it may be less the nature of the design than the nature of the outcome that accounts for the observed differences.

Attributes of the Sample

The effects were greater for the student than for adult age groups. For kindergarten-aged children ($n_s = 1$, $n_e = 28$) the average effect was .40; for elementary school students it was .37 ($n_s = 44$, $n_e = 1{,}166$); for middle school students it was .57 ($n_s = 4$, $n_e = 61$); and for adults it was .25 ($n_s = 2$. $n_e = 13$). Only a few studies provided information about the socioeconomic background of the sample: for low-SES students the effect size was .34 ($n_s = 3$, $n_e = 32$); for middle-SES students it was .35 ($n_s = 9$, $n_e = 322$); for upper-SES students it was .15 ($n_s = 1$, $n_e = 9$). Similarly, too few studies reported on the ethnicity of their sample, most claiming that they were "mixed" groups.

Attributes of the Diagnosis

The major source of diagnosis was physicians (.46, $n_s = 44$, $n_e = 815$), and the effects were substantially greater than when psychologists (.28, $n_s = 14$, $n_e = 24$) or teachers (.30, $n_s = 3$, $n_e = 24$) made the diagnosis. Six major methods of diagnosis were used to classify the people diagnosed with ADHD. The largest group was diagnosed through the use of the DSM-IV in conjunction with an unspecified battery of tests, and the overall effect was .38 ($n_s = 30$, $n_e = 996$); in an additional 5 studies ($n_e = 33$), diagnosis was achieved through the use of an (unspecified) bat-

tery of tests (effect = .35); the Conners was rarely used alone (effect = .23, $n_s = 3$, $n_e = 20$); the DICA (Diagnostic Instrument for Childhood and Adolescence) was used in 1 study ($n_e = 51$, effect = 1.05); and when the DSM was used alone the effect was .45 ($n_s = 22$, $n_e = 278$). The effects were highest when the diagnostic scale was a structured interview ($n_s = 2$, $n_e = 105$, effect = .72), followed by a single test ($n_s = 6$, $n_e = 120$, effect = .41); a checklist ($n_s = 27$, $n_e = 277$, effect = .41), and a battery of tests ($n_s = 30$, $n_e = 1000$, effect = .37).

Attributes of the Treatment

The average duration of the treatment was 58 days ($SD = 120$, $n = 70$ studies that reported this information). There was a significant relationship between the length of treatment and the effect size ($r = .44$). The majority of treatments were managed by physicians with an effect of .41 ($n_s = 43$, $n_e = 1{,}156$). Similar effects were found when treatments were managed by psychologists (effect = .35, $n_s = 11$, $n_e = 221$); by teachers (effect = .47, $n_s = 3$, $n_e = 42$); or by parents (.39 $n_s = 2$, $n_e = 8$). The effects were lower when physicians and psychologists collectively managed the treatment (effect = .24, $n_s = 3$, $n_e = 51$). The effects were greater when managed in the home (effect =. 47, $n_s = 25$, $n_e = 618$) than when managed in clinics (effect = .36, $n_s = 24$, $n_e = 534$) or in the school (effect = .33, $n_s = 13$, $n_e = 330$).

The various treatments were classified into five major types (pharmacological, school-based psychological/educational, non-school-based psychological, parent training, and multimodal; see Table 3). The effects of antidepressants were large

TABLE 3
Number of effects and effect sizes (ES) across all outcomes relating to the type of treatment

	Weighted by study			Unweighted least squares		
Intervention	n_s	*ES*	*SE*	n_e	*ES*	*SE*
Pharmacological	42	.45	.09	750	.44	.02
Stimulants	31	.35	.07	648	.43	.02
Antidepressants	8	.85	.35	60	.69	.24
Antipsychotic drugs	1	.12		26	.12	.01
Stimulants and antidepressants	3	.43	.21	16	.42	.09
School-based psychological/educational	8	.39	.09	92	.32	.03
Cognitive/self-regulation	2	.49	.27	46	.34	.04
Behavioral training	3	.50	.12	7	.58	.12
Educational	3	.29	.16	30	.24	.05
Other	1	.25		9	.25	.13
Non-school-based psychological	10	.39	.14	140	.29	.03
Cognitive/self-regulation	5	.58	.27	37	.42	.11
Social skills training	3	.31	.07	66	.28	.03
Behavioral training	1	.29		8	.29	.01
Biofeedback	1	.50		9	.50	.18
Other	1	.18		20	−.03	.05
Parent training	4	.31	.19	38	.27	.05
Multimodal	7	.28	.13	121	.37	.03

Note. n_s = number of studies, n_e = number of effects.

and positive. These positive effects were particularly large for behavioral outcomes (effect = 1.58). There were also negative effects of antidepressants on physical outcomes (effect = −.44). The most common stimulant was methylphenidate ($n_s = 31$), and the dosage was primarily between .08 and 1.00mg/kg. The correlation between the ranks of the mean dosage and overall effect sizes was -.09. Higher doses of methylphenidate (greater than .50 mg/kg) were associated with larger effects for attention, but the effects on impulsivity and hyperactivity were similar for high and low dosages. Psychologically based interventions ($n_s = 17$, $n_e = 232$), whether school-based or not, and multimodal interventions ($n_s = 2$, $n_e = 121$) had similar effects. Parent training uniformly had the lowest effect across all types of outcomes ($n_s = 4$, $n_e = 38$).

The Outcomes

The outcomes were divided into five types: cognitive, physical, behavioral, social, and personal/emotional (Table 4). Overall, the effects were largest for behav-

TABLE 4
Effect sizes (ES) for the various outcomes by number of studies and by number of effects

	Weighted by study			Unweighted least squares		
Outcome	n_s	*ES*	*SE*	n_e	*ES*	*SE*
Cognitive	38	.28	.05	266	.28	.03
General cognition	17	.35	.10	72	.37	.05
Language/reading	12	.20	.04	82	.23	.03
Math	10	−.01	.07	34	.00	.04
IQ	2	.15	.02	8	.13	.06
Memory	2	.68	.26	21	.64	.25
Physical	12	−.03	.11	46	−.11	.14
Behavioral	54	.56	.09	677	.50	.02
Better behavior	37	.66	.13	239	.65	.04
Impulsivity	29	.45	.10	91	.35	.08
Lower impulsivity	19	.48	.15	59	.47	.09
Lower disruption	12	.36	.07	32	.34	.06
Hyperactivity	28	.68	.13	110	.57	.05
Less hyperactivity	25	.72	.14	69	.72	.06
Less aggression	7	.45	.13	41	.36	.04
Attention	43	.32	.09	257	.32	.03
Greater attention	.37	.36	.10	191	.31	.04
More on task seatwork	3	.36	.08	20	.39	.04
Greater compliance	11	.40	.20	46	.44	.06
Social	18	.38	.10	85	.36	.03
Personal/emotional	14	.22	.03	67	.21	.26
Lower depression	5	.17	.08	8	.20	.09
General emotion	7	.26	.03	19	.26	.04
Higher self-esteem	7	.13	.04	30	.15	.05
Efficacy/attribution	5	.21	.14	29	.24	.06
Less anxiety	5	.23	.05	10	.27	.03

Note. n_s = number of studies, n_e = number of effects.

ioral outcomes (.56), followed by social (.38), cognitive (.28), and personal/emotional (.22) outcomes. There were zero effects for physical outcomes (−.03), although, as described below, there was great variability in physical outcomes depending on who made the assessment.

The behavioral effects were further divided into three categories: impulsivity, hyperactivity, and attention. In addition, a large number of effects could be classified only under the term "better behavior." The effects across all behavioral categories were systematically among the largest. The treatments reduced the negative behaviors of these students (for example, the students demonstrated less hyperactivity and impulsivity and were more compliant). The effects across the social outcomes averaged .38 and were primarily related to enhanced liking or a reduced disliking by peers, better peer interactions and prosocial skills, and general demonstration of appropriate social skills. Overall, there is no evidence to support the claim that these treatments have a marked effect on the personal or emotional outcomes (.22). All the physical effects were associated with pharmacological treatments, primarily stimulants (66% of the treatments were methylphenidate). The negative effects related to side effects such as a reduction in fine motor speed and skills, weight loss, sleep patterns, an increase in nausea, and shakiness.

The interaction of treatment and outcomes (Figure 2) indicates that pharmacological and multimodal treatments have the greatest effects on behavioral outcomes, and multimodal treatments have the greatest effects on social outcomes. There was far less variability in the effectiveness of interventions on cognitive outcomes, slightly favoring school-based treatments. There were no differences in the treatments on personal and emotional outcomes.

For each of the three behavioral subcategories it can be seen that the pharmacological treatments were effective (Figure 3). The non-school-based programs

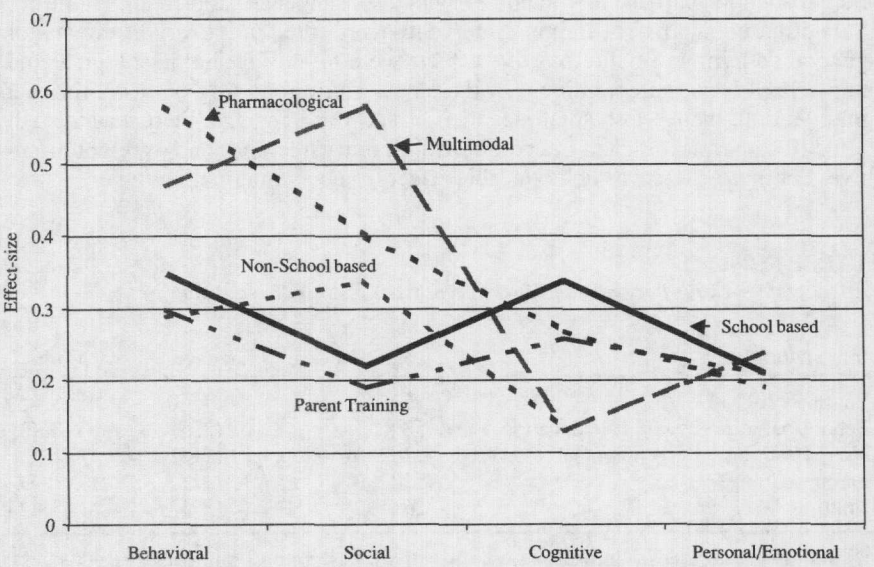

FIGURE 2. *Mean effect sizes for four outcome types by type of intervention.*

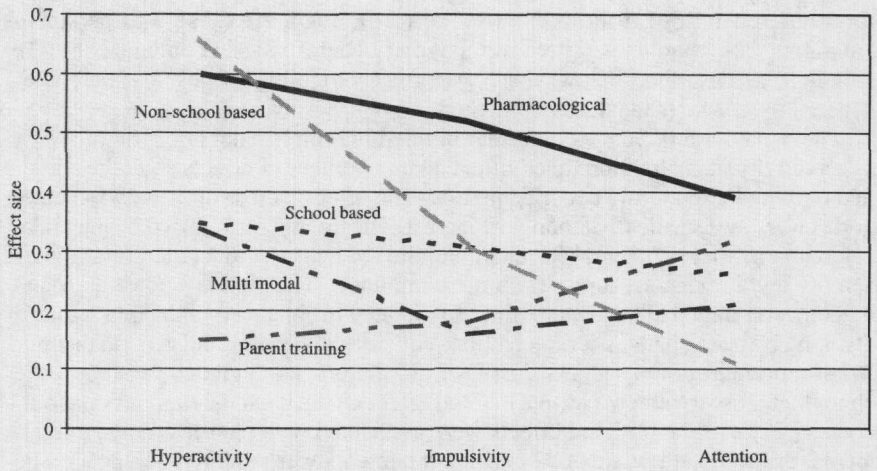

FIGURE 3. *Mean effect sizes for the three behavioral outcomes by the five intervention types.*

were as effective for hyperactivity as were the pharmacological treatments, but not for impulsivity and attention. The school-based and multimodal programs were generally less effective across the three behavioral outcomes. Parent training was not effective for hyperactivity or impulsivity, although there was a small effect for attention.

We were particularly interested in the effects of interventions on cognitive outcomes, so we divided these outcomes into general cognition (including nonspecified academic performance, memory, and IQ), language, and math (Table 5). School-based and parent-training interventions resulted in the largest effects for general cognition. Smaller positive effects were found for pharmacological and non-school-based interventions. In the single study reporting on a multimodal intervention, there was a small negative effect. For language, there was a small effect for pharmacological interventions, but other interventions were not effective. For math, there was no noticeable effect from any intervention type.

TABLE 5
Effect of the various interventions on different types of cognitive outcomes

Intervention	General cognition	Language	Math
Pharmacological	.36 (14, 79)[a]	.24 (7, 60)	.01 (5, 15)
School-based psychological/educational	.58 (2, 14)	.02 (2, 5)	.04 (2, 6)
Non-school-based psychological	.22 (2, 3)	<–.01 (1, 2)	.17 (1, 1)
Parent training	.53 (2, 3)	.12 (1, 2)	.12 (1, 1)
Multimodal	–.26 (1, 2)	.14 (5, 13)	–.11 (5, 11)

[a]Numbers in parentheses indicate n_s (number of studies) and n_e (number of effect sizes), respectively.

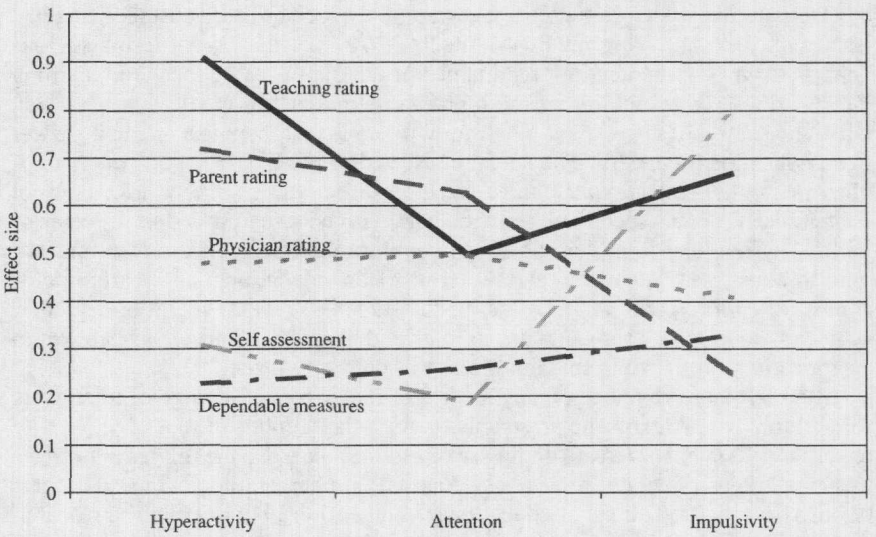

FIGURE 4. *Mean effect sizes for the three behavioral outcomes moderated by who administered the outcome measure.*

The effects on the outcomes were different depending on who made the outcome assessment. Largest effects were for teacher assessment (effect = .60, n_s = 13, n_e = 315). Other effects were markedly lower. When dependable scales were used (i.e., published scales administered by an independent, trained person), the effect was .33 (n_s =30, n_e = 524); for physicians or counsellors it was .31 (n_s = 6, n_e = 42); for parents it was .39 (n_s = 10, n_e = 156); and when self-assessed by the student the effect was .42 (n_s = 6, n_e = 104). Self-assessments of cognition were much greater (.63) than assessments based on dependable measures (.32) and teacher assessments (.19). For the behavioral outcomes, the effects were similar for dependable measures (.47), physician ratings (.40), parent ratings (.48), and self-assessment (.47), whereas, by comparison, teachers saw marked improvement (.89). There were no differences in social outcomes relating to who made the outcome assessment. Assessment of physical outcomes varied enormously. Dependable assessments of physical outcomes were large and negative (−.91), whereas teachers rated the physical outcomes as large and positive (1.92). Physician ratings for physical outcomes were zero. Most of the dependable measures assessed heart rate, blood pressure, and weight loss outcomes; teacher ratings related to sensorimotor skills, and the physician ratings related to sleep and general health outcomes.

Figure 4 shows that on hyperactivity and attention outcomes, the teacher, parent, and physician ratings were much higher than the self-assessments and dependable measures. For impulsivity, the self-assessments and teacher ratings greatly exceeded the physician and parent ratings and the dependable measures.

Discussion and Conclusions

Most research on ADHD has been conducted from within a medical framework and has focused on the effects of pharmacological treatments on behaviors. That

children are hyperactive, inattentive, or impulsive seems to be cast as a medical issue typically deserving medical remedies. We note the use of terms such as "pharmacological/medication treatments" or "therapy," as opposed to "learning interventions" as symptoms of the literature being cast in medical terms. Even when the setting of interest is a classroom, it seems that there is a medical answer to an educational concern. The social construction of ADHD has for many children meant that educational solutions to their difficulties at school are either not contemplated or take second place to medication. The research for this article is of particular significance in that it aimed to explore the effects of interventions not only in terms of behavior but also in terms of the educational and learning outcomes for children with ADHD (that is, the students' general cognition and their achievement in school subjects such as math and language). Thus, it is from within an educational framework that this study was conceptualized.

Although there have been many studies of the effects of a range of treatments for children with ADHD, the present meta-analysis is unique in several ways. The meta-analysis synthesized the findings from studies that span the full range of treatments, whereas previous studies have focused on pharmacological interventions or behavioral interventions or educational interventions, but not all. The research reviewed in this meta-analysis was conducted recently (1990–1998) and reflects the changes that have occurred in the last decade, particularly in terms of the increased frequency of diagnosis of ADHD.

The current meta-analysis supports the common claim that more boys than girls are diagnosed with ADHD (85%) and that the common label is now ADHD rather than ADD, primarily because the former is more inclusive. There are many cases, however, of comorbidity, and we noted much higher effects for persons also classified as having Tourette's syndrome or as intellectually disabled ("mentally retarded").

Overall, there were 1,497 effect sizes derived from 74 studies, based on 2,193 persons. The average treatment was 58 days, and the mean for the various interventions for persons with ADHD relating to various outcomes was .48, or .42 when the intellectually disabled and Tourette's syndrome samples were excluded. There were many moderators to this overall conclusion indicating much variability in the effects. This implies a clear message about the importance of making specific conclusions relating to ADHD programs and outcome effects.

This meta-analysis included studies that investigated the effects of a broad range of treatments on a range of outcome types. The overwhelming majority of interventions were pharmacological, and the principal focus of these interventions was on behavioral outcomes. The overall effect of medication on behavior was .58, which is lower than the average of .98 in Table 1. This could be because we have included more recent studies (although the correlation between year of study and outcomes was zero); because the earlier meta-analyses were concerned more specifically with children with hyperactivity as opposed to those specifically diagnosed with ADHD (thereby including people with a greater range of behavioral dysfunction); because many of the more recent studies used more dependable measures compared to an earlier dominance of general rating scales; and/or because the estimations of the effect sizes in earlier studies were inflated.

Very few studies ($n = 8$) assessed the effects of school-based interventions on children with ADHD. It is important that, from an educational perspective, there

were few studies that considered the effects of any sort of intervention on cognitive outcomes, particularly those related to academic achievement as assessed by "real-life" classroom tasks. The overall effect on cognitive outcomes was .28, which is lower than the average of .37 in Table 1 but similar to the average effect of .31 reported by DuPaul and Eckert (1997) in their meta-analysis of school-based programs. The reason for these lower effects is primarily that many of the previous studies focused on nonspecific cognitive outcomes, such as memory and IQ, rather than on in-class academic achievement, including achievement on math and language tasks. The greatest effect on cognitive outcomes was from school-based programs (.58), and this average effect was greater than that obtained from pharmacological interventions (.36). Notably, the cognitive effects from pharmacological interventions derived mostly from non-subject-specific memory tasks, whereas those from school-based interventions derived mostly from measures of general problem-solving ability.

A number of design issues need to be taken into account in interpreting the findings of both the current and previous meta-analyses of ADHD interventions. In our examination of the studies included in our meta-analysis it became obvious that many were based on very small sample sizes, and most reported statistically significant findings. As is evident in the majority of meta-analysis studies, treatment conditions that are published are biased toward a positive outcome (Lipsey & Wilson, 1993). In this respect, our findings are no different, in that the average effect size for the 6 unpublished studies was .35, as compared with the overall effect size of .42. This implies that the effects we report may be an *overestimation* of the true effect sizes. In addition, some published studies reported only results that were statistically significant. For instance, in one of the studies included in the current meta-analysis, it was reported that "[a]lthough a total of 54 variables was statistically analysed, only those with small p-values (<.05) will be reported" (Gualtieri, Keenan, & Chandler, 1991, p. 157). This led the authors to report on only 9 of the 54 variables! Further, there were many studies with data that were not useable in this meta-analysis, as they reported statistics such as percent increase, percentiles, or raw score gain.

A major finding was related to the importance of the outcome measures. Outcomes based on ratings by teachers and parents far exceeded ratings by self or by dependable scales (which included dependable scales administered by teachers and parents). This could be related to teachers and parents having more daily interactions with the ADHD students, and thus reports of the increased effects could be more dependable. Such a relationship is difficult to ascertain, however, because when teachers and parents used dependable scales they saw lesser effects. Certainly, the common argument that there are flow-over effects of the reduced behavior problems may arise from these increased ratings by teachers and parents. Perhaps any reduction in behavior problems could be generalized to an overall increase in educational outcomes—an oft-made conclusion—but this conclusion is not supported by the evidence presented in the studies reported in this meta-analysis. We strongly recommend that all studies involving ADHD outcomes (using whatever method of intervention) pay particular attention to the use of dependable measures.

Effects sizes obtained from the current meta-analysis need to be compared to the average effect sizes of the influences of instructional method (.47), learning

strategies (.61), and teacher background (.44), based on 225 meta-analyses of effects on the outcomes of schooling (Hattie, 1992). Overall effect sizes for pharmacological (.42), school-based (.39), and non-school-based (.37) interventions are not remarkable when considered in this light. We also note the overall effects found by DuPaul and Eckert (1997) of .69 for educational interventions and .94 for contingency management as being greater than that found by Hattie (1992) and Hattie, Biggs, and Purdie (1996). There are many educational innovations that affect achievement, far beyond medication and other treatments implemented for children with ADHD.

There is no doubt that medication can have major effects on the behavior of ADHD students. When one considers that one of the major purposes of schooling is improved learning and that effective learning is dependent on the presence of behaviors that help students to focus on learning tasks, it is reasonable to hope that children with ADHD will be helped in this respect by medication that decreases pathological learning behaviors such as impulsivity, hyperactivity, and inattention. However, the present findings do not indicate that such flow-over effects to learning or achievement occur. When children with ADHD are given medication, there is only a small improvement in their general cognitive abilities—less than when they participate in a school-based intervention, although these findings need to be interpreted with caution because of the small number of studies that have reported either on the effects of school-based interventions or on the effects of any sort of intervention on cognitive outcomes. Certainly, the improved behavior of children with ADHD has benefits for teachers and for the parents of these children, but for the children themselves the benefits appear to be limited to improved social functioning. There does not appear to be an improvement in emotional well-being or school-based achievement. Clearly, if educational outcomes are to be enhanced for ADHD students, then educational rather than medical answers need to be provided. Given the oft-cited importance of self-efficacy leading to higher achievement outcomes, the effects on self-efficacy are particularly low. This may explain in part why there seem to be few follow-through effects from behavior to achievement.

The side effects of medication cannot be attractive to ADHD students, their teachers, or their parents. Typical of the side effects are those reported by Hedges, Frederick, Reimherr, Rogers, Strong, and Wender (1995), who found that 39% of individuals had significant problems with side effects, particularly fatigue and confusion, and had difficulty staying on their medication. Fifty percent of individuals in the Hedges et al. study experienced nausea, and 17% experienced lowered energy, gas, diarrhea or pain, insomnia, tremor, muscular tension or teeth grinding. There have even been reports of death resulting from the pharmacological treatment of people with ADHD (Varley & McClellan, 1997). In the current meta-analysis, negative physical side effects of medication were reported in 7 studies (individual negative effects ranged from –.01 to –4.08), and these effects were related to negative effects on fine motor skills, weight, appetite, blood pressure, heart rate, and sleep patterns. It is possible that the overall negative effect was greater because some studies did not provide statistics for calculating effect sizes, simply reporting that as well as the positive outcomes on behavior there were side effects of medication.

Although there are positive effects of medication on the behavioral outcomes of people with ADHD, few long-term outcomes have been reported. When they are reported, they generally do not indicate significant long-term improvements

(DuPaul & Barkley, 1998). Williams et al. (1999) noted that medicated ADHD children are no more likely to attend university than nonmedicated ADHD children; nor are they less likely to become delinquent than their nonmedicated ADHD peers. One possible explanation for the weak impact of ADHD interventions on students' academic achievement may lie in the simple pre- and post-test designs used in most of the studies included in this meta-analysis. Willett, Singer, and Martin (1998) have shown in numerous publications in the last decade that "to measure individual change well, a truly longitudinal perspective must be adopted—a sample of people must be followed over time allowing the researcher to collect multiple waves of data at sensibly spaced intervals" (p. 397). Thus, to improve the precision of estimated treatment effects, future ADHD intervention studies must incorporate a longitudinal component into their design, preferably using recent methodological advancements such as individual growth modeling techniques (e.g., see Singer, 1998; Willett et al., 1998) that are based on multiple waves of data, rather than on the single-wave or two-wave data sets commonly used in ADHD research.

Although medication can reduce behavior problems and improve memory, this does not confirm a diagnosis of ADHD. Many authors have noted that there can be similar effects of medication on the activity, memory, and vigilance of students who are not diagnosed with ADHD (see Whalen & Henker, 1991, for a discussion of this issue). This is particularly important for ADHD because there are no laboratory or radiological confirmatory tests and no associated physical features; moreover, the diagnostic criteria have changed frequently, and the rates can differ dramatically across locations as well as across countries. Hence there is a tendency to assume that because some drug treatment can reduce the "symptoms" there is evidence of the existence of ADHD (see Jadad et al., 1999).

If the behavioral manifestations of ADHD are not conducive to learning and if the administration of stimulants such as methylphenidate, dextroamphetamine, or pemoline help children to have better attending behaviors and to be less distractible or hyperactive, then pharmacological intervention may be warranted as a first step. It is important, however, to not merely prescribe medication if enhanced educational outcomes is the aim. To enhance educational outcomes, sustained and deliberate attempts at educational interventions are necessary, and it is most likely that those educational programs and interventions that work with other students are also effective with ADHD students. It is also likely that the educational interventions that work with ADHD students work whether the ADHD students are medicated or not—but this possibility needs further research. We certainly agree with DuPaul and Eckert's (1997) suggestion that if we are looking to promote educational success among students with ADHD, we must use strategies that directly address their academic difficulties. Thus, although medical interventions can help ADHD children to control some of their dysfunctional behaviors in the short term and can provide relief to their families and teachers, if the improvement of educational outcomes is the aim, there is little evidence that medical intervention will succeed.

Notes

The research for this article was facilitated by a grant from the Australian Research Council. We wish to thank Kathryn Kelly and Andrea McCrindle, who assisted with the coding of the articles reviewed.

[1]In interpreting Tables 3 and 4, it is important to remember that the total number of subjects (n = 2,193) across the 74 studies is not the same as the total number of effect sizes (n = 1,141, 248, and 108 for normal, MR, and Tourette's people, respectively).

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