

ADHD and behavioral disorders: Assessment, management, and an update from DSM-5

■ ABSTRACT

Behavioral disorders in pediatric patients—primarily attention deficit hyperactivity disorder (ADHD)—pose a clinical challenge for health care providers to accurately assess, diagnose, and treat. In 2013, updated diagnostic criteria for behavioral disorders were published, including ADHD and a new diagnostic entity: disruptive mood dysregulation disorder. Revised criteria for ADHD includes oldest age for occurrence of symptoms, need for symptoms to be present in more than one setting, and requirement for number of symptoms in those aged 17 and older. Assessment of ADHD relies primarily on the clinical interview, including the medical and social history, along with the aid of objective measures. The clinical course of ADHD is chronic with symptom onset occurring well before adolescence. Most patients have symptoms that continue into adolescence, and some into adulthood. Many patients with ADHD have comorbid disorders such as depression, disruptive behavior disorders, or substance abuse, which need to be addressed first in the treatment plan. Treatment of ADHD relies on a combination of psychopharmacologic, academic, and behavioral interventions, which produce response rates up to 80%.

Behavioral disorders in pediatric patients—primarily attention deficit hyperactivity disorder (ADHD)—pose a clinical challenge for health care providers to accurately assess, diagnose, and treat. In 2013, the criteria for several disruptive behavioral disorders were updated in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*,¹ their first major revisions

since 1994. Among the most clinically relevant changes were revisions to the diagnosis of ADHD and the creation of a new diagnostic entity: disruptive mood dysregulation disorder (DMDD).

This article focuses on the updated diagnostic criteria published in the DSM-5 for behavioral disorders, describes the assessment of ADHD, and summarizes management strategies.

■ UPDATED DIAGNOSTIC CRITERIA

ADHD

This disorder is a chronic, neurologically based illness characterized by a persistent pattern of inattention and/or hyperactivity and impulsivity that are more inappropriate or disruptive than those in other children of a comparable age resulting in functional impairment in multiple settings, and these behaviors have been present for at least 6 months. Revised diagnostic criteria in DSM-5 used the same two categories for ADHD symptoms—inattention and hyperactivity-impulsive behaviors—but modified several diagnostic requirements.

Revised criteria

Impairment before age 12 instead of age 6. As a neurodevelopmental disorder, ADHD usually starts at a young age; teenagers presenting with newly developed ADHD-type symptoms probably do not have ADHD and efforts should be made to rule out other illnesses or social dynamics. The DSM-5 raised the age limit for onset of qualifying symptoms to before 12 years (previously by age 6) primarily to capture a cohort of pediatric patients, typically female, who present solely with inattention symptoms and may not display overt functional impairment early on.

Symptoms required in at least two settings. Symptoms must be present in at least two settings to qualify for a diagnosis of ADHD. This ensures that the behaviors occur globally; they do not occur just at school or at home but occur in both places.

Fewer symptoms required for diagnosis in adolescents.

Dr. Austerman reported that he has no financial interests or relationships that pose a potential conflict of interest with this article.

This article was developed from an audio transcript of Dr. Austerman's presentation at the "Perspectives in Pediatrics: From Theory to Practice" symposium held at the Global Center for Health Innovation, Cleveland, OH, May 8–10, 2014. The transcript was formatted and edited by *Cleveland Clinic Journal of Medicine* staff for clarity and conciseness, and was then reviewed, revised, and approved by Dr. Austerman.

doi:10.3949/ccjm.82.s1.01

Although the diagnostic criteria retain the same symptoms as those in DSM-IV for different age groups, individuals aged 17 and older are now required to display only five or more inattentive or hyperactive-impulsive symptoms. Previously, at least six were required.

Partial remission criteria

The concept of *partial remission* was introduced in DSM-5. This acknowledges that two-thirds of children diagnosed with ADHD do not have symptoms that functionally impact activities of daily living beyond age 18.

Oppositional defiant disorder

In DSM-5, oppositional defiant disorder (ODD) is defined by emotional and behavioral symptoms grouped into three categories:

- Constant anger or irritability
- Argumentative or defiant behavior (arguing with authority figures)
- Vindictiveness.

Because defiant behavior may represent difficulty with self-control, ODD is associated with executive functioning deficits that are present in ADHD. Children with ODD tend to perform best in situations in which they can dominate or exert authority. To qualify as ODD, the pattern of behavior must be consistent for longer than 6 months. A severity rating was added based on pervasiveness of ODD symptoms. Otherwise the diagnosis did not change.

Conduct disorder: Purposeful aggression

The hallmarks of conduct disorder are purposeful aggression (eg, bullying), destruction of property, deceitfulness or theft, and serious violation of rules (eg, running away from home, repeat truancy). Some consider conduct disorder to be a separate illness from ODD, whereas others consider it a continuum of the same disorder. Conduct disorder can manifest as violence, as in initiating physical fights, or it can manifest in behaviors such as truancy, stealing, lying, and running away from home without the physical-aggression aspect.

Intermittent explosive disorder

Failure to control aggressive impulses defines intermittent explosive disorder (IED). The aggressive outbursts can be verbal or behavioral and tend to be impulsive. A small subset of children display isolated aggression out of proportion to provocation. The disorder tends to manifest at ages 3 or 4, and a diagnosis requires a stable environment with no significant early childhood trauma. Most often these symptoms

are seen in children with intellectual disabilities or an autism spectrum disorder.

Disruptive mood dysregulation disorder

A new diagnostic category in DSM-5 is termed *disruptive mood dysregulation disorder* (DMDD). This captures many children who previously would have been diagnosed with pediatric bipolar disorder, even though most of them do not fulfill criteria for bipolar disorder as adults. The presence of baseline irritability separates this disorder from IED, which requires intermittent rapid and severe outbursts. The severe temper outbursts of DMDD must be recurrent, with an average of three occurrences per week, and have background irritability. The symptoms must have a duration of at least 12 months and be present in two settings. A diagnosis of DMDD cannot be made earlier than age 6, with onset before age 10.

ASSESSMENT OF ADHD

The clinical interview in conjunction with objective scales is the primary tool for diagnosing ADHD. The most frequent source of information is from the parents followed by the child's schoolteachers. Patient interview, although unreliable in young children, should also be part of the assessment. Comparing the patient's functional impairment against children of a similar age is necessary for an ADHD diagnosis.

The medical history can help rule out children with asthma or allergy being treated with corticosteroids and those with hypothyroidism and hyperthyroidism whose symptoms often fulfill the diagnostic criteria for ADHD.^{2,3} Symptoms of ADHD also may appear suddenly after a traumatic brain injury or other neurologic event.⁴ Other psychiatric illnesses, especially learning disorders, mood disorders, anxiety, other disruptive behavior disorders, or substance abuse, can mimic ADHD.

Ruling out other factors from a social history (eg, family conflict, bullying, sleep deprivation, being overscheduled with activities) adds to the reliability of an ADHD diagnosis. For example, repetitive uprooting and frequent changes in schools can cause academic problems that may be mistaken for ADHD, and use of stimulants may have failed to improve symptoms in these children.

Assessment scales

Pediatric assessment scales that can be performed in an office are more practical than standardized clinical assessments (Table 1). The Vanderbilt ADHD Diagnostic Teacher Rating Scale correlates highly with a

TABLE 1
Selected diagnostic tools for ADHD assessment

Scale or test	Notes and resources
Vanderbilt ADHD Diagnostic Teacher Rating Scale	http://www.nichq.org/childrens-health/adhd/resources/vanderbilt-assessment-scales
Vanderbilt ADHD Diagnostic Parent Rating Scale	http://www.nichq.org/childrens-health/adhd/resources/vanderbilt-assessment-scales
Conners, Third Edition	http://www.mhs.com/product.aspx?gr=cli&id=overview&prod=conners3 Successor to Conners Rating Scales–Revised (CRS–R)
Diagnostic Interview for Children and Adolescents (DICA-IV)	http://www.mhs.com/product.aspx?gr=edu&id=overview&prod=dicaiv Based on DSM-IV criteria
Schedule for Affective Disorders and Schizophrenia in School-Age Children–Present and Lifetime Version (K-SADS-PL)	http://bit.ly/K-SADS-PL_inst DSM-III-R and DSM-IV criteria

diagnosis of ADHD. We use the Vanderbilt ADHD Diagnostic Parent Rating Scale for children up to age 1 year. Other scales track symptoms and functional impairment over time and can be administered before the patient’s appointment. The Conners Third Edition scale can be used to establish a baseline before initiating therapy and to help monitor changes over time.

Standardized tests to bolster the utility of the clinical interview include the Diagnostic Interview Schedule for Children and Adolescents and the Schedule for Affective Disorders and Schizophrenia in School-Age Children–Present and Lifetime Version. Free training is available regarding use of some of these standardized tests.

Developmental course, risk factors

The clinical course of ADHD is chronic. The onset of hyperactivity usually occurs at age 3 or 4, with combined hyperactivity and inattention usually appearing from ages 5 to 8.^{5,6} The evolution of symptoms is progressive and constant. Between 50% and 80% have symptoms that continue into adolescence, and in about 40%, symptoms continue into adulthood.^{7,8} Some children with ADHD have a temperament-neuropsychological profile characterized by aggressiveness, irritability, and mood lability. Deficits in planning, delayed aversion, and temporal processing are present.

Risk factors include prematurity, prenatal complications, an anoxic event, nutritional deficits (specifically iron and zinc), and lack of appropriate socialization.^{9–11} The disorder is heritable, which is usually clear from the clinical interview. Rates of

delinquency and peer rejection are high. This may result in secondary comorbidity such as emotional, disruptive, or substance abuse problems.

MANAGEMENT STRATEGIES

Stimulants

The first-line pharmacologic treatment of ADHD is stimulants: methylphenidate, dexamethylphenidate, mixed amphetamine salts, dextroamphetamine, and lisdexamfetamine. Head-to-head trials of medications versus behavioral management favor medication use, even over the long term.^{12–14}

Methylphenidate and amphetamines are equally effective and have similar adverse effect profiles. Insomnia and anorexia are the most common side effects of stimulants. Cardiac effects include tachycardia, chest pain, and hypertension. Very rarely, stimulants have been associated with sudden cardiac death syndrome in patients with underlying cardiac problems. The consensus is that stimulants are safe in the general population. The need to obtain an electrocardiogram before initiating a stimulant was removed by the US Food and Drug Administration (FDA) unless it is otherwise indicated.¹⁵

The response rate to stimulants is in the range of 70%. About one-third of patients have side effects, and approximately 15% have side effects severe enough to requiring changing or withdrawing the medication.^{16,17}

Stimulants are available in several delivery systems. For the best effect, medications should be combined with behavioral management.

TABLE 2
ADHD comorbidities^{23,24}

Comorbidity	Rates
Oppositional defiant disorder	54%–67%
Conduct disorder	26%
Mood disorders	20%–30%
Substance abuse	12%–24%
Anxiety disorders	10%–40%
Tic disorders	18%

Alternatives to stimulants

If stimulants are ineffective, atomoxetine can be used to treat patients with inattention; however, its effect on hyperactivity and impulsivity is less pronounced than that of stimulants. Bupropion is another option for inattention. Both agents are well tolerated. Irritability and insomnia are side effects of atomoxetine, and liver damage is possible, so liver function tests must be ordered if the patient complains of upper-right-quadrant pain.

The evidence to support the use of modafinil is equivocal.^{18,19} Unlike stimulants, modafinil is associated with a slight increase in motivation.

Alpha-2 agonists are effective for treating aggression in the setting of ADHD, especially in younger children, and are well tolerated.²⁰ Extended-release forms are available.

Combination therapy

Polypharmacy is sometimes indicated in the treatment of ADHD. A stimulant used in combination with atomoxetine was shown to be superior to either treatment alone in improving symptoms of hyperactivity and inattention.²¹ The combination, however, markedly increased the incidence of appetite loss, insomnia, and irritability.

A more promising combination is a stimulant with an alpha agonist. Symptoms of hyperactivity and inattention were improved more with this combination than with a stimulant plus placebo, with no difference in side effects.²²

ADHD with comorbidities

Patients with ADHD, both adults and children, often have comorbid externalizing disorders and other emotional disorders, such as depression and anxiety, occurring in up to half of cases (**Table 2**).^{23,24} These comorbidities are important to consider when devel-

TABLE 3
Summary of drug therapy options for ADHD with comorbidities

ADHD + oppositional defiant disorder or conduct disorder

Stimulant or atomoxetine plus behavioral therapy
Stimulants + behavioral therapy + alpha agonist
Stimulants + behavioral therapy + second-generation antipsychotic

ADHD + mood disorders

Bipolar disorder
Second-generation antipsychotic; then add stimulant
Atomoxetine, alpha agonist, or bupropion
Major depressive disorder
Bupropion; then add stimulant
Selective serotonin reuptake inhibitor + stimulant
Cognitive behavior therapy + atomoxetine + alpha agonist

ADHD + substance abuse

Atomoxetine
Bupropion
Alpha agonist
Stimulant difficult to abuse (eg, lisdexamfetamine)

ADHD + anxiety

Atomoxetine
Selective serotonin reuptake inhibitor + stimulant or alpha agonist + cognitive therapy
Tricyclic antidepressants (for pediatric anxiety)

ADHD + tics

Alpha-2 antagonists
Atomoxetine

oping a treatment strategy. The following describes treatment options for the most common ADHD comorbidities (**Table 3**).

ODD or conduct disorder. The first-line therapy for these patients is a stimulant plus behavioral therapy. Adding an alpha agonist to this combination may be indicated if the comorbidity is severe. Second-generation antipsychotics also have been used as add-ons to stimulants with behavioral therapy, but weight gain and hormonal side effects are common.

Behavioral interventions are effective in targeting disruptive behavioral disorders, specifically multisystemic therapy. Multisystemic therapy is intensive

therapy that involves working with the patient's peer group or school, but most children must enter the legal system to receive this intervention. Multi-systemic therapy is the only intervention shown to improve symptoms associated with comorbid ADHD and conduct disorder.²⁵

Mood disorders. For these patients, the mood disorder is treated first. In doing so, symptoms of ADHD may disappear. For those with bipolar disease, a second-generation antipsychotic agent is superior to lithium in efficacy, maintenance of remission, and side effects in patients with a clear bipolar affective disorder, after which a stimulant can be added with less risk of developing manic symptoms. Using a stimulant first for this indication risks mood destabilization.

For patients with a major depressive disorder, bupropion can be used, although this indication is not FDA-approved, followed by the addition of a stimulant. One alternative is a selective serotonin reuptake inhibitor plus a stimulant; another is cognitive behavioral therapy plus atomoxetine and an alpha agonist.

Substance abuse. Patients with ADHD have high rates of substance abuse.^{26,27} Whether treatment of ADHD with stimulants reduces the risk of substance abuse is controversial. Because abuse of stimulants is common, start treatment with atomoxetine, bupropion, an alpha agonist, or a stimulant that is difficult to abuse (eg, lisdexamfetamine). Refer patients who are abusing substances to a specialist in substance abuse for behavioral management.

Anxiety. Atomoxetine is recommended for the treatment of anxiety that coexists with ADHD. A selective serotonin reuptake inhibitor in combination with a stimulant or alpha agonist, plus cognitive behavioral therapy, is another option for treating anxiety and ADHD. Tricyclic antidepressants have shown benefit in pediatric anxiety. Bupropion should not be used to target anxiety as it has been shown to have a limited effect on anxiety.

Tics. Stimulants may transiently exacerbate underlying tic disorders, but no longstanding difference in the course of tics has been observed with stimulant use.²⁸ Alpha-2 antagonists target both tics and ADHD, so their use is preferred.²⁹ Atomoxetine does not exacerbate tics but may reduce their frequency and severity.³⁰

Dietary factors

Although challenging to accomplish, management of diet, specifically removal of artificial food coloring and sodium benzoate preservatives, has been more efficacious than behavioral management in the long-

term reduction of core symptoms of ADHD.^{31,32} No herbal remedy has demonstrated efficacy in improving ADHD symptoms. The use of omega-3 fatty acids as a complement to stimulants has demonstrated efficacy in reducing core symptoms in ADHD.³³

Behavioral therapy

Several forms of behavioral therapy have shown utility in improving symptoms in ADHD. Evidence supports that ADHD responds to cognitive behavioral therapy.³⁴ In-school neurofeedback training for ADHD was shown to be better than cognitive training in improving inattention and hyperactivity-impulsivity at 6 months of follow-up.³⁵

Parental training has the most evidence to support its use in children with ADHD. The two most common forms are Pathways Triple P (Positive Parenting Program) and The Incredible Years. Triple P is an early intervention designed to promote positive parent-child relationships to reduce behavior problems.³⁶ The Incredible Years is a multicomponent program that emphasizes creating opportunities for active involvement, reinforcement of positive behavior, teaching skills, and setting clear limits, all of which are central to the social development strategy.³⁷

Many children with ADHD respond to in-school interventions, at least an evaluation to rule out learning disorders, which typically have high morbidity. Children may qualify for Individualized Education Program (IEP) services, such as peer tutoring,³⁸ computer-assisted instruction,^{39,40} and task-modification instruction.⁴¹ All of these have evidence to support their use.

REFERENCES

1. **American Psychiatric Association.** Diagnostic and Statistical Manual of Mental Disorders. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
2. **Hak E, de Vries TW, Hoekstra PJ, Jick SS.** Association of childhood attention-deficit/hyperactivity disorder with atopic diseases and skin infections? A matched case-control study using the General Practice Research Database. *Ann Allergy Asthma Immunol* 2013; 111:102-106.
3. **Pretorius E.** Corticosteroids, depression and the role of serotonin. *Rev Neurosci* 2004; 15:109-116.
4. **Keenan HT, Hall GC, Marshall SW.** Early head injury and attention deficit hyperactivity disorder: retrospective cohort study. *BMJ* 2008; 337:a1984.
5. **Applegate B, Lahey BB, Hart EL, et al.** Validity of the age-of-onset criterion for ADHD: a report of the DSM-IV field trials. *J Am Acad Child Adolesc Psychiatry* 1997; 36:1211-1221.
6. **Loeber R, Green SM, Lahey BB, Christ MAG, Frick PJ.** Developmental sequences in the age of onset of disruptive child behaviors. *J Child Fam Studies* 1992; 1:21-41.
7. **Barkley RA, Murphy KR, Fischer M.** Adult ADHD: What the Science Says. New York, NY: Guilford Publications; 2008.
8. **Rasmussen P, Gillberg C.** Natural outcome of ADHD with developmental coordination disorder at age 22 years: a controlled,

- longitudinal, community-based study. *J Am Acad Child Adolesc Psychiatry* 2000; 39:1424–1431.
9. Silva D, Colvin L, Hagemann E, Bower C. Environmental risk factors by gender associated with attention-deficit/hyperactivity disorder. *Pediatrics* 2014; 133:e14–e22.
 10. Lindström K, Lindblad F, Hjern A. Preterm birth and attention-deficit/hyperactivity disorder in schoolchildren. *Pediatrics* 2011; 127:858–865.
 11. Banerjee TD, Middleton F, Faraone SV. Environmental risk factors for attention-deficit hyperactivity disorder. *Acta Paediatr* 2007; 96:1269–1274.
 12. Brown RT, Amler RW, Freeman WS, et al; for the American Academy of Pediatrics Committee on Quality Improvement; American Academy of Pediatrics Subcommittee on Attention-Deficit/Hyperactivity Disorder. Treatment of attention-deficit/hyperactivity disorder: overview of the evidence. *Pediatrics* 2005; 115:e749–e757.
 13. The MTA Cooperative Group. A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. *Arch Gen Psychiatry* 1999; 56:1073–1086.
 14. MTA Cooperative Group. National Institute of Mental Health Multimodal Treatment Study of ADHD follow-up: 24-month outcomes of treatment strategies for attention-deficit/hyperactivity disorder. *Pediatrics* 2004; 113:754–761.
 15. Martinez-Raga J, Knecht C, Szerman N, Martinez MI. Risk of serious cardiovascular problems with medications for attention-deficit hyperactivity disorder. *CNS Drugs* 2013; 27:15–30.
 16. Barbaresi WJ, Katusic SK, Colligan RC, Weaver AL, Leibson CL, Jacobsen SJ. Long-term stimulant medication treatment of attention-deficit/hyperactivity disorder: results from a population-based study. *J Dev Behav Pediatr* 2006; 27:1–10.
 17. Schachter HM, Pham B, King J, Langford S, Moher D. How efficacious and safe is short-acting methylphenidate for the treatment of attention-deficit disorder in children and adolescents? A meta-analysis. *CMAJ* 2001; 165:1475–1488.
 18. Biederman J, Pliszka SR. Modafinil improves symptoms of attention-deficit/hyperactivity disorder across subtypes in children and adolescents. *J Pediatr* 2008; 152:394–399.
 19. Taylor FB, Russo J. Efficacy of modafinil compared to dextroamphetamine for the treatment of attention deficit hyperactivity disorder in adults. *J Child Adolesc Psychopharmacol* 2000; 10:311–320.
 20. Hirota T, Schwartz S, Correll CU. Alpha-2 agonists for attention-deficit/hyperactivity disorder in youth: a systematic review and meta-analysis of monotherapy and add-on trials to stimulant therapy. *J Am Acad Child Adolesc Psychiatry* 2014; 53:153–173.
 21. Wilens TE. Combined pharmacotherapy in pediatric psychopharmacology: friend or foe? *J Child Adolesc Psychopharmacol* 2009; 19:483–484.
 22. Wilens TE, Bukstein O, Brams M, et al. A controlled trial of extended-release guanfacine and psychostimulants for attention-deficit/hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry* 2012; 51:74–85.e2.
 23. Michielsen M, Comijs HC, Smeijjn EJ, Beekman ATF, Deeg DJH, Kooij JJS. The comorbidity of anxiety and depressive symptoms in older adults with attention-deficit/hyperactivity disorder: a longitudinal study. *J Affect Disord* 2013; 148:220–227.
 24. Jensen PS, Hinshaw SP, Kraemer HC, et al. ADHD comorbidity findings from the MTA study: comparing comorbid subgroups. *J Am Acad Child Adolesc Psychiatry* 2001; 40:147–158.
 25. Hazell P. Review of attention-deficit/hyperactivity disorder comorbid with oppositional defiant disorder. *Australas Psychiatry* 2010; 18:556–559.
 26. Wilens TE, Martelon M, Joshi G, et al. Does ADHD predict substance-use disorders? A 10-year follow-up study of young adults with ADHD. *J Am Acad Child Adolesc Psychiatry* 2011; 50:543–553.
 27. Charach A, Yeung E, Climans T, Lillie E. Childhood attention-deficit/hyperactivity disorder and future substance use disorders: comparative meta-analyses. *J Am Acad Child Adolesc Psychiatry* 2011; 50:9–21.
 28. Spencer T, Biederman J, Wilens T, et al. Efficacy of a mixed amphetamine salts compound in adults with attention-deficit/hyperactivity disorder. *Arch Gen Psychiatry* 2001; 58:775–782.
 29. Weisman H, Qureshi IA, Leckman JF, Scahill L, Bloch MH. Systematic review: pharmacological treatment of tic disorders—efficacy of antipsychotic and alpha-2 adrenergic agonist agents. *Neurosci Biobehav Rev* 2013; 37:1162–1171.
 30. Allen AJ, Kurlan RM, Gilbert DL, et al. Atomoxetine treatment in children and adolescents with ADHD and comorbid tic disorders. *Neurology* 2005; 65:1941–1949.
 31. McCann D, Barrett A, Cooper A, et al. Food additives and hyperactive behaviour in 3-year-old and 8/9-year-old children in the community: a randomised, double-blinded, placebo-controlled trial. *Lancet* 2007; 370:1560–1567.
 32. Schab DW, Trinh NH. Do artificial food colors promote hyperactivity in children with hyperactive syndromes? A meta-analysis of double-blind placebo-controlled trials. *J Dev Behav Pediatr* 2004; 25:423–434.
 33. Freeman MP, Hibbeln JR, Wisner KL, et al. Omega-3 fatty acids: evidence basis for treatment and future research in psychiatry. *J Clin Psychiatry* 2006; 67:1954–1967.
 34. Muñoz-Solomando A, Kendall T, Whittington CJ. Cognitive behavioural therapy for children and adolescents. *Curr Opin Psychiatry* 2008; 21:332–337.
 35. Steiner NJ, Frenette EC, Rene KM, Brennan RT, Perrin EC. In-school neurofeedback training for ADHD: sustained improvements from a randomized control trial. *Pediatrics* 2014; 133:483–492.
 36. Wiggins TL, Sofronoff K, Sanders MR. Pathways Triple P-positive parenting program: effects on parent-child relationships and child behavior problems. *Fam Process* 2009; 48:517–530.
 37. Haggerty KP, McGlynn-Wright A, Klima T. Promising parenting programs for reducing adolescent problem behaviors. *J Child Serv* 2013; 8(4).
 38. Greenwood CR. Longitudinal analysis of time, engagement, and achievement in at-risk versus non-risk students. *Except Child* 1991; 57:521–535.
 39. Mautone JA, DuPaul GJ, Jitendra AK. The effects of computer-assisted instruction on the mathematics performance and classroom behavior of children with ADHD. *J Atten Disord* 2005; 9:301–312.
 40. Rabiner DL, Murray DW, Skinner AT, Malone PS. A randomized trial of two promising computer-based interventions for students with attention difficulties. *J Abnorm Child Psychol* 2010; 38:131–142.
 41. Dunlap G, dePerczel M, Clarke S, et al. Choice making to promote adaptive behavior for students with emotional and behavioral challenges. *J Appl Behav Anal* 1994; 27:505–518.

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