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Editor-in-Chief

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IN THIS ISSUE

Autism in Children and Adolescents

Learning Objectives
After reading these articles, you should be able to:
1. Describe the evolution of the autism diagnosis from DSM-III through DSM-5 in children and adolescents. **2.** Discuss the impact of DSM changes over time on our understanding of autism and related disorders. **3.** Summarize some of the current findings in the literature regarding psychiatric treatment for children and adolescents.

From Infantile Autism to Autism Spectrum Disorder

Glen Elliott, MD, PhD, editor-in-chief,
The Carlat Child Psychiatry Report

Dr. Elliott has disclosed that he has no relevant financial or other interests in any commercial companies pertaining to this educational activity.

Changes in diagnostic criteria not only affect the clinical and public perception of a disorder, but also its perceived prevalence. Such is the case with autism. The evolution of the autism diagnosis since it was introduced into the Diagnostic and Statistical Manual (DSM) in 1980 inarguably has altered its reported prevalence, rising from an estimated 1 in 2,000 in 1980 to 1 in 68 in 2015 (https://www.cdc.gov/media/releases/2014/p0327-autism-spectrum-disorder.htm).

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In Summary

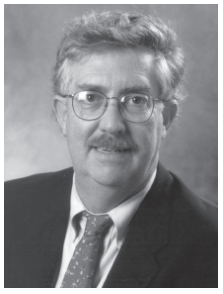
- DSM-III made autism a formal diagnosis, encouraging research and clinical interventions.
- DSM-IV greatly broadened the range of children and adolescents who qualified for autistic disorder or other forms of pervasive developmental disorders.
- DSM-5 formalized the growing clinical and research impression that autistic disorder is better described as a spectrum of problems with socialization, communication, and odd interactions with the environment.

Q&A With the Expert

Evolution of the Autism Diagnosis
Fred Volkmar, MD

Professor of psychology at the Yale Child Study Center, New Haven, CT

Dr. Volkmar has disclosed that he is an editor for Springer Publishing. Dr. Elliott has reviewed this article and found no evidence of bias in this educational activity.



CCPR: Where did autism arise, and how do we understand its evolution over the last 50 years?
Dr. Volkmar: The history of autism goes back many years. There is a small literature suggesting that some of the reports of so-called "feral children" in Europe starting in the very late 1700s/early 1800s, eg, *Victor, the Wild Boy of Aveyron* by Itard, may well have been children with autism who would either have bolted from their home or been abandoned by their families.

CCPR: So the condition is not new.
Dr. Volkmar: No, but it was not until Leo Kanner and Hans Asperger published clear descriptions of the condition in the mid-1940s that people began systematically thinking about it as a special phenomenon apart from the more general concept of mental retardation on the one hand and childhood schizophrenia on the other. There is some controversy about who actually "discovered" autism, but Kanner

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From Infantile Autism to Autism Spectrum Disorder (ASD)

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Below is a rundown of key DSM changes for autism and related disorders. These are summarized in the accompanying table.

DSM-III

DSM-III, published in 1980, was the first edition to include what was labeled "infantile autism." This diagnosis required all of the following:

- Onset before 30 months of age
- Pervasive lack of responsiveness to other people
- Gross deficits in language development
- Peculiar speech patterns such as immediate and delayed echolalia
- Bizarre responses to various aspects of the environment

These criteria would include only a small fraction of people with a current diagnosis of ASD—mostly younger individuals with significant cognitive impairments.

DSM-III-R

DSM-III-R, published in 1987, made several changes, including categorizing autistic disorder as a subtype of pervasive developmental disorder (PDD). By considering symptoms present in older individuals and eliminating the requirement of onset before 30 months of age, it expanded the definition of autism considerably. The new diagnosis of autistic disorder still had an age requirement but now was defined on the basis of meeting at least 8 of a total of 16 criteria divided into 3 domains.

- Onset during infancy or childhood
- Qualitative impairment in reciprocal social interactions, at least 2 of 5 criteria
- Qualitative impairment in verbal and nonverbal communication, at least 1 of 6 criteria
- A markedly restricted range of activities and interests, at least 1 of 5 criteria

Especially influential was the broadening of criteria regarding communication, which included, for example, an absence of imaginative play and inability to sustain a social conversation. DSM-III-R also introduced the diagnosis of pervasive developmental disorder, not otherwise specified (PDD NOS), which could be applied if patients met some criteria but not enough to meet a diagnosis of autistic disorder.

DSM-IV

DSM-IV, published in 1994, retained the diagnosis of autistic disorder introduced in DSM-III-R and kept the same domains of impairment. It further de-emphasized age of onset by requiring only some symptoms before 3 years of age, not the full syndrome. The total number of criteria dropped to 12, with 4 in each domain; patients had to meet 6 criteria in total. A diagnosis of autistic disorder required:

- Delays before 3 years of age in at least one of the following areas: social interaction, language used for social communication, or symbolic or imaginative play
- Qualitative impairment in reciprocal social interactions, at least 2 of 4 criteria
- Qualitative impairment in communication, at least 1 of 4 criteria
- A restricted range of activities and interests, at least 1 of 4 criteria

DSM-IV also expanded the number of disorders included under PDD, adding Asperger's disorder, Rett's disorder, and childhood disintegrative disorder. The criteria for autistic disorder and Asperger's disorder opened the door for a significant expansion in the number of individuals qualifying for a diagnosis. Research trying to establish whether autistic disorder and Asperger's disorder differed in major ways other than language met with equivocal results.

DSM-IV offered no specific measurement of severity of autistic disorder, but clinicians could use the Global Assessment of Function (GAF) to describe overall impairment.

DSM-5

DSM-5, published in 2013, introduced more major changes in the approach to diagnosing autism. The umbrella diagnosis of PDD was dropped, and autism spectrum disorder (ASD) replaced autistic disorder. Two diagnoses previously included under PDD—Rett's disorder and childhood disintegrative disorder—were omitted from DSM-5 altogether, the former because it is a well-defined genetic disorder and the latter because of its extremely low prevalence. Autistic disorder, Asperger's disorder, and PDD NOS were consolidated into ASD. Further, emphasis changed to 2 domains: a) impaired social interaction and communication and b) odd behaviors, and the deficits were explicitly described as "illustrative, not exhaustive," differing from the early practice of defining criteria explicitly. Thus, a diagnosis of ASD requires:

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From Infantile Autism to Autism Spectrum Disorder (ASD)

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- Symptoms early in development that may not manifest until social demands exceed capacity and may be masked later in life through learned strategies
- Persistent deficits in social communication and social interaction, with 3 "illustrations" (as opposed to specific criteria) such as poor socio-emotional reciprocity, impaired nonverbal communication, and difficulties developing and maintaining relationships
- A restricted range of activities and interests, with at least 2 areas of impairment, such as stereotyped motor movements, insistence on sameness, highly restricted interests, and either excessive or markedly diminished reactions to sensory input, similar to the 4 "illustrations" provided

DSM-5 introduced modifiers to the diagnosis, so clinicians specify intellectual impairment, language impairment, and other medical, genetic, and neurodevelopmental factors. Further, for patients with language problems who did not meet criteria for ASD, a new diagnosis of social (pragmatic) communication disorder was introduced, similar to the old Asperger's disorder. Finally, for individuals with ASD, DSM-5 created a 3-point severity scale for each domain: Level 1, "requiring support"; Level 2, "requiring substantial support"; and Level 3, "requiring very substantial support."

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Expert Interview

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was the first to publish a description of 11 cases in 1943 (Kanner L, *Nervous Child* 1943;(2):217–250. http://simonsfoundation.s3.amazonaws.com/share/071207-leo-kanner-autistic-affective-contact.pdf).

CCPR: And what was the essence of Kanner's description of the disorder?

Dr. Volkmar: Kanner defined two features he thought characterized autism. One was the "autism," or social withdrawal; the other was this funny category called "resistance to change." Resistance to change literally can be just that, but it also includes "insistence on sameness"—sort of two sides of the same coin. Further, he described stereotypic behaviors that he saw as an attempt on the child's part to maintain "sameness." Basically, he conceived of these kids as being socially rather clueless but overly attuned to and intolerant of changes in their environment. And, of course, by its very nature, social interaction is change. Then, things remained fairly static until the 1970s, when researchers began publishing books and papers about the disorder. Michael Rutter's work was especially influential.

CCPR: How did Rutter's work differ from Kanner's initial description?

Dr. Volkmar: Rutter basically systematized characteristics Kanner had described earlier, and his description strongly influenced the third edition of the Diagnostic and Statistical Manual (DSM-III), published in 1980, which was the first time psychiatry formally recognized autism as a disorder. In DSM-III, the criteria included a) onset before 30 months; b) a pervasive lack of responsiveness to others; c) severe delays in language development or, if language is present, d) peculiar speech patterns that could include immediate and delayed echolalia, metaphorical language, and pronominal reversal; e) atypical or bizarre responses to the environment; and f) no signs or symptoms suggestive of schizophrenia (see lead article for more details about the evolution of the autism diagnosis).

CCPR: DSM-III used a quite narrow definition of autism, consistent with Kanner's belief that it was a rare disorder.

Dr. Volkmar: Yes, DSM-III called the disorder "infantile autism"; there also was a diagnosis of "residual infantile autism" for people who had once had the diagnosis but lost it. Also, DSM-III created a new diagnosis called child-onset pervasive developmental disorder (PDD), which, like infantile autism, entailed marked social impairment, plus a broad array of intense emotional reactions or odd interactions with the environment, with an age of onset after 30 months to 12 years.

CCPR: How did this change with the next edition of DSM?

Dr. Volkmar: With the publication of DSM-III-R in 1987, there was an effort both to be more specific with respect to criteria and also to create more flexibility to account for different presentations. PDD became an umbrella diagnosis, with autistic disorder under it. Autistic disorder retained the three categories of problems in a) socialization, b) communication, and c) odd behaviors and trouble with change. Again, as elaborated in the accompanying article, each category had specific examples of qualifying behaviors, and a person received the diagnosis based on having "enough" criteria. Also, the specific cutoff of 30 months was abandoned, replaced by occurring during infancy or childhood. In addition, DSM-III-R added PDD, not otherwise specified (PDD NOS), intended for individuals with some symptoms of autism but not enough to meet criteria for autistic disorder.

CCPR: Did you agree with the changes made in DSM-III-R?

Dr. Volkmar: Well, in hindsight, it probably would have been better to use "autism and related conditions" or even "autism spectrum disorder." But, for many years, the emphasis was on autism as a specific, distinct disorder.

CCPR: What happened next?

Dr. Volkmar: In 1994, DSM-IV came out. I was very involved in that process, and we tried to correct several effects that DSM-III-R had had on how clinicians used these diagnoses. In my opinion, DSM-III-R was overly broad, especially among the more severely cognitively disabled: Many individuals previously diagnosed as having mental retardation were relabeled as having PDD. On the other hand, with its focus on young children, DSM-III-R's definition did not lend itself to diagnosing older, more cognitively nor-

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Expert Interview

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mal individuals. In DSM-IV, autistic disorder still included the same broad categories of social impairment, impaired language, and odd and restricted interests or behaviors, with some changes in specific criteria. The larger change was that PDD was broadened to include Asperger's disorder, childhood disintegrative disorder, and Rett's disorder, along with PDD NOS.

CCPR: Of those, it would seem that adding Asperger's disorder had the most marked effect.

Dr. Volkmar: Yes, with its requirement for relatively normal language development and focus on impaired social interaction plus restricted interests and odd behaviors, it more readily applied to older children, adolescents, and adults.

CCPR: What changed in 2013, when DSM-5 was released?

Dr. Volkmar: DSM-5 was a very different kettle of fish. It had some good things and some bad things, at least from my point of view. One of the good things about DSM-5 is the change of the name from autistic disorder to autism spectrum disorder (ASD), acknowledging the growing recognition, based partly on genetics, that autism is not the single disorder we once thought it to be.

"One of the good things about DSM-5 is the change of the name from autistic disorder to autism spectrum disorder, acknowledging the growing recognition, based partly on genetics, that autism is not the single disorder we once thought it to be."

Fred Volkmar, MD

CCPR: How else did DSM-5 change our perspective of autism?

Dr. Volkmar: Well, we used to say, "Oh, autism is very clear. It's the one true disorder in child psychiatry." Now we realize that there's a spectrum, which makes some sense, especially from a broad evolutionary perspective: If these changes were totally maladaptive, they presumably would have been lost over time; instead, they've persisted. That suggests that, for some people, some other combinations of these genes must be somewhat or even quite adaptive.

CCPR: Any drawbacks to the way DSM-5 defines ASD?

Dr. Volkmar: Paradoxically, even though it has the word "spectrum" in the label, ASD is quite narrowly defined. In DSM-IV, there were well over 2,000 combinations of criteria that could lead to a diagnosis of autistic disorder vs roughly 12 with DSM-5. In a way, it's a return to Kanner's conception of autism, and the new criteria bring it much closer to the DSM-III definition of infantile autism.

CCPR: Practically speaking, how does this change in criteria matter?

Dr. Volkmar: It means that a lot of DSM-IV autistic people who are at the higher range of cognitive ability no longer qualify for the diagnosis. Since 2013, several mini-studies and at least one meta-analysis of all the studies have shown that among those with higher levels of cognitive ability, roughly 80% do not keep a diagnosis of ASD (Smith IC, *J Autism Develop Dis* 2015;45(8):2541–2552). And, interestingly enough, the other problem area turns out to be very young children, many of whom no longer qualify for an ASD diagnosis. In both cases, especially in the U.S., this is a problem because if you don't qualify for a label, you don't get services.

CCPR: What's the solution, as you see it?

Dr. Volkmar: One temporary solution—though a less-than-ideal one—was incorporated into DSM-5. This was to grandfather people who have a well-established preexisting diagnosis into ASD. Of course, that does not address the issue of those first being diagnosed using DSM-5 criteria alone. I hope this narrowing of criteria will be rectified in the next iteration of DSM-5, especially with respect to very young children, where we believe intervention is the most likely to have the greatest benefit.

CCPR: Any other concerns with DSM-5?

Dr. Volkmar: Well, there is the loss of the diagnosis of Asperger's disorder. One certainly can debate the fine points of Asperger's or not Asperger's, but I think the diagnosis met a clear need that the ASD diagnosis does not address.

CCPR: Given your concerns about people being inappropriately excluded from an ASD diagnosis with DSM-5, how do you explain the perception that ASD has been rapidly increasing in prevalence over the past decade or so? Is the reported increased prevalence real? Is it just a definitional issue?

Dr. Volkmar: I think there are two aspects to this. One is, if you go back to think about yourself in grade school, did you know strange kids? Almost certainly. Did you know kids with the diagnosis of autism? Probably not. If you go back and look at the earliest editions of the Handbook of Autism in epidemiological terms and walk it out to what we now would define as a broader spectrum, it is on the order of 1 in 150–200 kids with autism.

CCPR: So much of the apparent increase is a matter of how we are defining our terms?

Dr. Volkmar: I think we've got this funny disconnect where both the lay public and professionals think about ASD in broad terms, resulting in high prevalence estimates, while "classical autism" remains relatively rare—though probably not as rare as originally believed. Another point is that the ASD label now, unfortunately, has a certain pizzazz. Parents know about it and would rather their child have ASD than, for example, intellectual disability. ASD is a more "hopeful" label, and you get more services around it.

CCPR: What about the actual data that the Centers for Disease Control and Prevention are reporting?

Dr. Volkmar: Many of the research studies that are done—even at the very high levels of the federal government—rely on sec-

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News of Note

Concerta: Brand vs Generic

In November 2016, the FDA announced that it was requiring two companies to withdraw their generic versions of Concerta (OROS methylphenidate) because of efficacy concerns. Such actions are quite unusual, and when they occur, they tend to shake doctors' confidence in the generic drug system—which branded drug companies are often eager to encourage. But the Concerta case is hardly an indictment of generic drugs, or even of generic stimulants. Let's dig a little deeper for some insight into the generic drug process.

Concerta was originally developed and marketed by Janssen Pharmaceuticals (a division of Johnson and Johnson). In 2010, the company lost its marketing exclusivity after its patent expired, and subsequently other companies manufactured generic versions. In approving generic products, the FDA mostly evaluates data showing that the generic formulation is "bioequivalent" to the brand product. This means both that the molecules are identical, and that the rate and extent of drug absorption is not significantly different. The FDA uses the designation "AB" for generics that are bioequivalent to brand, and "BX" for generics that are not equivalent and therefore not substitutable at pharmacies. Sometimes equivalency problems are discovered after a generic drug has been approved (a recent example is bupropion SR).

By 2013, a total of three companies, Actavis, Mallinckrodt, and Kudco, had received FDA approval to sell generic versions of Concerta, with all three generics classified as AB. But in December 2013, the FDA announced that it had received reports of lack of therapeutic effects with generic Concerta pills made by Mallinckrodt and Kudco (not the Actavis version of Concerta, whose bioequivalence has never been questioned). The reports from patients and physicians were specifically about duration of action, with the Mallinckrodt and Kudco products lasting for shorter periods than brand-name Concerta. This should not have been a surprise, because the companies did not have access to the OROS technology that acts as an osmotic pump but rather relied on coated-bead and bilayer technology (the Actavis generic uses the osmotic delivery system).

At the time, the FDA did not take any regulatory action, but said it would monitor and evaluate the issue. However, as more reports validated these concerns, in 2014, the FDA reclassified the Mallinckrodt and Kudco generics to BX, ie, therapeutically nonequivalent. The FDA allowed continued sales of both products, but asked the manufacturers to consider either voluntarily withdrawing them from the market or submitting more bioequivalence data. Neither company complied with the FDA's suggestions, though—in fact, Mallinckrodt instead opted to sue the FDA, a suit that was later dismissed by a Maryland federal judge.

In November 2016, presumably concluding that the companies were being intransigent, the FDA ordered that the two generic products be withdrawn. However, this process may take some time, and we recommend that you specify the manufacturer as Actavis on scripts when you prescribe generic Concerta.

The take-home lesson for the clinician: In the case of Concerta, not all generics are created equal. But we need to be clear that this was not a case of an adulterated or fake product. There were never any safety concerns, and Mallinckrodt and Kudco were selling genuine methylphenidate in a long-acting version—just not as long-acting as Concerta.

For more information, see the FDA's website at http://www.fda.gov/Drugs/DrugSafety/ucm422569.htm.

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Expert Interview

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ondary data, such as school-based records. One I recently reviewed derived its data from calling up parents and asking, "Has anyone ever mentioned autism to you in terms of talking about your child?"

CCPR: Not very rigorous.

Dr. Volkmar: Exactly. I think the reality is that we truly don't know. My guess is—and I think most people who know a lot about this would probably agree—that the actual prevalence has not changed much, if at all, since the mid-1940s. But, as I said, we don't really know, and part of the problem is trying to disentangle the more classical Kannerian autism from the broader spectrum.

CCPR: Any other factors that might be influencing our perception of prevalence?

Dr. Volkmar: Yes. With early intervention and detection, kids are doing better over time, which is great news. We've got more and more kids with ASD going to college. As far as I'm aware, there are five undergraduates at Yale right now with autism, self-identified. That used to never happen.

CCPR: So, you're saying that, either because we have broadened the definition considerably or because of earlier detection and intervention, we may be seeing kids who truly would qualify for the old DSM-III residual state autism?

Dr. Volkmar: Certainly it may be worth looking at that possibility again.

CCPR: Thank you, Dr. Volkmar, for sharing your extensive experience on this important topic.

Research Updates IN PSYCHIATRY

OCD

Internet-Delivered CBT for Adolescents With OCD

STUDY REVIEWED: Lenhard F et al. Therapist-guided, Internet-delivered cognitive-behavioral therapy for adolescents with obsessive-compulsive disorder: A randomized controlled trial. *J Am Acad Child Adolesc Psychiatry* 2017;56(1):10–19. doi:10.1016/j.jaac.2016.09.515

STUDY TYPE: Randomized controlled trial

The prevalence of obsessive-compulsive disorder (OCD) in childhood and adolescence is 2% (Angst J et al, *EurArch Psychiatry Clin Neurosci* 2004;254:156–164). Cognitive behavioral therapy (CBT) is a very effective treatment for pediatric OCD. So why aren't more parents taking advantage of it? It can be costly, and there aren't enough therapists who are well trained in using CBT for adolescents with OCD. The question is, how can we deliver this proven treatment to the patients who need it? In this study, Lenhard and colleagues attempted to determine whether Internet-delivered CBT is effective.

This 12-week study took place in Stockholm, Sweden. In it, 67 patients with OCD, between 12 and 17 years of age, were randomly assigned to a therapist-guided Internet CBT group (ICBT, n = 33) or to a waitlist (n = 34). Participants were recruited through advertising or referral by primary care doctors or mental health specialists. Outcomes were measured at baseline, 12 weeks (the end of treatment), and 3 months post-treatment. The ICBT program, designed by trained CBT therapists for a previous study, consisted of 12 online chapters with text, films, and animations. Some chapters were primarily for the patients, whereas others were designed for parents. Therapists were available to parents via email and phone throughout the study, but there were no face-to-face therapy appointments scheduled.

RESULTS

At the end of treatment, the ICBT group improved significantly more on the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS) scores compared with the waitlist group ($p < .001$); 9 ICBT subjects were responders, and 5 remitted. Interestingly, at the 3-month follow-up, there was even more improvement in the ICBT group, with 10 responders and 8 remitters. By contrast, no one in the waitlist group responded or remitted at any time point.

CCPR'S TAKE

This was a fairly small study, and it did not include an active control group, but nonetheless the results were impressive. At 3 months post-treatment, 18 of 33 adolescents responded or remitted. The authors point out that the response was not as robust as that seen in studies of adults using ICBT or in studies of face-to-face CBT in pediatric OCD populations. Still, clinicians spent only about 17 minutes weekly with each participant—far less than in face-to-face CBT—so cost was significantly reduced.

PRACTICE IMPLICATIONS

Internet CBT is not the perfect solution: Patients must have access to the Internet and an ICBT program and have a parent who is motivated enough to participate. This is therapist-guided CBT, not just a self-help program. Still, despite its limitations, ICBT may offer a feasible way of getting treatment to individuals who otherwise might suffer the fate of those on the waitlist—that is, no relief.

ADHD

Can a 10-Minute Intervention Improve Sleep in Children With ADHD?

STUDY REVIEWED: Peppers K et al. An intervention to promote sleep and reduce ADHD symptoms. *Journal of Pediatric Healthcare* 2016;30(6):43–48.

STUDY TYPE: Open, uncontrolled clinical trial

We know that kids with ADHD often have sleep issues, and that the stimulants we use to treat them can cause insomnia. What would happen if we focused our treatment on the insomnia portion of ADHD? Presumably kids would sleep better, but would their ADHD symptoms also improve?

The authors of this new study based this pilot project on an earlier randomized controlled trial of a sleep intervention with 244 Australian children (Hiscock H et al, *BMJ* 2015;350:h68. doi:10.1136/bmj.h68). In that study, children with ADHD were randomly assigned to either a brief intervention to improve sleep or a control condition. Those in the intervention group were seen twice by a clinician, who evaluated the sleep problem and provided tips on sleep hygiene. Clinicians recommended a regular bedtime routine, avoidance of caffeine after 3 pm, and no screen media in the bedroom. Children in the intervention group showed significant improvements in ADHD symptoms and sleep quality.

The goal of the current 20-week project was to see whether a similar intervention delivered by video and not requiring highly trained clinicians would also be successful. Twenty-three children, between 5 and 11 years of age, with both ADHD and sleep problems as assessed by the Children's Sleep Habits Questionnaire (CSHQ), were enrolled in an open, non-controlled trial. In this intervention, children and parents watched a single 6-minute video, which described good sleep hygiene practices like those in the Hiscock intervention described above. After the video, the provider gave the parents a written sleep hygiene plan. Six weeks after the intervention, the children were assessed again.

RESULTS

After 6 weeks, the children showed significant improvement in both ADHD and sleep symptoms. Improvement on the Parent Visual Analogue Scale was significant on questions 1–9 dealing with inattention ($p <. 001$) and questions 10–18 dealing with hyperactivity ($p < .004$). The scores on the CSHQ

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CME Post-Test

To earn CME or CE credit, you must read the articles and log on to www.TheCarlatChildReport.com to take the post-test. You must answer 75% of the questions correctly to earn credit. You will be given two attempts to pass the test. Tests must be taken by February 28, 2018. As a subscriber to *CCPR*, you already have a username and password to log onto www.TheCarlatChildReport.com. To obtain your username and password, please email info@thecarlatreport.com or call 978-499-0583.

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Below are the questions for this month's CME/CE post-test. This page is intended as a study guide. Please complete the test online at www.TheCarlatChildReport.com. Note: Learning Objectives are listed on page 1.

1. Autism has been formally recognized as a diagnosis in the Diagnostic and Statistical Manual (DSM) since: (LO #1)
   [ ] a. 1968 [ ] b. 1980 [ ] c. 1994 [ ] d. 2013

2. Based on changes from DSM-IV to DSM-5, approximately what percentage of patients with higher levels of cognitive ability do not keep the diagnosis of autism spectrum disorder? (LO #2)
   [ ] a. 20% [ ] b. 35% [ ] c. 55% [ ] d. 80%

3. DSM-IV differs most significantly from DSM-III-R and DSM-5 in which of the following ways? (LO #1)
   [ ] a. It used criteria that markedly constricted the number of individuals who qualified for an autism diagnosis or related disorder
   [ ] b. It introduced the diagnostic category of autism spectrum disorder (ASD)
   [ ] c. It broadened the category of pervasive developmental disorder (PDD) markedly, including autistic disorder, Asperger's disorder, and several others
   [ ] d. It was the first version to use PDD as an umbrella diagnosis for autism

4. Which of the following statements is true about DSM-5 in relation to autism? (LO #1)
   [ ] a. DSM-5 retained the three domains of social impairment, language impairment, and odd behavior
   [ ] b. DSM-5 characterized autism as a specific, single disorder
   [ ] c. DSM-5 required onset of the disorder to occur before 30 months of age
   [ ] d. DSM-5 combined social impairment and communication impairment into a single category

5. According to a recent study, although adolescents with OCD showed improvement after 12 weeks of Internet-guided cognitive behavioral therapy compared to a waitlist group, at 3-month follow-up, both groups had the same results. (LO #3)
   [ ] a. True [ ] b. False

Changes in the Diagnostic and Statistical Manual (DSM) for Autism and Related Disorders

	DSM-III	DSM-III-R	DSM-IV	DSM-5
Year Released	1980	1987	1994	2013
Labels	Infantile autism, pervasive developmental disorder (PDD)	PDD as overarching category for autistic disorder and PDD not otherwise specified (NOS)	PDD as overarching category for autistic disorder, Asperger's disorder, Rett's disorder, childhood disintegrative disorder, and PDD NOS	Autism spectrum disorder (ASD)[1]
Diagnostic Requirements	Pervasive nonresponsiveness to others, gross language deficits, odd speech if present, and bizarre responses to the environment	At least 8 of 16 criteria across 3 domains: impaired social interactions, impaired language, and restricted interests	At least 6 of 12 criteria across 3 domains: impaired social interactions, impaired communication, and restricted activities and interests	Dysfunction across 2 domains, with persistent deficits in social interaction and communication and at least 2 areas of impairment related to interests and activities
Onset of Symptoms	Before 30 months of age	During infancy or early childhood	Some delay in socialization or language before age 3	When expectations and demands exceed developmental capacity
Severity Specifiers	None	None	Optional use of Global Assessment of Functioning (GAF) but not specific for autistic disorder	Levels of impairment for each domain: 1) "requiring support", 2) "requiring substantial support," and 3) "requiring very substantial support"

[1]DSM-5 was the first to introduce the option of specifying modifiers related to intellectual impairment, language impairment, and other medical, genetic, and neurodevelopmental factors.

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This Issue's Focus:
Autism in Children and Adolescents

Next Time in *The Carlat Child Psychiatry Report:* Tourette's and Other Tic Disorders

Research Updates
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showed significant improvement from baseline to the 6-week re-assessment ($p < .001$).

CCPR'S TAKE

This study implies that a very brief and easy-to-administer sleep intervention may lead to improvement in both ADHD and insomnia symptoms in children. The study was limited by the lack of a control group, so it's possible that these improvements were the result of placebo factors having little to do with the intervention. In addition, there was no teacher rating of ADHD symptoms, decreasing our confidence that the improvement in those symptoms was robust enough to be apparent in school as well as at home. The original study by Hiscock et al from 2015 is more compelling: There were a larger number of participants, and it was a randomized controlled trial.

PRACTICE IMPLICATIONS

When you evaluate patients for ADHD, make sure to diligently ask about sleep issues, and take some extra time to talk about sleep hygiene, including providing parents with a sleep hygiene handout. This may pay dividends for improving sleep and ADHD symptoms.

Colleen Ryan, MD. Dr. Ryan has disclosed that she has no relevant financial or other interests in any commercial companies pertaining to this educational activity.