

Should We Be Rethinking How We Assess and Manage ADHD?

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Attention-deficit/hyperactivity disorder (ADHD) is one of the most common clinical problems assessed and treated by child and adolescent psychiatrists, yet many important questions remain unanswered. In this month's issue of the *Journal*, two articles address two puzzling issues. First, what can research tell us about how we should be conceptualizing and defining ADHD? Second, although it is well recognized that ADHD and depression co-occur, why do we see this overlap? The authors of both articles use nonclinical, population-derived twin samples in different, imaginative ways.

The first article, by Lubke and colleagues¹ capitalizes on a large sample of Dutch children who were assessed at ages 7, 10, and 12 years. The authors investigate whether ADHD, like high blood pressure or diabetes, represents an extreme of an underlying continuum by examining the pattern of statistical clustering of symptoms assessed using the Child Behavior Checklist attention scale. The second article, by Cole and colleagues,² is based on a U.K. sample of children aged 5 to 16 years and uses the twin design in a genetically informative way to examine the co-occurrence of ADHD and depressive symptoms.

Let us consider the first critical question: how should we, as clinicians, define ADHD? Is ADHD best considered as a qualitatively distinct category like an infectious disease, or as lying at the end of a continuum present in the general population, akin to hypertension and diabetes? What about ADHD subtypes—how valid is it to distinguish between these subtypes? The ADHD subtypes are included in the current *DSM-IV* classificatory system but are not generally

used in Europe, where *International Statistical Classification of Diseases, 10th Revision*, is more commonly used. Defining ADHD is, of course, not only clinically important but also highly topical, given the current deliberations on *DSM-V*. There has been much debate on the merits and limitations of defining disorders categorically versus dimensionally.³ Many have considered that each approach has its different advantages. For example, categorical diagnoses can be useful, given that many clinical decisions are categorical in nature, for example, “give medication or not,” or “refer to a child and adolescent psychiatrist or not.” However, there are also limitations of such an approach, as described in the article by Lubke and colleagues.¹ Regardless of viewpoint, what does the research evidence support?

Lubke et al.¹ show that the pattern of symptom clustering provides support for an ADHD continuum. They find that there is an underlying range of symptom severity, with most *DSM-IV* ADHD cases (a subsample assessed using the Diagnostic Interview Schedule for Children) falling into the extreme end of that continuum. They do not find evidence that *DSM-IV* subtypes can be distinguished. Genetic studies have been mixed with regard to this; some find that the subtypes are distinct from each other, whereas others do not.⁴ Thus, Lubke et al.,¹ in summary, found evidence in favor of conceptualizing ADHD as a continuum, with clinical cases lying at the extreme end, like hypertension does in relation to the continuum of blood pressure. These findings suggest that quantifying ADHD severity along a dimensional measure will be a helpful means of clinically communicating about patients with ADHD and of monitoring treatment response.

Is there any other evidence supporting this view? Indeed, there is. Previous population-based studies have tackled this issue differently and come up with the same answer. Twin and epidemiological studies also suggest that ADHD behaves as a continuum with regard to the pattern of heritability, association with environmental correlates, and in terms of future risk effects on antisocial behavior.⁵ A dimensional model of ADHD has also been supported in a clinical population using neuropsychological data.⁶ However, what is really useful

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about the Lubke et al. study is that they map their findings onto interview-derived *DSM-IV* diagnoses of ADHD.

Like every research study, this one also raises questions to be addressed in future work. For clinical purposes, we need validated thresholds to make a diagnosis and to make sensible judgments about treatment. As the authors point out, this could depend on age and sex. Context might also be important. Thresholds for diagnosing and treating hypertension and high blood sugar have changed over time and now differ for specific patient groups depending on risk. We also need to think about impairment resulting from symptoms or, as for diabetes, risk effects on future adjustment. This is especially important, given we have no biological markers for ADHD. Another issue is that current classification systems require the presence of ADHD symptoms or impairment in more than one setting, and many clinicians will require teacher reports. How do we integrate information from two sets of informants in a way that improves our diagnostic and treatment decisions?

The second paper deals with a scenario well-recognized by clinicians. Why does ADHD so commonly co-occur with depression? Using genetic designs is a powerful way to address this question. Again, this is a topical issue, with new genetic findings suggesting that traditional boundaries between disorders, such as schizophrenia and bipolar disorder, or autism and schizophrenia, may be unfounded at a biological level. Cole et al.² find that the co-occurrence of ADHD and depressive symptoms is predominantly explained by shared genetic liability. As the authors state, this suggests that susceptibility genes that influence mood may also affect ADHD. How confident are we that this truly might be the case? It should be noted that this finding does not stand in isolation. Family studies have previously shown that ADHD diagnoses and major depressive disorder share a common familial liability.⁷ What does this mean clinically? It suggests that clinicians need to be not only mindful of screening for depression in children with ADHD but also aware that mood disorders could affect parents of children with ADHD for biological as well as social reasons. The presence of parent depression is important when planning treatment and ensuring successful engagement with the family.

Is it all shared genes? It is important to remember that the genetic liability from twin studies may also include gene-environment correlation effects (environmental risk effects arising from risk genes) and gene-environment interaction (genetic sensitivity to environmental factors). The even more interesting finding in our view is that Cole et al.² show significant environmental pathways between ADHD and depressive symptoms. This begs the question as to what these environmental factors might be and whether they are amenable to intervention. Another possibility that could be ex-

plored in future longitudinal studies is whether ADHD leads to depression through an environmental route. For example, twin and medication studies suggest that ADHD in boys leads to increased mother-son hostility.⁸ It is interesting to speculate if future studies will show whether altered family relationships or parenting⁹ as a consequence of ADHD could account for the link with depression. In addition to looking at the underlying biological and psychosocial mechanisms linking ADHD and depression, another interesting future aim would be to examine a larger sample of adolescents, given that rates of depression, especially in female subjects, rise sharply in this age group.

In summary, here are two interesting articles on ADHD. What are the take-home messages for clinicians? First, it seems that it will be useful to define ADHD along a diagnostic continuum of severity, and perhaps, we ought to worry less about subtypes. Second, we need to screen for depression in families of children with ADHD, as well as in the children themselves. Third, there has been much guidance on the treatment of ADHD; clinicians need to keep an eye open for future research that looks at modifiable mechanisms underlying comorbidity in ADHD. Such research findings might guide interventions aimed at preventing or reducing comorbid depression.

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