

Current Issues in the Diagnosis of Attention Deficit Hyperactivity Disorder, Oppositional Defiant Disorder, and Conduct Disorder

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Annu. Rev. Clin. Psychol. 2012. 8:77–107

First published online as a Review in Advance on December 21, 2011

The *Annual Review of Clinical Psychology* is online at clinpsy.annualreviews.org

This article's doi:
10.1146/annurev-clinpsy-032511-143150

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1548-5943/12/0427-0077\$20.00

Keywords

diagnostic criteria, classification, DSM, subtypes, developmental issues

Abstract

This review evaluates the diagnostic criteria for three of the most common disorders for which children and adolescents are referred for mental health treatment: attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD). Although research supports the validity and clinical utility of these disorders, several issues are highlighted that could enhance the current diagnostic criteria. For ADHD, defining the core features of the disorder and its fit with other disorders, enhancing the validity of the criteria through the lifespan, considering alternative ways to form subtypes of the disorder, and modifying the age-of-onset criterion are discussed relative to the current diagnostic criteria. For ODD, eliminating the exclusionary criteria of CD, recognizing important symptom domains within the disorder, and using the cross-situational pervasiveness of the disorder as an index of severity are highlighted as important issues for improving classification. Finally, for CD, enhancing the current subtypes related to age of onset and integrating callous-unemotional traits into the diagnostic criteria are identified as key issues for improving classification.

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Disorders, Fifth Edition (DSM-V) ADHD and Disruptive Behavior Disorders Work Group (Regier et al. 2009) has been evaluating the current methods for classifying a group of disorders that include attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD). This evaluation has included:

- conducting literature reviews on key issues related to the diagnostic criteria for each disorder,
- identifying key limitations in existing criteria for which research offers a clear suggestion for improvement,
- conducting secondary data analyses when data are available to test possible improvements to the diagnostic criteria,
- developing proposed changes to the diagnostic criteria as indicated by these reviews and secondary data analyses, and
- testing the reliability and ease of use of these changes in field trials.

Throughout this process, comments from the field have been solicited in the form of announcements in scientific journals and direct email solicitations for comment to selected researchers. Also, a Web site (<http://www.dsm5.org>) has been established to update the field to changes being considered and to allow public comment on them.

The goal of the current review is to provide the perspective from two work group members on key issues that have emerged from the intensive scrutiny of these common disorders, which we believe are crucial for improving their classification for both research and practice. However, we emphasize that this review is not an official statement from the work group, and at times our perspective may be different from the official stance of the work group.

Also, our review does not address broad issues on the appropriate uses of the DSM for guiding clinical practice, research, and treatment development. We also do not provide a comprehensive discussion of the most appropriate ways to define a “mental disorder.” Both of these issues have been discussed in recent comprehensive reviews (First & Wakefield

INTRODUCTION

Since 2007, the American Psychiatric Association’s *Diagnostic and Statistical Manual of Mental*

2010, Hyman 2010). However, we note that it is important to specify and prioritize the goals of a classification system, like the DSM, so as to guide any recommendations for improving the system. One possible set of priorities is that changes should improve clinical practice, guide research on etiology, and foster the development of new and more effective treatments for mental disorders. Few would argue with changes that clearly enhance all of these important uses of the DSM. However, in practice, proposed changes may accomplish one of these goals (e.g., promote further research or reflect recent findings) but at the expense of another (e.g., make the diagnosis more difficult to use by most practicing clinicians). How are priorities then to be weighed?

As a result, a clear, overarching framework is needed for the DSM or any similar classification system, one that states which goal or goals take priority when considering changes to the system. The DSM, while aiming to enhance etiological research by providing reliable diagnostic criteria for study, has historically placed a high priority on informing clinical practice and on being clinically usable (Regier et al. 2009). Thus, it emphasizes phenomenological descriptions of symptoms or signs that predict the need for treatment (i.e., have been associated with current impairment or high risk for future impairment). This can be compared to the National Institute of Mental Health Research Domain Criteria (RDoC), which were developed for research purposes (Insel et al. 2010). This system emphasizes crosscutting hypothetical mechanisms related to causal processes (e.g., negative affect, positive affect, cognition, social processes, arousal/regulatory systems) and their associated hypothesized neural circuits for classifying groups of participants for research studies (Sanislow et al. 2010). Both systems attempt to utilize the current research to evaluate potential changes in the diagnostic criteria. However, the different primary goals of the two systems can, in some instances, lead to different ways of interpreting the available research as to how diagnostic criteria should or should not be changed.

In our evaluation of the DSM criteria for ADHD, ODD, and CD, we start with the DSM-IV definition of these disorders. Paralleling the task of the work group on which we served, we briefly highlight key research to underscore strengths and limitations in the existing definitions. We emphasize potential improvements in diagnostic criteria for guiding clinical practice, but we also note ways to improve the diagnostic criteria for guiding causal research. As noted by the developers of the RDoC, an integration of a process-oriented approach such as the RDoC and the clinical syndrome approach such as the DSM is an appropriate future goal but is likely beyond the state of current research in most areas (Sanislow et al. 2010). However, we note changes that can set the stage for such integration in the future. Although the majority of this review focuses on research specifically related to ADHD, ODD, and CD, several cross-cutting issues require consideration in the classification of any mental disorders and are of particular importance to the diagnosis of ADHD, ODD, and CD.

CROSS-CUTTING ISSUES IN THE DEVELOPMENT OF DIAGNOSTIC CRITERIA

Dimensional Versus Categorical Classification

The first broad issue pertains to dimensional classification versus categorical description of psychopathology. Perhaps the most common criticism of the DSM approach to classification of disorders in general (Krueger et al. 2005) and to classifying children and adolescents specifically (Hudziak et al. 2008), is the focus on placing individuals into discrete categories when making diagnoses. This issue may be the one that most clearly separates how the DSM is used in clinical practice (i.e., using discrete diagnostic categories) from how it is often used in research (i.e., using continuous measures of symptoms). This categorical approach ignores the consistent findings that the symptoms of many disorders form a continuous dimension rather than a discrete taxon (Marcus & Barry 2011),

DSM: *Diagnostic and Statistical Manual of Mental Disorders* (III, Third Edition; III-R, Third Edition-Revised; IV, Fourth Edition)

ADHD: attention deficit hyperactivity disorder

ODD: oppositional defiant disorder

CD: conduct disorder

Diagnostic criteria: rules that specify how a diagnosis should be made, including number and types of symptoms, duration of symptoms, exclusionary criteria, etc.

RDoC: National Institute of Mental Health Research Domain Criteria

Dimensional classification: using methods for classifying disorders that recognize that diagnostic thresholds are somewhat arbitrary; rather than focusing on how disordered people differ from nondisordered people, it focuses on the important continuous dimensions on which people may vary

meaning that persons who differ on number of symptoms typically differ more in severity than in type. Such findings are not consistent with the implication of diagnostic thresholds that appear to cleanly designate “disordered” from “non-disordered” individuals. Further, the reliance on categorical diagnoses ignores the fact that among disordered individuals there often are clear variations in severity (DeShazo-Barry et al. 2002) and that individuals just below the threshold for a disorder may still have clinically impairing levels of symptoms (Lewinsohn et al. 2004). Thus, psychiatric classification in general, and the DSM in particular, needs to begin to move toward incorporating dimensional approaches to classification (Regier et al. 2009).

But how is this to be done? At times the debate over whether a dimensional is better than a categorical classification system is confounded with how best to make categorical decisions. For example, one can debate whether a categorical decision on the presence of a disorder should be based on whether the child is more severe than others of his or her same age (i.e., normative comparison) or on whether the child is sufficiently impaired (Achenbach 2009). However, at some point in the use of diagnostic criteria in clinical practice, discrete decisions are required, including “does this person need treatment?” and “for what should they be treated?” An adequate classification system must provide sufficient guidance on how to make these decisions. Although it is unlikely that a diagnostic classification system can completely abandon all categorical decisions or cut points, recognition of dimensional variation will improve the system and may, in the long run, help resolve long-standing problems with the DSM system (such as excessive rates of “comorbid” disorders and excessive within-disorder heterogeneity; Rutter 2011).

Therefore, the goal for improving classification systems is not to replace categorical diagnoses completely but rather to integrate dimensional approaches into diagnostic criteria to increase their utility for both research and practice. At minimum, such approaches should recognize that:

- a diagnostic threshold is necessarily somewhat arbitrary (a clinical tool, rather than an epistemological assertion), and individuals just below the threshold may have impairing symptoms,
- individuals within a diagnostic category may vary greatly on severity of symptoms and degree of impairment, and
- diagnostic criteria may need to vary depending on the person’s age, culture, and gender.

In our discussion of the criteria for ADHD, ODD, and CD, we note some opportunities for accomplishing these objectives. However, we also recognize some of the problems and dangers in such an approach. For example, recognizing “subthreshold” levels of symptoms, which may need to be a focus of intervention, creates a danger of overinclusiveness. That is, people who do not meet criteria for a disorder may be diagnosed as subthreshold and exposed to stigma similar to what is associated with having a psychiatric disorder. Such subthreshold classification may be subject to high risks of unreliability and arbitrary application, weakening the overall credibility and utility of the diagnostic system. Further, it is not clear whether or how third-party payers would recognize those below the diagnostic threshold for reimbursement for services. Finally, recognizing gradations in severity within a diagnostic criteria (e.g., mild, moderate, severe) could add to the number of arbitrary distinctions made by the criteria, going from one (i.e., disordered or not) to multiple (e.g., mild or moderate, moderate or severe) somewhat arbitrary—and potentially unreliable—distinctions.

One final topic with regard to integrating dimensions into the DSM is the opportunity to enhance the meta-structure or the overall organization in the way disorders are grouped. That is, in the DSM-IV, disorders are organized into a large number of chapters based largely on clinical presentation, such as whether the disorders are typically first diagnosed in infancy, childhood, or adolescence or whether they share symptom presentation (e.g., anxiety disorders, dissociative disorders;

APA 2000). Currently, ADHD, ODD, and CD are classified with disorders first diagnosed in youths. This is problematic for a number of reasons. First, the designation of “typically diagnosed” prior to adulthood is somewhat arbitrary in that persons with many disorders not placed in this category (e.g., phobic disorders) are often first diagnosed prior to adulthood (Kessler et al. 2007), and this structure seems to minimize the importance of having criteria for ADHD, ODD, and CD that are appropriate for individuals throughout the lifespan (Barkley et al. 2008). Second, it leads to the grouping of disorders that seem to have little in common (e.g., separation anxiety disorder and CD) in symptom presentation, etiology, or treatment.

Thus, an important consideration when integrating a more dimensional approach to the DSM is to determine whether disorders can be better organized or grouped in a way that takes into account common dimensions, such as shared risk factors or common symptom clusters. For example, there are rather extensive data to suggest that ODD, CD, hyperactivity, impulsivity, and substance use (but not inattention) tend to cluster into an overarching externalizing or disinhibition dimension (Lahey et al. 2008, Walton et al. 2011) that seems to share substantial genetic influences (Lahey et al. 2011, Markon & Krueger 2005). Similar data are available to support grouping anxiety and depressive disorders into an internalizing or emotional dimensional (Lahey et al. 2008). Such groups could enhance the conceptual appeal of the manual by explaining common comorbidities and could encourage research that investigates the shared liability to the disorders (or dimensions) within the group, as well as nonshared factors that lead to the different disorders (Krueger & Markon 2011, Lahey et al. 2011). However, one problem with this approach is the difficulty in finding dimensions that work to organize all disorders within a classification system (Andrews et al. 2010). Further, some disorders may have subtypes or else components that differ on where they cluster; notably, within ADHD,

symptoms of hyperactivity/impulsivity cluster with externalizing, but inattention less so, and the overlap of all these symptoms with developmental problems complicates the picture.

Developmental Considerations in Classification

The second major cross-cutting issue relevant to this review concerns how to reflect important developmental considerations in diagnostic criteria (Pine et al. 2011, Wakschlag et al. 2010). We highlight four considerations that are particularly salient for ADHD, ODD, and CD. First, how do symptoms of a disorder differ from normal behavior at different developmental periods, and do the diagnostic criteria reflect these differences? Symptoms of ADHD, ODD, and CD (e.g., behaviors like inattention, impulsiveness, argumentativeness, aggression) are often found to some degree in typically developing children. Therefore, it is important to determine whether the disorders related to these symptoms reflect deviations in severity from normal development (e.g., greater number or frequency of attentional problems) or whether they reflect deviations in the kind of behavior (e.g., presence of vindictiveness) (Wakschlag et al. 2010). Then, diagnostic criteria must be evaluated to determine if they reflect these departures from typical development.

Second, do the manifestations of a disorder change sufficiently over the course of development to warrant these changes being reflected in the diagnostic criteria? Thus, rather than having a special category for disorders first evident in infancy, childhood, or adolescence, it would be valuable to know how all disorders are typically manifested at different developmental stages and to determine whether sufficient data are available to support having differences in diagnostic criteria at various ages. As discussed in more detail below for ADHD, these differences in criteria can be either in type of symptoms or in the diagnostic threshold across various age groups (Barkley et al. 2008).

Externalizing: a broad dimension of disinhibited behavior that includes conduct problems, aggression, impulsivity, illegal behavior, and substance abuse

Subtypes: within a diagnostic category, distinct subgroups that differ on important symptom dimensions, severity, etiological factors, or response to treatment

APD: antisocial personality disorder

ADD: attention deficit disorder

Third, is the developmental timing of the disorder critical for its classification? For CD, the age at which symptoms first emerge seems to be critical for designating important subgroups of the disorder that differ on severity, course, and etiology (Moffitt et al. 2008). For ADHD, the presence of symptoms prior to adulthood, or even prior to adolescence, seems to be critical for designating the disorder itself and differentiating it from other disorders that may have similar symptom presentations (Kieling et al. 2010). However, as we discuss later, the exact timing of onset needed to validly distinguish ADHD from other disorders is open to question.

Fourth, how do disorders relate to each other over the lifespan? For example, many youths diagnosed with ADHD show an anxiety disorder as an adult (Barkley & Brown 2008). It is critical to determine if this is due to common underlying processes (e.g., problems in emotional dysregulation) that may be manifested differently at different ages or whether anxiety and other signs of emotional distress are a secondary complication of stressors associated with ADHD. As another example, children with CD are at risk for showing antisocial personality disorder (APD) as adults (Moffitt et al. 2008). CD and APD share several indicators (e.g., failure to conform to societal norms, deceitfulness, and aggressiveness). It is important to determine whether CD is a risk factor for APD, and if so, why (e.g., problems in disinhibition, presence of callous-unemotional traits), or whether these disorders are manifestations of the same underlying processes with age-specific manifestations reflected in the different criteria.

These broad issues (i.e., integrating dimensional and categorical approaches to classification, considering developmental issues in diagnostic criteria) are relevant for enhancing the diagnostic criteria for any disorder in the DSM. In the following sections, we discuss these issues in more detail in relation to ADHD, ODD, and CD, as well as focus on other important considerations for improving the diagnostic criteria for these specific disorders.

ATTENTION DEFICIT HYPERACTIVITY DISORDER

Our discussion of ADHD touches on four main issues that have emerged as central for DSM-5. These arise in the context of diagnosis of a disorder, ADHD, that has functioned rather well at least in the developed world, with massive data supporting its validity, both internally and externally, and with extensive research showing that persons with this diagnosis respond positively to a number of different types of treatment (Barkley 2002). Further, there is evidence that this diagnosis is frequently used by clinicians. Thus, until breakthroughs in understanding pathophysiology are in hand, the diagnosis of the disorder likely does not require major changes. Yet some key issues persist that, if addressed, could improve the usefulness and accuracy of the diagnosis for both research and clinical practice.

What Is the Core Disorder?

The precursors of contemporary ADHD were syndromes termed “minimal brain dysfunction,” “minimal brain damage,” and “hyperkinetic reaction.” It is important to note that in those early definitions, the syndrome that was studied included a broad set of problems, often including learning problems, motor and coordination problems, and even sleep problems. By the early 1960s, the syndrome in use had become too overinclusive to enable effective diagnosis, treatment, or research approaches (Clements & Peters 1962). In DSM-III and then DSM-IV, decisions were made to identify a core element of ADHD and to break off the learning and motor disorders into separate categories. In 1980, DSM-III adopted the term attention deficit disorder (ADD), reflecting a conceptual belief that the primary or core dysfunctions in this syndrome had to do with attention (particularly, problems in vigilance and sustained attention).

DSM-III proposed three behavioral dimensions (i.e., inattention, hyperactivity, and impulsivity) in its definition of ADD (APA 1980).

All three dimensions needed to be present for ADD with hyperactivity disorder. The DSM-III included a second type of ADD, called ADD without hyperactivity, in which the child did not have significant levels of hyperactivity. However, this group could have impulsivity, and specific symptom counts for making the diagnosis were not provided. After the publication of the manual, a small literature emerged on children with this nonhyperactive subtype. DSM-III-R (APA 1987) removed this distinction, installed a single behavioral dimension, and renamed the disorder attention deficit hyperactivity disorder, reflecting both problems with the proposed item factor structure and questions about whether the core deficit was truly inattention.¹ DSM-IV retained the name of ADHD but established a modified item list that corrected the invalidity problems in the DSM-III list by using two psychometrically defensible symptom dimensions (i.e., inattention/disorganization and hyperactivity/impulsivity), albeit with only three symptoms of impulsivity in the latter dimension. It restored a subtyping scheme, based on these dimensions: ADHD predominantly inattentive type (ADHD-PI), ADHD predominantly hyperactive-impulsive type (ADHD-PHI), and ADHD combined type (ADHD-C).

By the 1990s, the field was moving fully away from conceptions of inattention as the core of the syndrome. First, interest grew rapidly in the problem of executive functioning, and many experts became interested in ADHD as a dysexecutive syndrome (i.e., characterized by poor planning, disorganization, poor memory, and the like). Second, there was renewed interest in the root of impulsivity, leading to renewed interest both in conceptions of disinhibition (i.e., failure to interrupt a triggered response when needed) and conceptions of temporal discounting of reward (i.e., tendency to put less emphasis on rewards that are not immediate).

¹Although conceptions of ADHD core dysfunction continue to evolve, the name is not likely to undergo further changes. As discovered in the 1980s, name changes result in phenomenal bureaucratic problems involving the legality of prescribing treatments for conditions, drug approvals, and service eligibility.

As DSM-5 has approached, the field has had to consider a plethora of hypotheses about the core dysfunctions at the root of ADHD, including impulsivity (temporal discounting of reward), attention (particularly when framed as executive functioning and vigilance), and variability of responding. Models emerged that proposed there were multiple core problems, putting emphasis on the two-dimension structure (for a review, see Nigg 2006).

However a fundamental finding over the period 1994 to 2010 was that the two-dimensional structure worked very well in that the two dimensions differentially predicted types of impairment, types of comorbidity, and even neuropsychological findings, as exhaustively reviewed by Willcutt et al. (2011). Thus, a strong base of data established that ADHD was at least a two-component syndrome. The two dimensions are variously conceptualized, but can be broadly conceptualized for our purposes as (a) inattention, which includes conceptual features of disorganization and dysexecutive syndrome, and (b) hyperactivity/impulsivity, which includes conceptual features of abnormal reward discounting, social disinhibition and intrusiveness, and emotional dysregulation. This second dimension tends to overlap with oppositional and conduct problems, whereas the first dimension tends to overlap with learning problems.

One controversy has been whether impulsivity should be separated from hyperactivity (as it was in DSM-III), which may be more applicable to adults but would require adding additional items to reflect adult impulsivity. Although the two-dimensional structure of ADHD symptoms has been well established, it is still possible that, if additional items were added, a separate impulsivity dimension would emerge especially for adults and that the assessment of adults would be enriched. However, adding items introduces a host of potential problems: (a) the factor structure might change, rendering obsolete a generation of productive research on the two symptom dimensions, or (b) adults and children would have different diagnostic criteria. This latter possibility might

Dysexecutive syndrome: a clinical syndrome that includes being disorganized, losing things, forgetting things, not following through, and being impulsive that is seen in brain-injured patients and is sometimes used as a shorthand to describe some symptoms of ADHD

be acceptable, but it raises the problematic prospect that an individual who fails to meet criteria at age 17 could suddenly meet ADHD criteria at age 18 despite no change in presentation. That issue would have to either be tolerated or resolved. This topic is considered again below in the discussion of ADHD in adults.

Taken together, there has been a great deal of research considering the core features of ADHD, and it is difficult to apply this large body of research to diagnostic classification. Further, it is clear that much work is still needed in this area. However, the strongest conclusion is that ADHD's core is a two-component structure, including both inattention (with attendant problems in behavioral organization and apparent behavioral problems related to poor executive function) and hyperactivity/impulsivity.

Where Does ADHD Fit in the Conceptual Meta-Structure: Is ADHD a Neurodevelopmental or Behavioral Condition?

The shift from the diagnosis of minimal brain dysfunction to ADD in the 1970s and 1980s signaled to some extent a conceptual shift from ADHD as a neurodevelopmental condition to ADHD as a behavioral condition. As a result, it became increasingly important to clarify whether attention deficits and hyperactivity overlapped or were distinct from conduct problems, aggression, and defiance in the externalizing/disruptive domain of child behavior problems (Hinshaw 1987). By the 1990s and into the 2000s, as we noted previously, studies had begun to explore an empirical meta-structure of DSM disorders that tended to mirror what was earlier discovered using empirical studies of problem lists in children (Achenbach et al. 1989, 1991). That is, one can empirically create a behavioral dimension of externalizing problems that includes a shared latent variable for defiant, aggressive, antisocial, and hyperactive-impulsive behaviors, with a weaker but significant loading for inattention/dysexecutive behaviors. As noted previously, this externalizing dimension is set

against a higher-order internalizing dimension for mood and anxiety disorders/problems. However, a crucial limitation with those studies was that the models tested excluded learning disorders, autism spectrum disorders, motor disorders, and language disorders. Thus, it remains unknown whether ADHD, and inattention in particular, clusters more clearly with learning, motor, and social problems than with conduct problems and aggression.

However, one study to consider this question found that ADHD clustered strongly with ODD but also with autistic symptoms, motor coordination, and reading problems and that sibling cross correlations loaded better for autistic, motor, and reading problems than for behavior problems (Couto et al. 2009). Indeed, when one considers the overlap among symptoms of these disorders, it is striking that ADHD symptoms have a substantial phenotype overlap with motor delays (Fliers et al. 2009, Piek et al. 1999, Pitcher et al. 2003, Rasmussen & Gillberg 2000); autism spectrum disorders (ASDs), in that 50% of children with ASD are hyperactive, and about 20% of children with ADHD have sufficient social oddity to raise questions about ASD overlap (Reiersen et al. 2008); cognitive problems (Nigg et al. 2005); and learning disabilities (Willcutt et al. 2010)—with which there may be genetic overlap (Couto et al. 2009, Loo et al. 2004, Willcutt et al. 2002). Additionally, at a group level, ADHD is associated with early-appearing alterations or immaturities in neural development (Castellanos et al. 2002, Shaw et al. 2007). These early brain alterations are not explained by differences in the structure of the genome (Castellanos et al. 2003) and so may be due to stochastic epigenetic changes or to environmental influences on gene expression. Further, theories of ADHD increasingly emphasized its early developmental progression in relation to unfolding self-regulation (Nigg et al. 2006). Although some of these correlates may also be applicable to CD, their conceptual and empirical associations with ADHD are quite compelling.

In all, despite heavy overlap among both behavioral and developmental problems of

varying kinds, distinguishing these domains in some way is also necessary to begin to isolate causal structure and to specify particular domains of clinical response. To that end, it appears that ADHD indeed has important and substantial overlap with CD and ODD, particularly when one emphasizes the hyperactive/impulsive component of ADHD. However, the syndrome overall, and the inattention dimension in particular, appear to share overlaps with developmental problems. Thus, on balance, ADHD is best thought of as a neurodevelopmental condition rather than primarily or solely a behavioral condition. That is, it is a condition rooted in early developmental alterations in the nervous system that likely have roots in prenatal, neonatal, or very early postnatal development and that are associated often with a spectrum of developmental setbacks or delays. It thus makes conceptual and empirical sense to group it in the meta-structure with ASDs, learning disorders, language delays, and related developmental conditions.

Should ADHD Include Subtypes? If So, Which Ones?

As noted above, the DSM-III created a subtype called ADD without hyperactivity. This was done to reflect the experience of some clinicians that some children seem to present with inattention but without hyperactivity. DSM-IV restructured this subtype, gave it an operational definition, and added an additional subtype: ADHD-PHI. An important point of controversy subsequently was that the DSM-IV definition of ADHD-PHI seemed to differ conceptually from the definition of attention deficit disorder without hyperactivity used in the DSM-III, in that children with ADHD-PHI could have above-average levels of hyperactivity (or hyperactivity/impulsivity) so long as they did not reach the threshold of six symptoms (Diamond 2005, Milich et al. 2001). Some experts also believed that the inattentive children were characterized by sluggish tempo rather than high activity (McBurnett et al. 2001, Penny et al. 2009). Items reflecting

sluggish cognitive tempo were excluded from the DSM-IV symptom list because they failed in field trials to add predictive validity to the disorder (Frick et al. 1994), and ongoing research has not yet convincingly supported their use, at least in children (Todd et al. 2004).

Additional questions arose in the mid-2000s, when two groups reported that ADHD subtypes were very unstable over time (Lahey et al. 2005, Todd et al. 2008). This was validated in a meta-analysis of five data sets by Willcutt et al. (2011). Some instability is to be expected developmentally. For example, children normatively become less hyperactive as they develop, so heterotypic continuity should provide for some transition from combined type (ADHD-C) to ADHD-PI, or from ADHD-PHI to no-ADHD. Further, problems with inattention and executive problems should become more apparent with age, as cognitive demands steadily increase on children, and finally outpace the abilities of children who are lagging in their development of executive functioning or attentional control. Thus, one might also normatively expect later onset of ADHD-PI than ADHD-C. Finally, one might also expect some changes in subtype due to measurement error: children assessed as having five symptoms of inattention and six symptoms of hyperactivity at time 1 could easily have six and six at time 2, creating an apparent “change” of subtype due solely to measurement imprecision.

Unfortunately for such defenses of the subtypes, Willcutt et al. (2011) documented that the changes in subtype assignment from year to year do not appear to be explained solely by these “expected” sources of subtype variation. A meaningful percentage of children change from eight or nine symptoms to two or three symptoms, and an important minority of children change from ADHD-PI to ADHD-C or ADHD-PHI (Lahey et al. 2005, Lahey & Willcutt 2010). Further challenges to the subtype conception arose from failure to find unique neuropsychological or cognitive problems associated with ADHD-PI versus ADHD-C. Rather, the literature has tended to find that cognitive problems accrue to ADHD-PI and

Sluggish tempo: refers to a tendency of some children with ADHD to appear to have low energy, to move slowly, to have slow reaction times, and to think slowly

ADHD-C, but less so to ADHD-PHI (Willcutt et al. 2011). Moreover, when subtypes do differ, it has almost invariably been the case that ADHD-C has worse cognitive problems than ADHD-PI, consistent with a simple severity heuristic. That is, ADHD-C by definition features more symptoms on average than ADHD-PI, so one should expect more problems in any correlated domain that is not specific to a subtype configuration. A parsimonious conclusion might be that ADHD-C is simply a more severe condition than ADHD-PI. Thus, there are substantial grounds to weaken the DSM-IV subtype logic to remove the impression that these are fixed, stable types.

One possible solution is to simply eliminate subtypes. However, there are two problems with such an approach. First, conceptually, children with ADHD are heterogeneous in their presentation, and it is important to consider how this might be properly conveyed in a clinical manual. Second and perhaps more compelling is that important gaps remain in this literature. Notably, there are virtually no neurobiological (genetic or neuroimaging) data comparing ADHD-PI and ADHD-C (or ADHD-PHI). Those studies that do exist have been too small (low powered) to enable much to be concluded. However, one large study reported that functional connectivity patterns in the brain differed in ADHD-C and ADHD-PI (Shannon et al. 2011). A critical need in future research will be to further test such differences between subtypes, including their temporal stability over time.

A further gap in the available research on subtypes is that some definitions that classify children without hyperactivity more rigorously than is done by the DSM-IV criteria, such as defining children who do not show even minimal levels of hyperactivity or who are perhaps sluggish and inattentive, may form a distinct etiological group of children without hyperactivity compared to those with ADHD-C. Volk et al. (2009) reported that a natural latent class, identified through latent class analysis in a large population sample, was well-identified using a simple cutoff of two or fewer symptoms

of hyperactivity impulsivity and five or more symptoms of inattention-disorganization.² Following up on this finding, Goth-Owens et al. (2010) reported that children with ADHD-PI, but restricted to two or fewer symptoms of hyperactivity/impulsivity—and, importantly, who never met ADHD-C by history according to a clinician-administered semistructured interview of a parent—formed about half of the ADHD-PI group. That group, unlike the group with three to five symptoms of hyperactivity/impulsivity, had slower output speed on two simple and widely used clinical measures—Trailmaking A and Stroop Color and Word naming. Carr et al. (2010), using adolescents from the same sample, found that attentional blink response (a measure of early-stage gating of information) was atypical in children with ADHD-PI with two or fewer symptoms of hyperactivity/impulsivity compared to children with ADHD-C (and typically developing children). Once again, the effect was not apparent when children with three to five symptoms of hyperactivity/impulsivity were included. Derefinko et al. (2008) likewise found a distinct pattern of cognitive results in a subgroup of ADHD-PI youths with sluggish cognitive tempo. In a meta-analysis of family inheritance studies, Stawicki and colleagues (2006) found that relatives of children with ADHD-PI tended to have both ADHD-PI and ADHD-C in greater-than-expected rates, whereas relatives of children with ADHD-C had only ADHD-C at elevated rates, suggesting that ADHD-PI includes two distinct genetic types.

Thus, there seems sufficient reason to further consider whether a subgroup of youths with ADHD-PI shows an etiologically distinct disorder when symptoms of hyperactivity/impulsivity are very low or when indicators of

²Note that in DSM-IV field trials, five symptoms of inattention was the empirically best cut point to identify ADHD, but the cut point was set to six to ease clinical use. Thus one might expect clinical assignment errors at five symptoms of inattention; interestingly, the Todd et al. (2008) analysis also concluded that five symptoms of inattention was the appropriate class cut off.

sluggish tempo are present. However, it is unknown whether such children would evidence stability of presentation over time or whether such a putative subtype would also be quite temporally unstable, like the DSM-IV subtypes.

A further and important complication is that the major review by Willcutt et al. (2011) compiled evidence on correlates of symptom domains of inattention-disorganization versus hyperactivity/impulsivity. It was very striking that across a large number of studies, essentially all the distinct correlates of putative subtypes are explainable by varying levels of the two symptom dimensions. This picture holds for putative ADD without hyperactivity, based on most studies conducted in the 1980s and 1990s on the prior DSM-III construct, as well as for ADHD-PI. That dimensions can account for subtype findings suggests that subtypes are little more than helpful shorthands for capturing variation on the dimension rather than being entities reflecting true differences in etiology, course, or treatment need. Thus, despite some encouraging findings from recent experimental studies, there is a need for further evidence as to configural uniqueness and temporal stability before one could be very confident in a putative new “restrictive inattentive” subtype. In the end, it is premature to firmly recommend a new subtype, yet it does seem important to encourage further work on this possibility.

For the moment, then, the most parsimonious picture from the available research is that the two symptom dimensions of ADHD are sufficient to capture relevant clinical features,

and subtypes do not add clear additional information. However, with regard to clinical utility, it is useful to have a shorthand that indicates that a child is currently presenting with mostly inattentive or combined/hyperactive symptoms. Therefore, at present the best solution is to weaken the designation of subtypes by defining them as presentations, because they appear to reflect the child’s current clinical profile rather than a true subtype. However, it is also advisable to add a fourth presentation, for restrictive inattentive, that ensures very low levels of hyperactive symptoms, to stimulate research on children who present in that manner. **Table 1** summarizes these four presentations along with a proposal for adults, discussed next.

Developmental Considerations: ADHD in Preschool and Adults

Most data supporting the validity of the diagnosis of ADHD pertain to school-age children (and then mostly to boys and mostly to Caucasian/European boys). Developmentally, however, ADHD emerges often in preschool, and there is a need for more diagnostic precision to evaluate children in preschool who are on a trajectory of stable ADHD. The DSM-IV criteria can be used in preschoolers (Lahey et al. 2004), but research and treatment validity remain limited, and many in the field would like to see refinement of the diagnostic criteria for preschoolers. Doing so, however, is beyond the limited resources of the current DSM-5 effort, as it would likely require a special

Table 1 Proposed methods for specifying current presentations of attention deficit hyperactivity disorder

| Current presentation | Number of symptoms | Number of symptoms |
|---------------------------------|--------------------|---------------------------|
| | Inattention | Hyperactivity/impulsivity |
| Children | | |
| Combined presentation | ≥6 | ≥6 |
| Primarily hyperactive/impulsive | <6 | ≥6 |
| Primarily inattentive | ≥6 | <6 |
| Restrictive inattentive | ≥6 | ≤2 |
| Adults | | |
| All | ≥4 and/or | ≥4 |

preschool field trial and considerable work on item evaluation, integration with nascent efforts to develop taxonomies for toddlers, and many ethical considerations.

Even more pressing, however, has been the rise in clinical demand for services for adults with ADHD. Although the literature on ADHD in adults dates back more than 30 years (Wood et al. 1976), the presentation of adults was not well studied until after DSM-IV was developed. Several issues have concerned experts about adults with ADHD: (a) some symptoms seem to clearly be inapplicable to adults (e.g., runs about or climbs on things); (b) the cut point of six symptoms may underidentify adults with impairment needing treatment; and (c) the features that cause impairment in adults may be different from those that cause impairment in children, so that the current symptom list fails to efficiently capture the adult manifestation. On this last point, there have been calls to include more impulsivity items in the DSM criteria as well as more items directly reflecting presumed executive functioning (Barkley et al. 2008), perhaps to replace the weakest items in the current item set.

Resolving all of these issues at once would require examination of an alternative item set for adults. However, even with the current item set, the issue of cut point requires reflection. Because ADHD symptoms show some normative variation with development into adulthood, the DSM-IV cut points could underidentify adults (Barkley et al. 2002). In a sample of adults first identified with ADHD in childhood and evaluated at age 27 years, Barkley et al. (2008) found that a cut point of four or more symptoms was most efficient in distinguishing adults with and without ADHD—although discrimination from adults with other psychiatric disorders was not reliable at that point. In a sample of 121 adults first identified as having ADHD in children and assessed at a mean age 41 years, Mannuzza et al. (2011) found that 97% of those with four or more symptoms of inattention and 93% of those with four or more symptoms of hyperactivity/impulsivity had clinically significant impairment; 100%

of those with five or more symptoms had clinically significant impairment. Adults with other psychiatric disorders were not examined. Thus, although the evidence is quite limited, two studies suggest that, using the DSM-IV symptoms list, a cut point of four or five symptoms may be more appropriate for adults than the current six symptoms. A change in cut point might raise the number of adults formally meeting criteria, but doing so may be more accurate, and retention of a requirement that adults have onset during childhood would be expected to limit the expansion of cases.

With regard to adding to or replacing the existing DSM-IV items for adults, there is much to consider. Several empirically based alternative sets for adults have been generated (Achenbach 2011, Barkley et al. 2008, Conners et al. 1999). If the symptom list were changed, either by rewriting the symptoms or by adding more impulsivity or executive function items, cut points would have to be carefully reevaluated in adequate data sets. As for particular item sets, Barkley et al. (2008) presented preliminary data suggesting that alternative items they labeled as tapping everyday executive functioning could be used to create a shorter symptom list for adults. Following up on this, Kessler et al. (2010) assessed 131 second-stage respondents from the National Comorbidity Survey data set using 12 additional executive functioning items and two additional impulsivity items along with the DSM-IV items. By adding these items to the DSM-IV symptom list, they were able to create a three-factor set of items: executive functioning (planning and disorganization; three DSM items and six new executive items), inattention/hyperactivity, and impulsivity. The best predictive item set for adults with ADHD was an item set that included two existing DSM-IV symptoms (makes careless mistakes; difficulty sustaining attention) and two new executive items (difficulty prioritizing work; cannot complete tasks on time). They noted that the executive functioning symptom factor was specific to ADHD and did not load on other DSM-IV disorders, whereas inattention, hyperactivity, and impulsivity did, suggesting additional benefit.

Taken together, these studies, although each was small with important limitations, converge on a conclusion that diagnostic criteria for adults with ADHD can be improved. At minimum, the diagnostic threshold should be reevaluated in new field trials, and likely changed to four or five symptoms. Going beyond that, consideration should be given to new field trials that can examine alternative, perhaps smaller, item sets that can be used to diagnose ADHD in adulthood. Such a change would mean that adults and children with ADHD have different symptoms sets; that would introduce complex challenges for research and practice, but might in return yield more than sufficient return in clinical benefits and new discoveries about the nature of this disorder in adulthood.

Defining Age of Onset for ADHD

The DSM-IV specified a minimum age of onset of 7 years for ADHD. This approach is consistent with seeing ADHD as a developmental condition, which should be present in some form early in life, to differentiate it from other conditions with similar symptom presentations. However, in practice, this cutoff has not been supported by research. Specifically, children identified as having symptoms that emerge after age 7 have the same profile, course, impairment, severity, treatment response, and neurobiological findings as those identified as having onset prior to age 7 (Kieling et al. 2010). Part of the problem, particularly when evaluating onset retrospectively in older children or adults, is that age of onset cannot be reliably recalled by informants (Moffitt et al. 2008). Another part of the problem is that ADHD symptoms may exist but may not be sufficient to cause impairment until later in life, confounding efforts of clinicians or informants to track the history of the disorder. Prospective studies suggest that nearly all persons identified with ADHD over the lifespan could have been identified by age 12 to 14 on the basis of the symptoms they showed at that time (Kieling et al. 2010).

Such findings clearly suggest that the current use of age 7 as the minimum age of onset

lacks validity. Less clear is the most appropriate alternative. One might argue that no age-of-onset criteria should be included, but this raises the danger that ADHD as a developmental condition will be lost entirely and that it will not be well differentiated from recent onset reactions in older individuals. Yet clearly any age-of-onset criteria is an arbitrary guidepost, meant to remind clinicians that the condition should have onset relatively early in development. Raising the age of onset makes sense in light of the clear lack of validity to the age 7 onset, but eliminating age of onset entirely raises additional problems. The best solution appears to be setting age of onset to an intermediate, though also arbitrary, point such as prior to “onset of puberty” or “age 12.”

OPPOSITIONAL DEFIANT DISORDER

Since the publication of DSM-III (APA 1980), the manual has included two diagnoses that focus on conduct problem behaviors and that form the disruptive behavior disorders (DBDs): oppositional defiant disorder (ODD) and conduct disorder (CD). In the most current DSM (APA 2000), the key components to the ODD diagnostic criteria are a recurrent pattern of negativistic, defiant, disobedient, and hostile behavior toward authority figures that persists for at least six months (APA 2000).

A significant number of factor analyses have supported the distinction between the symptoms that form the diagnostic criteria for ODD and the antisocial and aggressive behaviors that form the criteria for CD (Frick et al. 1993, Lahey et al. 2008). However, ODD is often considered as a developmental precursor to CD (Moffitt et al. 2008). That is, for a significant number of children who develop CD, ODD often emerges first, followed by the onset of the more severe CD symptoms. Furthermore, a review of the 25 epidemiological studies conducted in 16 different countries found very consistent prevalence rates for ODD across geographic regions, supporting the cross-cultural validity of the disorder (Canino et al. 2010).

DBD: disruptive behavior disorder

Most importantly, ODD in childhood has been shown to predict problems in adjustment in adolescence (Loeber et al. 2009) and adulthood (Rowe et al. 2010).

Is ODD a Useful Diagnostic Construct?

Although the diagnosis of ODD has been included in the DSM since 1980, significant concerns have been raised about its usefulness as a diagnostic construct over the ensuing decades. Moffitt et al. (2000) summarize two of the major concerns that have been expressed about this disorder. First, the oppositional and argumentative behaviors that form the criteria for this disorder are commonly displayed in normally developing children, especially at certain developmental periods, such as early in preschool years (i.e., the terrible twos) and in adolescence (Wakschlag et al. 2007). Second, ODD and the behaviors that form the criteria for this disorder are frequently comorbid with a host of other disorders (Rowe et al. 2002). As a result, it is not clear if ODD is simply a non-specific marker for problems in adjustment or if it is an indicator of a meaningful and unique clinical construct. In short, there are concerns that ODD overpathologizes a normative behavior pattern and that, unless it is accompanied by another disorder (e.g., ADHD, CD), it is transient and benign and should not be considered as a separate disorder.

Given these concerns, it is important to determine whether ODD predicts problems in adjustment independent of the various comorbidities that are often associated with it. Otherwise, eliminating this disorder from the DSM would be supported. Although a significant amount of research has been conducted on children with ODD, the majority of these studies have failed to consider the disorder separate from possible co-occurring problems in adjustment. A few studies have tested the incremental utility of ODD in predicting problems in adjustment, controlling for the most common co-occurring disorders (i.e., ADHD and CD). Controlling for co-occurring

ADHD, children with ODD are more likely to show greater levels of CD, substance use, and emotional disorders concurrently (Garland & Garland 2001, Harpold et al. 2007) and predictively (Biederman et al. 2008a,b; Burke et al. 2010). Importantly, this incremental predictive utility of ODD relative to ADHD is found in children as young as ages 3 to 5 (Gadow & Nolan 2002). Also, controlling for CD, children with ODD are more likely to show emotional disorders (Loeber et al. 2009). For example, in an epidemiological sample of 1,420 children and adolescents ages 9 to 16, ODD predicted risk for diagnoses of anxiety and depression in adulthood (ages 19 to 21) even in the absence of CD (Rowe et al. 2010).

This research suggests that ODD does have some important diagnostic utility, even controlling for other disorders, particularly ADHD and CD. Thus, it supports maintaining the diagnosis of ODD as a separate clinical entity. Further, this research also has some important implications for how the relationship between ODD and CD is conceptualized. Specifically, the fact that ODD predicts problems in adjustment, even controlling for the presence of CD, suggests that ODD provides some important diagnostic information (e.g., risk for emotional disorders) not provided by the diagnostic criteria for CD.

The Relationship Between ODD and CD

The available research on the relationship between ODD and CD can be summarized by several findings. First, ODD and CD often share a number of dispositional and environmental risk factors, and unique risk factors to the two disorders are rarely found (Boden et al. 2010, Rowe et al. 2002). Second, as noted previously, ODD indicates risk for CD, particularly for early onset of CD. In a longitudinal study of children, half of whom met criteria for ADHD, 71% to 78% of children who developed CD between the ages of 4 and 9 met criteria for ODD earlier in development, whereas the rate was only 30%

of those who met criteria for CD after age 10 (Burke et al. 2010). Third, despite this developmental relationship between ODD and CD, a large percentage of children with ODD do not have CD, nor do they go on to develop CD (Maughan et al. 2004, Rowe et al. 2002). Similarly, only a minority of children with CD have a diagnosis of ODD, and the proportion of youths with CD without ODD increases from childhood to adolescence (Burke et al. 2010).

Taken together, this research does not support the current diagnostic criteria for ODD used by DSM-IV, which exclude a diagnosis of ODD when the person meets criteria for CD. Specifically, the presence of ODD may designate a group of children with CD who have problems with emotional regulation (Frick & Morris 2004, Lahey & Waldman 2003) that may place them at risk for the development of emotional disorders (Rowe et al. 2010). Thus, the presence of ODD seems to provide important additional information to the diagnosis of CD.

Recognizing the Heterogeneity in ODD Symptoms

Despite this link to emotional disorders, ODD is often considered a behavioral disorder. This is reflected in the name given to it, which focuses on the oppositional and defiant behavioral features. However, the symptoms of the

disorder include a number of indicators of negative affect or, more specifically, anger and irritability. Several recent factor analyses have supported a multidimensional conceptualization of the criteria for ODD (Burke 2011, Burke et al. 2010a, Rowe et al. 2010). Although the items on the factors vary somewhat across the samples, these analyses are consistent in suggesting that the angry/irritable mood dimension forms a separate factor from the defiant/headstrong behavior dimension. What is less clear from these analyses is the appropriate placement of the symptom “is often spiteful and vindictive,” which does not consistently load with the other symptom dimensions (Burke et al. 2010a, Rowe et al. 2010) and that may be more related to the severe conduct problems associated with CD (Stringaris & Goodman 2009a). Thus, on the basis of this research, the symptoms of ODD can be grouped into the three dimensions described in **Table 2**.

Although the symptoms of ODD form separate factors, it is important to note that these dimensions are highly correlated, with correlations ranging from 0.62 to 0.78 (Stringaris & Goodman 2009a). These correlations suggest that a large number of youths high on one dimension would also be elevated on another. This strong association among symptoms supports maintaining them within a single diagnostic construct. Importantly, cluster analyses of these domains suggest that it is not

Table 2 Dimensions of oppositional defiant symptoms

| |
|--|
| Angry/irritable mood |
| Often loses temper |
| Is often touchy or easily annoyed by others |
| Is often angry and resentful |
| Defiant/headstrong behavior |
| Often argues with adults |
| Often actively defies or refuses to comply with adults' request or rules |
| Often deliberately annoys people |
| Often blames others for his or her mistakes or misbehavior |
| Vindictiveness |
| Is often spiteful or vindictive |

Note: Symptom clusters are based on the factor analyses of Burke (2011), Burke et al. (2010), and Rowe et al. (2010).

Pervasiveness: the number of settings in which symptoms of disorder are present; symptoms can be situationally specific or present across many different settings

uncommon for children to show significant levels of the defiant/headstrong dimension without the angry/irritable mood, but it is rare to show the mood symptoms without the behavioral problems (Burke 2011).

The available research does support the importance of reflecting the different symptom dimensions within the diagnostic criteria, as illustrated in **Table 2**. For example, in a cross-sectional study of 18,415 participants (ages 5 to 16) in a national mental health survey in the United Kingdom, all three dimensions of ODD were related to CD (Stringaris & Goodman 2009a). However, the angry/irritable dimension was also related to emotional disorders, the defiant/headstrong dimension was related to ADHD, and the spiteful/vindictive symptom was related to indicators of callous and unemotional traits. Similar divergent predictions from the different ODD dimensions have been found longitudinally, with most studies finding that all three dimensions predict risk for later CD, but the angry/irritable dimension specifically predicts risk for later emotional disorders (Burke et al. 2010a, Rowe et al. 2010, Stringaris & Goodman 2009b). These results suggest that the angry/irritable dimension of the ODD symptoms largely accounts for the findings that ODD is related to and predictive of emotional disorders. Further, they support the need to reflect the multidimensional nature of the ODD symptoms in the criteria for this disorder.

The Importance of Pervasiveness as an Indicator of Severity

A final important issue in evaluating the current diagnostic criteria for ODD is whether and how to reflect the pervasiveness of the symptoms. That is, the current criteria for ODD do not require that the symptoms be present in more than one situation, and as a result, could be present only at home. This is of concern because, again, it could result in labeling behaviors that are developmentally normative as pathological (Wakschlag et al. 2007) and risk labeling a child who has a problem within the

parent-child relationship as a child with a mental disorder (Moffitt et al. 2008).

Unfortunately, the research directly addressing this issue is limited. However, several findings argue against requiring a pervasiveness criterion for meeting the diagnostic threshold. First, the current criteria for ODD do not require the symptoms of ODD to be present in more than one situation, and despite this, a large number of studies have shown that ODD predicts problems in current and future adjustment. Second, when ODD is reported by parents alone, it is associated with problems in adjustment, albeit problems are not as severe as when ODD is reported by multiple informants (Drabick et al. 2007). Third, Youngstrom (2011) provided secondary data analyses on 292 youths who were diagnosed with ODD using structured interviews and who were drawn from consecutive referrals between the ages of 5 and 18 to an outpatient community mental health service. The structured interview specifically assessed whether the impairments associated with ODD were present in three different contexts: at home, at school, and with peers. Of those meeting criteria for ODD, 11% reported impairment only at home, 27% reported impairment in two settings, and 62% reported impairment in all three settings. Most importantly, those who reportedly only had impairments at home still showed significant problems in adjustment, albeit not as significant as those with impairments in two settings. Furthermore, those impaired in two settings showed fewer problems in adjustment than those impaired in all three settings.

Thus, more research is clearly needed to determine whether a diagnosis of ODD should be limited to those who show impairments in more than one setting, but the available evidence does not support this requirement at present. However, the available research does suggest that the level of pervasiveness of the ODD symptoms may be an important indicator of the severity of the disorder. In support of this possibility, Youngstrom (2011) reported that the number of settings in which the symptoms of ODD were present predicted problems in adjustment,

even controlling for the number of ODD symptoms that were present. Similarly, Wakschlag et al. (2007) observed preschool children ages 3 to 5 in three interactional contexts: two with a parent and one with an experimenter. It was not unusual for children with ODD to show problems in only one setting; however, those who showed problems in more than one setting had a more severe behavioral disturbance.

CONDUCT DISORDER

The second DBD diagnosis is conduct disorder (CD). CD is defined as a repetitive and persistent pattern of behavior that violates the rights of others or in which major age-appropriate societal norms or rules are violated (APA 2000). The symptoms of the disorder fall into four main dimensions:

- aggression to people and animals,
- destruction of property,
- deceitfulness or theft, and
- serious violations of rules.

CD is an important psychiatric disorder for a number of reasons. Specifically, it is highly related to criminal and violent behavior (Frick et al. 2005). Further, it is associated with problems in adjustment across the lifespan. This includes mental health problems (e.g., substance abuse), legal problems (e.g., risk for arrest), educational problems (e.g., dropping out of school), social problems (e.g., poor marital adjustment), occupational problems (e.g., poor job performance), and physical health problems (e.g., poor respiratory function) (Odgers et al. 2007, 2008). Even in a sample of young children (ages 4 and 5), CD predicted significant behavioral and educational difficulties five years later (Kim-Cohen et al. 2009).

Thus, the predictive utility of the diagnosis of CD, as it is currently defined, has been well established. Furthermore, there has been a significant amount of research on the social and biological causes of the disorder, which has led to a number of comprehensive causal models to explain its development (Frick & Viding 2009, Moffitt 2006). Perhaps the major issue in

advancing classification of this disorder is in determining how best to capture the great heterogeneity of persons with the disorder. Specifically, children and adolescents with CD can differ greatly on the course of the disorder and in the potential causal processes leading to the disorder. As a result, a number of different methods have been proposed for classifying important subgroups of youths with CD.

The Importance of Age of Onset

One method for subtyping children with CD that has received substantial support from research (Frick & Viding 2009, Moffitt 2006) is the distinction between those whose CD symptoms emerge prior to adolescence (i.e., childhood onset) and those in whom the onset of CD symptoms coincides with the onset of adolescence (i.e., adolescent onset). The extensive research supporting this distinction can be summarized by a few key points. First, there are important differences in the life-course trajectory of the two groups. That is, children in the childhood-onset group often begin showing mild conduct problems as early as preschool or early elementary school, and their behavioral problems tend to increase in rate and severity throughout childhood and into adolescence. Furthermore, the childhood-onset group is more likely to show aggressive behaviors in childhood and adolescence and is more likely to continue to show antisocial and criminal behavior into adulthood (Moffitt et al. 2002, Odgers et al. 2008). Second, childhood-onset conduct problems seem to be more strongly related to neuropsychological deficits (e.g., deficits in executive functioning), cognitive deficits (e.g., low intelligence), and temperamental/personality risk factors (e.g., impulsivity and problems in emotional regulation) (Frick & Viding 2009, Moffitt 2006). This group also is more likely to come from homes with greater family instability, with more family conflict, and with parents who use less-effective parenting strategies (Frick & Viding 2009). When children within the adolescent-onset group differ from children in the childhood-onset group,

Life-course trajectory: the phenomenology of symptoms of a disorder across development; it includes the stability of the symptoms and potential changes in their manifestations (i.e., heterotypic continuity) across various developmental stages

it is in showing higher levels of rebelliousness and being more rejecting of conventional values (Dandreaux & Frick 2009, Moffitt et al. 1996).

The different outcomes and risk factors for the two subtypes of antisocial individuals have led to theoretical models that propose very different causal mechanisms operating across the two groups. For example, Moffitt (2006) has proposed that children in the childhood-onset group develop their problem behavior through a transactional process involving a difficult and vulnerable child (e.g., impulsive, with verbal deficits) who experiences an inadequate rearing environment (e.g., poor parental supervision, poor-quality schools). This dysfunctional transactional process disrupts the child's socialization, leading to poor social relations with persons both inside (e.g., parents and siblings) and outside the family (e.g., peers and teachers). These disruptions lead to enduring vulnerabilities that can negatively affect the child's psychosocial adjustment across multiple developmental stages. In contrast, children in the adolescent-onset pathway have problems that are more likely to be limited to adolescence and are related to fewer risk factors. Thus, this group is conceptualized as showing an exaggeration of the normative process of adolescent rebellion (Moffitt 2006). Given that their behavior is viewed as an exaggeration of a process specific to adolescence and not due to an enduring vulnerability, their antisocial behavior is less likely to persist beyond adolescence. However, they may still have impairments that persist into adulthood due to the consequences of their adolescent antisocial behavior (e.g., a criminal record, dropping out of school, substance abuse) (Moffitt & Caspi 2001).

Thus, this research provides strong support for the clinical and theoretical importance of the childhood- and adolescent-onset distinction. However, there are several important limitations in this method of subtyping. First, it is not firmly established what should be the exact age to differentiate childhood- and adolescent-onset groups. In an early test of the differential predictive utility of different age cut-offs, Robins (1966) found that youths who

were 11 years or younger at the onset of their serious conduct problems were more than twice as likely to be diagnosed with APD as adults. Since this study, cut-offs for defining childhood-onset have ranged from age 10 (APA 2000) to 14 (Patterson & Yoerger 1997, Tibbetts & Piquero 1999) for the onset of the first serious conduct problem. This difficulty in defining a clear cut point for the age of onset has led some researchers to suggest that this distinction should be more dimensional than categorical (Lahey et al. 1999). Another problem with establishing the age of onset for CD relates to difficulties in persons' retrospective recall of past behaviors, especially for older adolescents, which makes it difficult to pinpoint accurately the age at which a child first showed severe conduct problems (Moffitt et al. 2008). Thus, additional research is needed to refine the most appropriate methods for defining important differences in the age of onset of CD symptoms and how best to assess this important developmental dimension. However, at present, there does not appear to be sufficient evidence to support an alternative for the current threshold of symptoms being present before the age of 10 to designate the childhood-onset subtype (APA 2000).

Second, this broad approach to subtyping does not make a distinction within the childhood-onset group about those who are most likely to continue to show problems into adolescence and adulthood (i.e., life-course persistent) and those who show problems that are limited to childhood. Specifically, a significant proportion of children within the childhood-onset group show relatively mild and transient conduct problems (Odgers et al. 2007, Tremblay 2003). Thus, it is important to consider ways to distinguish important subgroups within the broader category of childhood-onset CD.

Integrating Callous-Unemotional Traits into Definitions of CD

One possible method for distinguishing an important subgroup within those youths with

childhood-onset CD may be based on the presence of callous-unemotional (CU) traits, characterized by a lack of guilt and empathy. It is similar to the distinction made within samples of antisocial adults using the construct of psychopathy that has proven to designate an important subgroup of antisocial individuals who show a more severe, violent, and difficult-to-treat pattern of antisocial behavior (Hare & Neumann 2006, Patrick 2006). It is also similar to a distinction made in the DSM-III between those with CD who were “undersocialized,” characterized by “a failure to establish a normal degree of affection, empathy, or bond with others” (APA 1980, p. 45).

Several recent qualitative (Frick & Dickens 2006, Frick & White 2008) and quantitative (Edens et al. 2007, Leistico et al. 2008) reviews have been published showing that CU traits are predictive of a more severe, stable, and aggressive pattern of behavior in antisocial youths. Importantly, there is research showing that children and adolescents with CD who are also high on CU traits exhibit a particularly severe and persistent form of the disorder that is not captured by current diagnostic criteria. Specifically, Kahn and colleagues (2011) reported on a multisite study showing that children with CD who also exhibited significant levels of CU traits displayed higher rates of aggression and cruelty to others than those with CD only. Further, in a national representative sample ($n = 5,326$) of children ages 5 to 16, Rowe et al. (2009) reported that 46% of children with CD had high rates of CU traits, and those with CU traits showed a more stable pattern of CD. Finally, McMahon and colleagues (2010) reported that CU traits in grade 7 predicted adult arrests, number of APD symptoms, and an APD diagnosis two years post high school, even after controlling for number of CD symptoms, number of ADHD symptoms, number of ODD symptoms, and childhood-onset of CD.

In addition to this predictive utility of adding CU traits to the diagnosis of CD, there also appear to be some important implications for causal research. Frick & White (2008) provided a comprehensive review of the research

documenting several emotional, cognitive, personality, and social differences between antisocial youths with and without CU traits. In particular, antisocial youths with CU traits show deficits in the processing of negative emotional stimuli and, even more specifically, deficits in their reactivity to signs of fear and distress in others. They also are less sensitive to punishment cues, especially when a reward-oriented response set is primed, and they show more positive outcome expectancies in aggressive situations with peers. Antisocial youths with CU traits tend to be more fearless and thrill seeking. Finally, the conduct problems of youths with CU traits are less strongly related to dysfunctional parenting practices.

These various differences seem to suggest that the causal factors leading to CD may differ for children with and without significant CU traits. Frick and colleagues (2011) have summarized different neurobiological underpinnings for the two groups of children with CD that may explain many of the emotional and cognitive differences across the two groups. Further, Frick & Viding (2009) have outlined different developmental mechanisms underlying the behavioral problems of children with CD who have significant levels of CU traits (e.g., a fearless and uninhibited temperament affecting conscience development) compared to those with CD who do not show significant levels of CU traits (e.g., problems in emotional regulation, executive functioning deficits). Again, distinctions among these developmental pathways do not seem to be captured well by the current diagnostic criteria for CD. Although children with a childhood-onset to CD show higher rates of CU traits than those in the adolescent-onset group (Dandreaux & Frick 2009, Silverthorn et al. 2001), CU traits seems to designate an important subgroup within the childhood-onset group. For example, within a community sample of 7-year-old twins, CU traits designated a distinct group of children with conduct problems who had substantially higher genetic risk associated with their problem behavior compared to the group with conduct problems but without CU traits (Viding et al. 2005).

CU traits:
callous-unemotional
traits

Perhaps one of the most important reasons for integrating CU traits into the diagnostic criteria for CD is based on research showing differential responses to treatment for youths with and without CU traits. Specifically, Frick & Dickens (2006) reviewed five studies showing that CU traits were associated with poorer treatment outcomes in samples of antisocial youths. However, several more recent studies suggest that children with CU traits may be difficult to treat but that certain types of treatment may still be effective (Kolko & Pardini 2010). For example, Hawes & Dadds (2005) reported that clinic-referred boys (ages 4 to 9) with conduct problems and CU traits were less responsive to a parenting intervention than boys with conduct problems who were low on CU traits. However, children with and without CU traits seemed to respond equally well to the first part of the intervention, which focused on teaching parents methods of using positive reinforcement to encourage prosocial behavior. In contrast, only the group without CU traits showed added improvement with the second part of the intervention, which focused on teaching parents more effective discipline strategies. Waschbusch and colleagues (2007) reported that children (ages 7 to 12) with conduct problems and CU traits responded less

well to behavior therapy alone than did children with conduct problems without CU traits. However, these differences largely disappeared when stimulant medication was added to the behavior therapy. Finally, Caldwell et al. (2006) demonstrated that adolescent offenders with CU traits improved when treated with an intensive intervention that utilized reward-oriented approaches, targeted the self-interests of the adolescent, and taught empathy skills. Specifically, they reported that adolescent offenders high on CU traits who received the intensive treatment were less likely to recidivate in a two-year follow-up period than were offenders with these traits who underwent a standard treatment program in the same correctional facility.

On the basis of this research supporting the predictive utility, theoretical importance, and clinical utility of integrating CU traits in the diagnosis of CD, the DSM-V work group has proposed adding a specifier to the diagnosis of CD that designates youths with significant CU traits (Frick & Moffitt 2010). This proposed specifier is provided in **Table 3**. Although inclusion of this specifier is supported by the available research, several potential problems with this change need to be considered. First, one concern is whether the emotional and interpersonal traits that form the criteria for the specifier can

Table 3 Proposed criteria for a specifier “with significant callous–unemotional traits” to the diagnosis of conduct disorder^a

| |
|--|
| 1. Meets full criteria for conduct disorder |
| 2. Shows 2 or more of the following characteristics persistently over at least 12 months and in more than one relationship or setting. The clinician should consider multiple sources of information to determine the presence of these traits, such as whether the person self-reports them as being characteristic of him or herself and if they are reported by others (e.g., parents, other family members, teachers, peers) who have known the person for significant periods of time |
| <ul style="list-style-type: none"> ■ Lack of remorse or guilt: Does not feel bad or guilty when he/she does something wrong (except if expressing remorse when caught and/or facing punishment). |
| <ul style="list-style-type: none"> ■ Callous/lack of empathy: Disregards and is unconcerned about the feelings of others. |
| <ul style="list-style-type: none"> ■ Unconcerned about performance: Does not show concern about poor/problematic performance at school, work, or in other important activities. |
| <ul style="list-style-type: none"> ■ Shallow or deficient affect: Does not express feelings or show emotions to others, except in ways that seem shallow or superficial (e.g., emotions are not consistent with actions; can turn emotions “on” or “off” quickly) or when they are used for gain (e.g., to manipulate or intimidate others). |

^aFrom Frick & Moffitt (2010).

be reliably assessed by clinicians. This is an important consideration that is being tested by the DSM-V field trials, although there is evidence to suggest that personality traits, such as those included in the proposed specifier, can be assessed reliably through a number of different methods (Clark 2007).

Second, the pejorative connotations associated with the term “callous-unemotional traits” are a cause of concern. Several factors were considered in selecting this descriptive term for these traits. First, although there is no research directly testing the effects of the label “callous-unemotional traits,” there is an empirical literature studying the negative effects of the use of the term “psychopathy” when applied to children and adolescents (for a review, see Murrie et al. 2007). To summarize across studies, the findings indicate that the term “psychopathy” does affect decision-making by professionals (e.g., clinician’s estimation of treatability), but it does not have any more negative effects than using the term “conduct disorder” itself. Thus, it appears that any term used to describe individuals with antisocial traits will become associated with negative connotations. Second, previous attempts to capture CU traits in the DSM attempted to minimize potential stigmatizing effects of the label by using the term “undersocialized.” It is not clear, however, that this term has any less negative connotations, and the lack of clarity in this term led to great variability in how the construct was conceptualized and assessed by researchers and clinicians. Third, there is a danger in using terms that seem to connote a less severe disturbance (e.g., uncaring) for the specifier in an effort to decrease the potential for stigmatization. Such definitions could actually be more harmful by resulting in children and adolescents with less severe disturbances being diagnosed by clinicians.

Should There Be Gender-Specific CD Criteria?

A final important issue that has been debated relative to the diagnosis of CD is whether there should be gender-specific criteria for the

disorder. Specifically, CD is about two to three times more likely in boys than in girls (Moffitt et al. 2001). Thus, there has been some debate as to whether this difference in prevalence reflects true gender differences in the causes of CD (Odgers et al. 2008) or whether these differences reflect problems in the criteria for CD, which may not be as appropriate for girls as for boys (Hartung & Widiger 1998). Two specific potential sources of biases in the criteria for CD have been considered. First, some have questioned whether the threshold for the diagnosis of CD (i.e., three symptoms) is too high for girls and have suggested that gender-specific thresholds should be used (Zoccolillo et al. 1996). Second, others have suggested that the types of symptoms that form the criteria should be broadened to include types of aggressive and antisocial behavior that may be more likely to be exhibited by girls. For example, when girls behave aggressively, they are more likely to exhibit relational forms of aggression (e.g., attempts to harm one’s relationships with others) than physical aggression (e.g., attempts to physically harm others) (Crapanzano et al. 2010).

There is some evidence to support these positions. For example, girls with one or two symptoms of CD show impairments in their functioning, both concurrently (Keenan et al. 2010) and predictively (Messer et al. 2006). Also, a significant number of girls show relational aggression but not physical aggression, yet show impaired social functioning (i.e., bullying) and a number of correlates often associated with CD (e.g., anger dysregulation, impulsivity) (Crapanzano et al. 2010). Despite this supportive research, however, the research basis does not appear strong enough at present to justify developing gender-specific criteria for CD (Moffitt et al. 2008). Although girls with subclinical levels of CD are at risk for current and future impairment, so too are boys with subclinical levels (Moffitt et al. 2001). Further, even in girls, the three-symptom threshold designates a substantially more impaired group than those with either one or two symptoms (Keenan et al. 2010). Finally, in a study of relational aggression in girls, many girls high on relational

Stigmatization:
negative assumptions made about persons with a disorder, such as the probability that they will harm others or their amenability to treatment

aggression were also high on ODD and CD (Keenan et al. 2008). More importantly, those high on relational aggression without either diagnosis were not more impaired than those who were not high on relational aggression.

One additional issue related to the diagnosis of CD in boys and girls relates to the relevance of the current subtypes across gender. A consistent finding in research is that a childhood-onset to CD is much more rare in girls than in boys (Moffitt & Caspi 2001, White & Piquero 2004). However, despite the predominance of adolescent-onset CD in girls, there is evidence that girls with CD show poor outcomes in adulthood and show a large number of the dispositional and contextual risk factors that are more characteristic of childhood-onset antisocial behavior in boys (Frick & Dickens 2006). To reconcile these findings, Silverthorn & Frick (1999) proposed that although a small number of girls may show a childhood onset to their antisocial behavior, and they seem to be very similar to boys with childhood-onset CD, it is more likely for girls with CD to show an adolescent onset to their antisocial behavior, despite having very similar risk factors to boys with childhood-onset CD. They described this as a delayed-onset pathway to CD, to suggest that girls' severe antisocial behavior is often delayed until adolescence, coinciding with biological (e.g., hormonal changes associated with puberty) and psychosocial (e.g., less parental monitoring and supervision, greater contact with deviant peers) changes that encourage antisocial behavior in girls with predisposing vulnerabilities (e.g., CU traits, problems in emotion regulation).

In an initial test of this model, adjudicated adolescent girls who largely showed an adolescent onset to their antisocial behavior also showed high levels of CU traits, problems with impulse control, and a number of other social and temperamental vulnerabilities that were more similar to childhood-onset boys than to adolescent-onset boys (Silverthorn et al. 2001). However, despite this initial positive finding, additional tests of this model have been mixed (Moffitt & Caspi 2001, Odgers et al. 2008,

White & Piquero 2004). As a result of these conflicting findings, the possibility of a delayed-onset trajectory in girls does not appear to currently have the support needed to be integrated into diagnostic classification systems for CD.

SUMMARY AND CONCLUSIONS

The current review first focused on a few general issues that are important in the DSM classification of childhood disorders; namely, the importance of integrating dimensional considerations into the DSM system and the importance of considering developmental issues in the diagnostic criteria. Our first conclusion was that it is unlikely that classification systems like the DSM can completely move away from defining diagnostic thresholds. However, we do conclude that future research on classification should explore ways of integrating dimensional approaches to the diagnostic process, especially approaches that recognize that persons just below the diagnostic threshold of a disorder may require treatment and that within persons with the same diagnosis there may be a continuum of severity. In our discussion of specific disorders, we provide one example of an attempt to recognize this continuum of severity within the diagnostic category of ODD. That is, within children and adolescents with ODD, the more settings in which the child shows the symptoms, the more severe the disorder appears to be.

Another important consideration in taking a more dimensional approach to classification is whether disorders can be better organized or grouped in a way that takes into account common dimensions, such as shared risk factors or common symptom clusters. Again, the disorders that were the focus of the review provide evidence for both the promise and challenge in doing this. For example, there are rather extensive data to suggest that ODD and CD tend to cluster into an overarching externalizing or disinhibited dimension that seems to share substantial genetic influences. However, whereas ADHD is often comorbid with these disorders, this effect is largely carried by hyperactivity/impulsivity and does not apply as

well to inattention. Altogether, the available evidence suggests that it may be better grouped with other developmental disorders.

Another overarching issue that was a focus of the current review is the need to consider developmental issues in the classification of mental disorders. Although often stated, the method for accomplishing that goal is rarely well specified. We articulated several specific ways that this can be done, including having diagnostic criteria reflect:

- how symptoms of a disorder differ from normal behavior at different developmental periods,
- how the manifestations of a disorder change over development,
- important differences in the developmental timing of the disorder, and
- how disorders relate to each other over the lifespan.

Our discussion of the individual disorders of ADHD, ODD, and CD attempted to highlight these issues and how they relate to improving the current diagnostic criteria for these disorders. For example, a key issue in the diagnostic criteria for ADHD is whether the current criteria are appropriate in both symptom type and in the diagnostic threshold for preschool children and adults. As another example, research has indicated that the timing of onset of the first CD symptom seems to be critical for defining important subtypes of the disorder.

Our review also focused on several additional issues relevant to the classification of ADHD, ODD, and CD. Overall, research has generally supported the validity and clinical utility for each these disorders. However, several key issues were highlighted for each disorder that could improve the utility of the disorders for both research and practice.

For ADHD, the key issues all center around how to correctly reflect developmental change and continuity. First, we have pointed out that the age of onset of age 7 for ADHD lacks validity and should be changed. Viable changes include “in childhood,” “12–14” or “12.” We recommend the latter as the simplest, provided

sufficient data can be obtained showing that effects on prevalence will be minimal. Second, ADHD should continue to be understood as a developmental, and not merely a behavioral, condition in light of new and growing evidence that it is associated with early emerging physical, neural, and maturational changes. Progress on etiology and early intervention/prevention will require understanding these early developmental events and stages. Third, on the other end of the developmental range, ADHD in adults has come of age as a clinical and scientific topic, and this should be reflected in the DSM-5 by changes to the criteria that appropriately reflect the syndrome’s presentation in adults. Accumulating sufficient field trial data to support such changes, however, remains difficult. At minimum, the diagnostic cut point should be changed to four or five instead of six symptoms for adults. Finally, and perhaps most complex, subtyping is confounded by developmental instability in symptom presentation of children with ADHD. Until this is better understood, it is recommended that subtypes be reconceptualized as current presentation. Within that framework, room can be made, without creating new disorders, types, or diagnoses, to expand research and recognition of a poorly studied group of children who present as inattentive with no symptoms of hyperactivity, to determine whether they too are simply a current presentation or whether they have distinct trajectories.

For ODD, we reviewed research suggesting that this disorder was highly related to both concurrent and future problems in adjustment, even controlling for co-occurring conditions. This includes predicting risk for anxiety and depressive disorders, even controlling for CD. Thus, these findings do not support the exclusionary criteria currently used for the diagnosis of ODD, in which this disorder is not identified if CD is present. Further, the link between ODD and emotional disorders appears to be largely accounted for by the symptoms of ODD that focus on negative emotionality (i.e., anger and irritability). Such findings suggest that the criteria for the disorder should recognize the

different symptom dimensions that form the disorder. Recognizing that ODD is not just a behavioral disorder but one that also includes problems regulating emotions can help to address confusion about the diagnosis of bipolar disorder in cases in which children show behavioral problems with mood dysregulation problems (Axelson et al. 2011). Future research needs to determine whether the group of children with ODD with severe problems in mood dysregulation and those with only the behavioral symptoms show different causal processes and show different responses to treatment, which could suggest that they should be considered as having different disorders (APA 2010).

Finally, for CD, two additional key issues for diagnostic classification were considered, with different conclusions reached for each. First, a substantial body of evidence supports the predictive and clinical utility for designating children with CD who also show substantial numbers of CU symptoms. This group seems to show a large number of distinct emotional, cognitive, personality, and social factors. As a result, identifying those with CU traits could be important for guiding future research on the different causal pathways that can lead to CD. Second, and in contrast, there have been several theories and some empirical support for considering gender-specific criteria for CD, either in the types of symptoms, in the diagnostic threshold, or in the subtypes of the disorder.

However, the research basis does not appear to be strong or consistent enough to recommend specific changes to the criteria of CD to reflect these gender differences at the present time.

These differing conclusions related to the criteria for CD illustrate one of the greatest difficulties we face when considering what recommendations to make for changing the diagnostic criteria for any disorder. That is, we fully endorse the importance of having research guide potential changes in all cases. Yet this will never result in the complete elimination of subjectivity in the process. At what point is the evidence strong enough to justify recommending changes to the current criteria? How are competing priorities (empirical validity, clinical utility) or competing findings (neuroimaging, treatment response) to be weighted? Guidelines have been proposed for making such judgments (Kendell & Jablensky 2003), but they will remain judgments. Even in this review, our focus on certain issues and the exclusion of others (Burke et al. 2010) could provoke debate by other experts with competing interpretations or recommendations. However, we have noted the scientific basis for our recommendations, and we look forward to similar input from others. If this review spurs further scholarly debate—and submission of new data—about these changes, and others we have not considered, then the entire field will be the richer.

SUMMARY POINTS

1. Consensus is emerging that diagnostic classification needs to begin to move toward integrating categorical and dimensional approaches.
2. Several developmental issues need to be considered in the classification of disorders. These include how symptoms of a disorder differ from normal behavior across development, how the manifestations of a disorder change over development, whether the developmental timing of the disorder is critical for its classification, and how disorders relate to each other over the lifespan.
3. Arguments can be made for classifying ADHD as a behavioral disorder with ODD and CD or as a developmental disorder with learning disabilities, autism spectrum disorders, and movement disorders. The developmental classification on balance best characterizes what is informative about ADHD in the nosology, and it is the recommended assignment.

4. ADHD is a two-factor disorder, and this structure should be retained, but the subtypes should be reconceptualized as current clinical presentation due to their instability.
5. The age 7 age-of-onset requirement for ADHD lacks validity and should be revised.
6. Criteria for ADHD for adults need to be reconsidered in light of recent data; the cut point may be better placed at four symptoms than the current six for adults.
7. Retaining the diagnosis of ODD in the classification of childhood disorders appears to be important because it is associated with current and future impairment, even controlling for common co-occurring conditions.
8. ODD seems to predict risk for later emotional disorders, even controlling for CD, which suggests that the presence of CD should not be an exclusionary criteria for ODD.
9. The emotional symptoms of ODD (i.e., anger and irritability) seem to contribute to the link between ODD and emotional disorders. As a result, the different symptom domains that form ODD should be reflected in the diagnostic criteria for the disorder.
10. The pervasiveness of ODD across settings appears to be an important indicator of the severity of the disorder.
11. The presence of CU traits appears to be important for designating an important subgroup of youths with CD. As a result, the presence of these traits should be incorporated into the diagnostic criteria for CD.

DISCLOSURE STATEMENT

The authors are members of the DSM-V ADHD and Disruptive Behavior Disorders Work Group. However, this review represents the perspective of the authors and does not represent an official position of the DSM-V work group.

ACKNOWLEDGMENTS

We would like to acknowledge our colleagues who served with us on the DSM-V ADHD and Disruptive Behavior Disorders Work Group and with whom we have had numerous discussions that have greatly informed our perspective on these issues: Glorisa Canino, Xavier Castellanos (co-chair), Terrie Moffitt, Luis Rohde, and David Shaffer (chair).

LITERATURE CITED

- Achenbach TM. 2009. Some needed changes in DSM-V: But what about children? *Clin. Psychol. Sci. Pract.* 16:50–53
- Achenbach TM. 2011. *Adult Behavior Checklist*. Burlington, VT: Res. Cent. Children, Youth, Families
- Achenbach TM, Conners CK, Quay HC, Verhulst FC, Howell CT. 1989. Replication of empirically derived syndromes as a basis for taxonomy of child/adolescent psychopathology. *J. Abnorm. Child Psychol.* 17:299–323
- Achenbach TM, Howell CT, Quay HC, Conners CK. 1991. National survey of problems and competencies among four- to sixteen-year-olds: parents' reports for normative and clinical samples. *Monogr. Soc. Res. Child Dev.* 56:1–131
- Am. Psychiatr. Assoc. (APA). 1980. *Diagnostic and Statistical Manual of Mental Disorders*. Washington, DC: Am. Psychiatr. Assoc. 3rd ed.

- Am. Psychiatr. Assoc. (APA). 1987. *Diagnostic and Statistical Manual of Mental Disorders*. Washington, DC: Am. Psychiatr. Assoc. 3rd ed. Rev. ed.
- Am. Psychiatr. Assoc. (APA). 2000. *Diagnostic and Statistical Manual of Mental Disorders*. Washington, DC: Am. Psychiatr. Assoc. 4th ed. Text rev.
- Am. Psychiatr. Assoc. (APA). 2010. *Justification for Temper Dysregulation Disorder with Dysphoria*. DSM-5 Childhood and Adolescent Disorders Work Group. Washington, DC: Am. Psychiatr. Assoc. <http://www.dsm5.org/Proposed%20Revision%20Attachments/Justification%20for%20Temper%20Dysregulation%20Disorder%20with%20Dysphoria.pdf>
- Andrews G, Goldberg DP, Krueger RF, Carpenter WT, Hyman SE, et al. 2010. Exploring the feasibility of a meta-structure for DSM-V and ICD-11: Could it improve utility and validity? *Psychol. Med.* 39:1993–2000
- Axelson DA, Birmaher B, Findling RL, Fristad MA, Kowatch RA, et al. 2011. Concerns regarding the inclusion of temper dysregulation disorder with dysphoria in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. *J. Clin. Psychiatry*. In press
- Barkley RA. 2002. International consensus statement on ADHD. *Clin. Child Fam. Psychol. Rev.* 5:89–111
- Barkley RA, Brown TE. 2008. Unrecognized attention-deficit/hyperactivity disorder in adults presenting with other psychiatric disorders. *CNS Spectrums* 13:977–84
- Barkley RA, Fischer M, Smallish L, Fletcher K. 2002. The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. *J. Abnorm. Psychol.* 111:279–89
- Barkley RA, Murphy KR, Fischer M. 2008. *ADHD in Adults: What the Science Says*. New York: Guilford
- Biederman J, Petty CR, Dolan C, Hughes S, Mick E, et al. 2008a. The long-term longitudinal course of oppositional defiant disorder and conduct disorder in ADHD boys: findings from a controlled 10-year prospective longitudinal study. *Psychol. Med.* 38:1027–36
- Biederman J, Petty CR, Monuteaux MC, Mick E, Parcell T, et al. 2008b. The longitudinal course of comorbid oppositional defiant disorder in girls with attention-deficit/hyperactivity disorder: findings from a controlled 5-year prospective study. *J. Dev. Behav. Pediatr.* 29:501–7
- Boden JM, Fergusson DM, Horwood LJ. 2010. Risk factors for conduct disorder and oppositional defiant disorder: evidence from a New Zealand birth cohort. *J. Am. Acad. Child Adolesc. Psychiatry* 49:1125–33
- Burke JD, Hipwell AE, Loeber R. 2010a. Dimensions of oppositional defiant disorder as predictors of depression and conduct disorder in girls. *J. Am. Acad. Child Adolesc. Psychiatry* 49:484–92
- Burke JD, Waldman I, Lahey BB. 2010b. Predictive validity of childhood oppositional defiant disorder and conduct disorder: implications for DSM-V. *J. Abnorm. Psychol.* 119:739–51
- Burke JD. 2011. The relationship between conduct disorder and oppositional defiant disorder and their continuity with antisocial behaviors: evidence from longitudinal clinical studies. In *Externalizing Disorders of Childhood: Refining the Research Agenda for DSM-V*, ed. D Shaffer, E Leibenluft, RA Rhode. Arlington, VA: Am. Psychiatr. Assoc. In press
- Caldwell M, Skeem J, Salekin R, Van Rybroek G. 2006. Treatment response of adolescent offenders with psychopathy features: a 2-year follow-up. *Crim. Just. Behav.* 33:571–96
- Canino G, Polanczyk G, Bauermeister JJ, Rohde LA, Frick PJ. 2010. Does the prevalence of CD and ODD vary across cultures? *Soc. Psychiatry Psychiatr. Epidemiol.* 45:695–704
- Carr L, Henderson J, Nigg JT. 2010. Cognitive control and attentional selection in adolescents with ADHD versus ADD. *J. Clin. Child Adolesc. Psychol.* 39:726–40
- Castellanos FX, Lee PP, Sharp W, Jeffries NO, Greenstein DK, et al. 2002. Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder. *JAMA* 288:1740–48
- Castellanos FX, Sharp WS, Gottesman RF, Greenstein DK, Giedd JN, Rapoport JL. 2003. Anatomic brain abnormalities in monozygotic twins discordant for attention deficit hyperactivity disorder. *Am. J. Psychiatry* 160:1693–96
- Clark LA. 2007. Assessment and diagnosis of personality disorder. Perennial issues and an emerging reconceptualization. *Annu. Rev. Psychol.* 58:227–57
- Clements SD, Peters JE. 1962. Minimal brain dysfunctions in the school-age child. Diagnosis and treatment. *Arch. Gen. Psychiatry* 6:185–97

- Conners CK, Erhardt D, Epstein JN, Parker JDA, Sitarenios G, Sparrow E. 1999. Self-ratings of ADHD symptoms in adults I: factor structure and normative data. *J. Attention Disord.* 3:141–51
- Couto JM, Gomez L, Wigg K, Ickowicz A, Pathare T, et al. 2009. Association of attention-deficit/hyperactivity disorder with a candidate region for reading disabilities on chromosome 6p. *Biol. Psychiatry* 66:368–75
- Crapanzano AM, Frick PJ, Terranova AM. 2010. Patterns of physical and relational aggression in a school-based sample of boys and girls. *J. Abnorm. Child Psychol.* 38:433–45
- Dandreux DM, Frick PJ. 2009. Developmental pathways to conduct problems: a further test of the childhood and adolescent-onset distinction. *J. Abnorm. Child Psychol.* 37:375–85
- Derefinko KJ, Adams ZW, Milich R, Fillmore MT, Lorch EP, Lynam DR. 2008. Response style differences in the inattentive and combined subtypes of attention-deficit/hyperactivity disorder. *J. Abnorm. Child Psychol.* 36:745–58
- DeShazo-Barry T, Lyman RD, Klinger LG. 2002. Academic underachievement and attention-deficit hyperactivity disorder: the negative impact of symptom severity on school performance. *J. Sch. Psychol.* 40:259–83
- Diamond A. 2005. Attention-deficit disorder (attention-deficit/hyperactivity disorder without hyperactivity): a neurobiologically and behaviorally distinct disorder from attention-deficit/hyperactivity disorder (with hyperactivity). *Dev. Psychopathol.* 17:807–25
- Drabick DAG, Gadow KD, Loney J. 2007. Source-specific oppositional defiant disorder: comorbidity and risk factors in referred elementary schoolboys. *J. Am. Acad. Child Adolesc. Psychiatry* 46:92–101
- Edens JF, Campbell JS, Weir JM. 2007. Youth psychopathy and criminal recidivism: a meta-analysis of the psychopathy checklist measures. *Law Hum. Behav.* 31:53–75
- First MB, Wakefield JC. 2010. Defining “mental disorder” in DSM-V. *Psychol. Med.* 40:1779–82
- Fliers E, Vermeulen S, Rijdsdijk F, Altink M, Buschgens C, et al. 2009. ADHD and poor motor performance from a family genetic perspective. *J. Am. Acad. Child Adolesc. Psychiatry* 48:25–34
- Frick PJ, Morris AS. 2004. Temperament and developmental pathways to severe conduct problems. *J. Clin. Child Adolesc. Psychol.* 33:54–68
- Frick PJ, Blair RJR, Castellanos FX. 2011. Callous-unemotional traits and developmental pathways to the disruptive behavior disorders. In *Advances in Child and Adolescent Psychopathology. Volume 1: Disruptive Behavior Disorders*, ed. PH Tolan, BL Leventhal. New York: Springer. In press
- Frick PJ, Dickens C. 2006. Current perspectives on conduct disorder. *Curr. Psychiatry Rep.* 8:59–72
- Frick PJ, Lahey BB, Applegate B, Kerdyck L, Ollendick T, et al. 1994. DSM-IV field trials for the disruptive behavior disorders: use of symptom utility estimates. *J. Am. Acad. Child Adolesc. Psychiatry* 33:529–39
- Frick PJ, Lahey BB, Loeber R, Tannenbaum LE, Van Horn Y, et al. 1993. Oppositional defiant disorder and conduct disorder: a meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clin. Psychol. Rev.* 13:319–40
- Frick PJ, Moffitt TE. 2010. *A Proposal to the DSM-V Childhood Disorders and the ADHD and Disruptive Behavior Disorders Workgroup to Include a Specifier to the Diagnosis of Conduct Disorder Based on the Presence of Callous-Unemotional Traits*. Washington, DC: Am. Psychiatr. Assoc. <http://www.dsm5.org/Proposed%20Revision%20Attachments/Proposal%20for%20Callous%20and%20Unemotional%20Specifier%20of%20Conduct%20Disorder.pdf>
- Frick PJ, Stickle TR, Dandreux DM, Farrell JM, Kimonis ER. 2005. Callous-unemotional traits in predicting the severity and stability of conduct problems and delinquency. *J. Abnorm. Child Psychol.* 33:471–87
- Frick PJ, Viding EM. 2009. Antisocial behavior from a developmental psychopathology perspective. *Dev. Psychopathol.* 21:1111–31
- Frick PJ, White SF. 2008. Research review: the importance of callous-unemotional traits for developmental models of aggressive and antisocial behavior. *J. Child Psychol. Psychiatry* 49:359–75**
- Gadow KD, Nolan EE. 2002. Differences between preschool children with ODD, ADHD, and ODD+ADHD symptoms. *J. Child Psychol. Psychiatry* 43:191–201
- Garland EJ, Garland OM. 2001. Correlation between anxiety and oppositionality in a children’s mood and anxiety disorder clinic. *Can. J. Psychiatry* 46:953–58
- Goth-Owens TL, Martinez-Torteya C, Martel MM, Nigg JT. 2010. Processing speed weakness in children and adolescents with non-hyperactive but inattentive ADHD (ADD). *Child Neuropsychol.* 16:577–91

A comprehensive review of the emotional, cognitive, personality, and social factors associated with antisocial behavior in those with and without significant levels of CU traits.

A treatment trial illustrating both the difficulties and promise in treating children with CU traits.

Cogent summary of the case for revising the ADHD age-of-onset criteria.

A longitudinal study with multiple annual assessments demonstrating the instability of the DSM-IV ADHD subtypes.

- Hare RD, Neumann CS. 2006. The PCL-R assessment of psychopathy: development, structural properties, and new directions. In *Handbook of Psychopathy*, ed. CJ Patrick, pp. 58–90. New York: Guilford
- Harpold T, Biederman J, Gignac M, Hammerness P, Surman C, et al. 2007. Is oppositional defiant disorder a meaningful diagnosis in adults? *J. Nerv. Ment. Dis.* 195:601–5
- Hartung CM, Widiger TA. 1998. Gender differences in the diagnosis of mental disorders: conclusions and controversies of the DSM-IV. *Psychol. Bull.* 123:260–78
- Hawes DJ, Dadds MR. 2005. The treatment of conduct problems in children with callous-unemotional traits. *J. Consult. Clin. Psychol.* 73:1–5**
- Hinshaw SP. 1987. On the distinction between attentional deficits/hyperactivity and conduct problems/aggression in child psychopathology. *Psychol. Bull.* 101:443–63
- Hudziak JJ, Achenbach TM, Althoff RR, Pine DS. 2008. A dimensional approach to developmental psychopathology. In *Dimensional Approaches in Diagnostic Classification: Refining the Research Agenda for DSM-V*, ed. JE Helzer, HC Kraemer, RF Krueger, H Wittchen, PJ Sirovatka, DA Regier, pp. 101–14. Arlington, VA: Am. Psychiatr. Press
- Hyman SE. 2010. The diagnosis of mental disorders: the problem of reification. *Annu. Rev. Clin. Psychol.* 6:155–79
- Insel TR, Cuthbert B, Garvey M, Heinssen R, Kozak M, et al. 2010. Research Domain Criteria (RDoC): developing a valid diagnostic framework for research on mental disorders. *Am. J. Psychiatry* 167:748–52
- Kahn RE, Frick PJ, Youngstrom E, Findling RL, Youngstrom JK. 2011. The effects of including a callous-unemotional specifier for the diagnosis of conduct disorder. *J. Child Psychol. Psychiatry*. In press
- Keenan K, Coyne C, Lahey BB. 2008. Should relational aggression be included in DSM-V? *J. Am. Acad. Child Adolesc. Psychiatry* 47:86–93
- Keenan K, Wroblewski K, Hipwell A, Loeber R, Stouthamer-Loeber M. 2010. Age of onset, symptom threshold, and expansion of the nosology of conduct disorder in girls. *J. Abnorm. Psychol.* 119:689–98
- Kendell R, Jablensky A. 2003. Distinguishing between the validity and utility of psychiatric diagnoses. *Am. J. Psychiatry* 160:4–12
- Kessler RC, Amminger GP, Aguilar-Gaxiola S, Alonso J, Lee S, Ustun TB. 2007. Age of onset of mental disorders: a review of recent literature. *Curr. Opin. Psychiatry* 20:359–64
- Kessler RC, Green JG, Adler LA, Barkley RA, Chatterji S, et al. 2010. Structure and diagnosis of adult attention-deficit/hyperactivity disorder: analysis of expanded symptom criteria from the Adult ADHD Clinical Diagnostic Scale. *Arch. Gen. Psychiatry* 67:1168–78
- Kieling C, Kieling R, Rohde LA, Frick PJ, Moffitt T, et al. 2010. The age of onset of attention-deficit hyperactivity disorder. *Am. J. Psychiatry* 167:14–16**
- Kim-Cohen J, Arseneault L, Newcombe R, Adams F, Bolton H, et al. 2009. Five-year predictive validity of DSM-IV conduct research diagnosis in 41/2–5 year old children. *Eur. Child Adolesc. Psychiatry* 18:284–91
- Kolko DJ, Pardini DA. 2010. ODD dimensions, ADHD, and callous-unemotional traits as predictors of treatment response in children with disruptive behavior disorders. *J. Abnorm. Psychol.* 119:713–25
- Krueger RF, Markon KE. 2011. A dimensional-spectrum model of psychopathology: progress and opportunities. *Arch. Gen. Psychiatry* 68:10–11
- Krueger RF, Watson D, Barlow DH. 2005. Introduction to the special section: toward a dimensionally based taxonomy of psychopathology. *J. Abnorm. Psychol.* 114:491–93
- Lahey BB, Pelham WE, Loney J, Kipp H, Ehrhardt A, et al. 2004. Three-year predictive validity of DSM-IV attention deficit hyperactivity disorder in children diagnosed at 4–6 years of age. *Am. J. Psychiatry* 161:2014–20
- Lahey BB, Pelham WE, Loney J, Lee SS, Willcutt E. 2005. Instability of the DSM-IV subtypes of ADHD from preschool through elementary school. *Arch. Gen. Psychiatry* 62:896–902**
- Lahey BB, Rathouz PJ, Van Hulle C, Urbano RC, Krueger RF, et al. 2008. Testing structural models of DSM-IV symptoms and common forms of child and adolescent psychopathology. *J. Abnorm. Child Psychol.* 36:187–206

- Lahey BB, Van Hulle CA, Singh AL, Waldman ID, Rathouz PJ. 2011. Higher order genetic and environmental structure of prevalent forms of child and adolescent psychopathology. *Arch. Gen. Psychiatry* 68:181–89
- Lahey BB, Waldman ID. 2003. A developmental propensity model of the origins of conduct problems during childhood and adolescence. In *Causes of Conduct Disorder and Juvenile Delinquency*, ed. BB Lahey, TE Moffitt, A Caspi, pp. 76–117. New York: Guilford
- Lahey BB, Waldman ID, McBurnett K. 1999. The development of antisocial behavior: an integrative causal model. *J. Child Psychol. Psychiatry* 40:669–82
- Lahey BB, Willcutt EG. 2010. Predictive validity of a continuous alternative to nominal subtypes of attention-deficit/hyperactivity disorder for DSM-V. *J. Clin. Child Adolesc. Psychol.* 39:761–75
- Leistico AR, Salekin RT, DeCoster J, Rogers R. 2008. A large-scale meta-analysis relating the Hare measures of psychopathy to antisocial conduct. *Law Hum. Behav.* 32:28–45
- Lewinsohn PM, Shankman SA, Gau JM, Klein DN. 2004. The prevalence and co-morbidity of subthreshold psychiatric conditions. *Psychol. Med.* 34:613–22
- Loeber R, Burke J, Pardini DA. 2009. Perspectives on oppositional defiant disorder, conduct disorder, and psychopathic features. *J. Child Psychol. Psychiatry* 50:133–42
- Loo SK, Fisher SE, Francks C, Ogdie MN, MacPhie IL, et al. 2004. Genome-wide scan of reading ability in affected sibling pairs with attention-deficit/hyperactivity disorder: unique and shared genetic effects. *Mol. Psychiatry* 9:485–93
- Mannuzza S, Castellanos FX, Roizen ER, Hutchison JA, Lashua EC, Klein RG. 2011. Impact of the impairment criterion in the diagnosis of adult ADHD: 33-year follow-up study of boys with ADHD. *J. Atten. Disord.* 15:122–29
- Marcus DK, Barry TD. 2011. Does attention-deficit/hyperactivity disorder have a dimensional latent structure? A taxometric analysis. *J. Abnorm. Psychol.* 120:427–42
- Markon KE, Krueger RF. 2005. Categorical and continuous models of liability to externalizing disorders: a direct comparison in NESARC. *Arch. Gen. Psychiatry* 62:1352–59
- Maughan B, Rowe R, Messer J, Goodman R, Meltzer B. 2004. Conduct disorder and oppositional defiant disorder in a national sample: developmental epidemiology. *J. Child Psychol. Psychiatry* 45:609–21
- McBurnett K, Pfiffner LJ, Frick PJ. 2001. Symptom properties as a function of ADHD type: an argument for continued study of sluggish cognitive tempo. *J. Abnorm. Child Psychol.* 29:207–13
- McMahon RJ, Wikiewitz K, Kotler JS, Conduct Probl. Prev. Res. Group. 2010. Predictive validity of callous-unemotional traits measured early in adolescence with respect to multiple antisocial outcomes. *J. Abnorm. Psychol.* 119:752–63
- Messer J, Goodman R, Rowe R, Meltzer H, Maughan B. 2006. Preadolescent conduct problems in girls and boys. *J. Am. Acad. Child Adolesc. Psychiatry* 45:184–91
- Milich R, Balentine AC, Lynam DR. 2001. ADHD Combined Type and ADHD Predominantly Inattentive Type are distinct and unrelated disorders. *Clin. Psychol. Sci. Pract.* 8:463–88
- Moffitt TE. 2006. Life-course persistent versus adolescence-limited antisocial behavior. In *Developmental Psychopathology, Volume 3: Risk, Disorder, and Adaptation*, ed. D Cicchetti, DJ Cohen, pp. 570–98. New York: Wiley. 2nd ed.
- Moffitt TE, Arseneault L, Jaffee SR, Kim-Cohen J, Koenen KC, et al. 2008. Research review: DSM-V conduct disorder: research needs for an evidence base. *J. Child Psychol. Psychiatry* 49:3–33
- Moffitt TE, Caspi A. 2001. Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways in males and females. *Dev. Psychopathol.* 13:355–76
- Moffitt TE, Caspi A, Dickson N, Silva P, Stanton W. 1996. Childhood-onset versus adolescent-onset antisocial conduct problems in males: natural history from ages 3 to 18 years. *Dev. Psychopathol.* 8:399–424
- Moffitt TE, Caspi A, Harrington H, Milne BJ. 2002. Males on the life-course-persistent and adolescence-limited antisocial pathways: follow-up at age 26 years. *Dev. Psychopathol.* 14:179–207**
- Moffitt TE, Caspi A, Rutter M, Silva PA. 2001. *Sex Differences in Antisocial Behavior: Conduct Disorder, Delinquency, and Violence in the Dunedin Longitudinal Study*. New York: Cambridge Univ. Press

A comprehensive summary of the differential outcomes of boys on the life-course-persistent and adolescence-limited antisocial pathways from a large birth cohort.

A comprehensive summary of the poor adult outcomes for which boys with CD are at risk, including mental health, physical health, occupational, educational, and legal outcomes.

- Murrie DC, Boccaccini MT, McCoy W, Cornell D. 2007. Diagnostic labeling in juvenile court: How do descriptions of psychopathy and conduct disorder influence judges? *J. Clin. Child Adolesc. Psychol.* 36:228–41
- Nigg JT. 2006. *What Causes ADHD?: Understanding What Goes Wrong and Why*. New York: Guilford
- Nigg JT, Hinshaw SP, Huang-Pollock C. 2006. Attention deficits and hyperactivity. In *Development and Psychopathology*, ed. D Cicchetti, D Cohen, pp. 358–403. New York: Wiley. 2nd ed.
- Nigg JT, Willcutt EG, Doyle AE, Sonuga-Barke EJ. 2005. Causal heterogeneity in attention-deficit/hyperactivity disorder: Do we need neuropsychologically impaired subtypes? *Biol. Psychiatry* 57:1224–30
- Ogders DL, Caspi A, Broadbent JM, Dickson N, Hancox RJ, et al. 2007. Prediction of differential adult health burden by conduct problem subtypes in males. *Arch. Gen. Psychiatry* 64:476–84**
- Ogders DL, Moffitt TE, Broadbent JM, Dickson N, Hancox RJ, et al. 2008. Female and male antisocial trajectories: from childhood origins to adult outcomes. *Dev. Psychopathol.* 20:673–716
- Patrick CJ. 2006. Back to the future: Cleckley as a guide to the next generation of psychopathy research. In *Handbook of Psychopathy*, ed. CJ Patrick, pp. 605–18. New York: Guilford
- Patterson GR, Yoerger K. 1997. A developmental model for late-onset delinquency. In *Motivation and Delinquency*, ed. DW Osgood, pp. 119–77. Lincoln: Univ. Nebraska Press
- Penny AM, Waschbusch DA, Klein RM, Corkum P, Eskes G. 2009. Developing a measure of sluggish cognitive tempo for children: content validity, factor structure, and reliability. *Psychol. Assess.* 21:380–89
- Piek JP, Pitcher TM, Hay DA. 1999. Motor coordination and kinaesthesia in boys with attention deficit-hyperactivity disorder. *Dev. Med. Child Neurol.* 41:159–65
- Pine DS, Costello EJ, Dahl R, James R, Leckman JF, et al. 2011. Increasing the developmental focus of DSM-5: broad issues and specific potential applications in anxiety. In *The Conceptual Evolution of DSM-5*, ed. DA Regier, WE Narrow, EA Kuhl, DJ Kupfer. Arlington, VA: Am. Psychiatr. Publ.
- Pitcher TM, Piek JP, Hay DA. 2003. Fine and gross motor ability in males with ADHD. *Dev. Med. Child Neurol.* 45:525–35
- Rasmussen P, Gillberg C. 2000. Natural outcome of ADHD with developmental coordination disorder at age 22 years: a controlled, longitudinal, community-based study. *J. Am. Acad. Child Adolesc. Psychiatry* 39:1424–31
- Regier DA, Narrow WE, Kuhl EA, Kupfer DJ. 2009. The conceptual development of DSM-V. *Am. J. Psychiatry* 166:645–50
- Reiersen AM, Constantino JN, Todd RD. 2008. Co-occurrence of motor problems and autistic symptoms in attention-deficit/hyperactivity disorder. *J. Am. Acad. Child Adolesc. Psychiatry* 47:662–72
- Robins LN. 1966. *Deviant Children Grown Up*. Baltimore, MD: Williams & Wilkins
- Rowe R, Costello EJ, Angold A, Copeland WE, Maughan B. 2010. Developmental pathways in oppositional defiant disorder and conduct disorder. *J. Abnorm. Psychol.* 119:726–38
- Rowe R, Maughan B, Moran P, Ford T, Briskman J, Goodman R. 2009. The role of callous and unemotional traits in the diagnosis of conduct disorder. *J. Child Psychol. Psychiatry* 51:688–95
- Rutter M. 2011. Research review: child psychiatric diagnosis and classification: concepts, findings, challenges, and potential. *J. Child Psychol. Psychiatry* 52:647–60
- Sanislow CA, Pine DS, Quinn KJ, Kozak MJ, Garvey MA, et al. 2010. Developing constructs for psychopathology research: research domain criteria. *J. Abnorm. Psychol.* 119:631–39
- Shannon BJ, Raichle ME, Snyder AZ, Fair DA, Mills KL, et al. 2011. Premotor functional connectivity predicts impulsivity in juvenile offenders. *Proc. Natl. Acad. Sci. USA* 108:11241–45
- Shaw P, Eckstrand K, Sharp W, Blumenthal J, Lerch JP, et al. 2007. Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *Proc. Natl. Acad. Sci. USA* 104:19649–54
- Silverthorn P, Frick PJ. 1999. Developmental pathways to antisocial behavior: the delayed-onset pathway in girls. *Dev. Psychopathol.* 11:101–26
- Silverthorn P, Frick PJ, Reynolds R. 2001. Timing of onset and correlates of severe conduct problems in adjudicated girls and boys. *J. Psychopathol. Behav. Assess.* 23:171–81

- Stawicki JA, Nigg JT, von Eye A. 2006. Family psychiatric history evidence on the nosological relations of DSM-IV ADHD combined and inattentive subtypes: new data and meta-analysis. *J. Child Psychol. Psychiatry* 47:935–45
- Stringaris A, Goodman R. 2009a. Three dimensions of oppositionality in youth. *J. Child Psychol. Psychiatry* 50:216–23
- Stringaris A, Goodman R. 2009b. Longitudinal outcome of youth oppositionality: Irritable, headstrong, and hurtful behaviors have distinctive predictions. *J. Am. Acad. Child Adolesc. Psychiatry* 48:404–12**
- Tibbetts SG, Piquero AR. 1999. The influence of gender, low birth weight, and disadvantaged environment in predicting early onset of offending: a test of Moffitt's interactional hypothesis. *Criminology* 37:843–77
- Todd RD, Huang H, Todorov AA, Neuman RJ, Reiersen AM, et al. 2008. Predictors of stability of attention-deficit/hyperactivity disorder subtypes from childhood to young adulthood. *J. Am. Acad. Child Adolesc. Psychiatry* 47:76–85
- Todd RD, Rasmussen ER, Wood C, Levy F, Hay DA. 2004. Should sluggish cognitive tempo symptoms be included in the diagnosis of attention-deficit/hyperactivity disorder? *J. Am. Acad. Child Adolesc. Psychiatry* 43:588–97
- Tremblay RE. 2003. Why socialization fails. In *Causes of Conduct Disorder and Delinquency*, ed. BB Lahey, TE Moffitt, A Caspi, pp. 182–226. New York: Guilford
- Viding E, Blair RJR, Moffitt TE, Plomin R. 2005. Evidence for substantial genetic risk for psychopathy in 7-year-olds. *J. Child Psychol. Psychiatry* 46:592–97**
- Volk HE, Todorov AA, Hay DA, Todd RD. 2009. Simple identification of complex ADHD subtypes using current symptom counts. *J. Am. Acad. Child Adolesc. Psychiatry* 48:441–50
- Wakschlag LS, Briggs-Gowan MJ, Carter AS, Hill C, Danis B, et al. 2007. A developmental framework for distinguishing disruptive behavior from normative misbehavior in preschool children. *J. Child Psychol. Psychiatry* 48:976–87
- Wakschlag LS, Tolan P, Leventhal BL. 2010. "Ain't misbehavin'": towards a developmentally-specified nosology for preschool disruptive behavior. *J. Child Psychol. Psychiatry* 51:3–22
- Walton KE, Ormel J, Krueger RF. 2011. The dimensional nature of externalizing behaviors in adolescence: evidence from a direct comparison of categorical, dimensional, and hybrid models. *J. Abnorm. Child Psychol.* 39:553–61
- Waschbusch DA, Carrey NJ, Willoughby MT, King S, Andrade BF. 2007. Effects of methylphenidate and behavior modification on the social and academic behavior of children with disruptive behavior disorders: the moderating role of callous/unemotional traits. *J. Clin. Child Adolesc. Psychol.* 36:629–44
- White NA, Piquero AR. 2004. A preliminary empirical test of Silverthorn and Frick's delayed-onset pathway in girls using an urban, African-American, US-based sample. *Crim. Behav. Ment. Health* 14:291–309
- Willcutt EG, Nigg JT, Rohde LA, Tannock R, Solanto M, et al. 2011. Meta-analysis of DSM-IV ADHD dimensions and subtypes. *J. Abnorm. Psychol.* In press**
- Willcutt EG, Pennington BF, Duncan L, Smith SD, Keenan JM, et al. 2010. Understanding the complex etiologies of developmental disorders: behavioral and molecular genetic approaches. *J. Dev. Behav. Pediatr.* 31:533–44
- Willcutt EG, Pennington BF, Smith SD, Cardon LR, Gayan J, et al. 2002. Quantitative trait locus for reading disability on chromosome 6p is pleiotropic for attention-deficit/hyperactivity disorder. *Am. J. Med. Genet.* 114:260–68
- Wood DR, Reimherr FW, Wender PH, Johnson GE. 1976. Diagnosis and treatment of minimal brain dysfunction in adults: a preliminary report. *Arch. Gen. Psychiatry* 33:1453–60
- Youngstrom E. 2011. *Secondary Data Analyses Testing the Use of Pervasiveness as a Severity Index*. Washington, DC: Am. Psychiatr. Assoc. <http://www.dsm5.org>
- Zoccolillo M, Tremblay R, Vitaro F. 1996. DSM-III-R and DSM-III criteria for conduct disorder in preadolescent girls: specific but insensitive. *J. Am. Acad. Child Adolesc. Psychiatry* 35:461–70

An important longitudinal test of the differential outcomes associated with the different symptoms dimensions included in the diagnostic criteria for ODD.

A large twin study showing the different genetic influence to conduct problems in children with and without CU traits. The findings also provide important data to suggest that CU traits add to the current definition of childhood-onset CD.

Comprehensive review and meta-analysis of research regarding validity of DSM-IV dimensions and subtypes.
