AACAP OFFICIAL ACTION

Practice Parameter for the Assessment and Treatment of Psychiatric Disorders in Children and Adolescents With Intellectual Disability (Intellectual Developmental Disorder)

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Intellectual disability (intellectual developmental disorder) (ID/IDD) is both a psychiatric disorder and a risk factor for co-occurring psychiatric disorders in children and adolescents. *DSM-5* introduced important changes in the conceptualization and diagnosis of ID/IDD, and current research studies clarify assessment and treatment of co-occurring psychiatric disorders in this population. Optimal assessment and treatment of psychiatric illness in children and adolescents with ID/IDD includes modifications in diagnostic and treatment techniques, appreciation of variations in the clinical presentation of psychiatric disorders, an understanding of the spectrum of etiologies of behavioral disturbance, and knowledge of psychosocial and medical interventions.

Key words: practice parameter, intellectual disability, child psychiatry, assessment, treatment

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The purpose of this Practice Parameter is to guide clinicians in the assessment and treatment of co-occurring psychiatric disorders in children and adolescents with intellectual disability (intellectual developmental disorder) (ID/IDD). This parameter updates the previous 1999 AACAP parameter[1] on the management of co-occurring disorders and incorporates new research findings and changes in the field. In particular, important changes in the definition and diagnosis of ID/IDD and in the field of genetics have occurred.

In the *DSM-5,* ID/IDD, unlike earlier classifications of mental retardation, is recognized as a psychiatric disorder. A goal of the *DSM-5* revision was to emphasize that the psychiatrist has a role in assessing intellectual deficits and in using the severity table to determine adaptive functioning. *DSM-5* shifts the focus away from the IQ number to emphasize adaptive reasoning and functioning and facilitates child and adolescent psychiatrists being actively involved in the diagnosis of ID/IDD, including etiological workup and specifiers of severity, as described in the diagnostic manual. An understanding of these procedures and processes assists clinicians working with this population to be most effective in treatment.

This parameter assumes familiarity with child development and the principles of child psychiatric diagnosis and treatment. As people with ID/IDD are a heterogeneous group with a wide range of disabilities and strengths, recommendations should be adapted to an individual's needs.

METHODOLOGY

This parameter is an update of the "Practice Parameters for the Assessment and Treatment of Children, Adolescents and Adults with Mental Retardation and Comorbid Mental Disorders," which was based upon a review of literature published prior to 1999. The current Practice Parameter is based on a comprehensive search of publications produced since 1999. Articles were retrieved from PubMed, PsychInfo, Cochrane, and CENAHL databases. Searches were inclusive and used both MeSH headings and keywords.

PubMed was searched using the MeSH Major Topic intellectual disability. The initial search yielded 69,413 articles. This search was limited to English, humans, "all child (0 to 18 years)" and the years from January 1, 1999, to January 21, 2019. In addition, this retrieval was limited to the following: classical article, clinical trial, comparative study, controlled clinical trial, evaluation studies, guideline,

historical article, meta-analysis, multicenter study, practice guideline, randomized controlled trial, review, systematic review, twin study, or validation studies. The limited search resulted in 3,651 retrievals.

The PsycInfo thesaurus term "Intellectual Disability" gave an assigned heading of Mental Retardation. A search for "Mental Retardation" or "Intellectual Disability" as keywords resulted in 47,532 articles. This search was then limited to English language, human, Childhood: birth to age 12 years, Adolescence: age 13−17 years, and the years from January 1, 1999, to January 21, 2019. The final retrieval was limited to Peer Reviewed Journal, Journal Article, or Review-Book, resulting in a final yield of 3,512 articles.

The Cochrane Database of Systematic Reviews was searched using "mental retardation" or "intellectual disabil*". The reviews were limited from January 1, 1999, to January 21, 1999, resulting in 316 reviews identified.

The CINAHL database was searched with the terms Mental Retardation or intellectual disability. Limits applied included English; Human; January, 1999–January, 2019; All child (0−18 years); peer-reviewed articles; abstract available. Medline records were excluded from this search. The search produced 552 articles.

The 8,031 articles found through this process were exported into the EndNote referencing program. A total of 44 articles identified by hand-searching or recommended by experts were added. After removing 483 duplicate references, the resulting yield from the comprehensive search was 7,590 articles. The titles and abstracts of all 7,590 articles were reviewed and 1,067 articles were included for full-text examination (Figure 1). A total of 258 publications were included in the qualitative synthesis based on their relevance to the parameter topic, weight in the hierarchy of evidence, quality of individual studies, and relevance to clinical practice.

DEFINITIONS

In this parameter, unless otherwise noted, the term *Intellectual Disability (Intellectual Developmental Disorder)* (ID/IDD) refers to intellectual disability as an intellectual developmental disorder. This parameter uses the term *Global Developmental Delay* when clinical severity cannot be reliably assessed in early childhood (under age 5) and the term *Unspecified Intellectual Disability (Intellectual Developmental Disorder)* for children over age 5 when other impairments make assessment difficult. The term *child* or *children* refers to both children and adolescents, and *parents* refers to the child's primary caregivers, regardless of whether they are the biological or adoptive parents or legal guardians.

HISTORICAL REVIEW

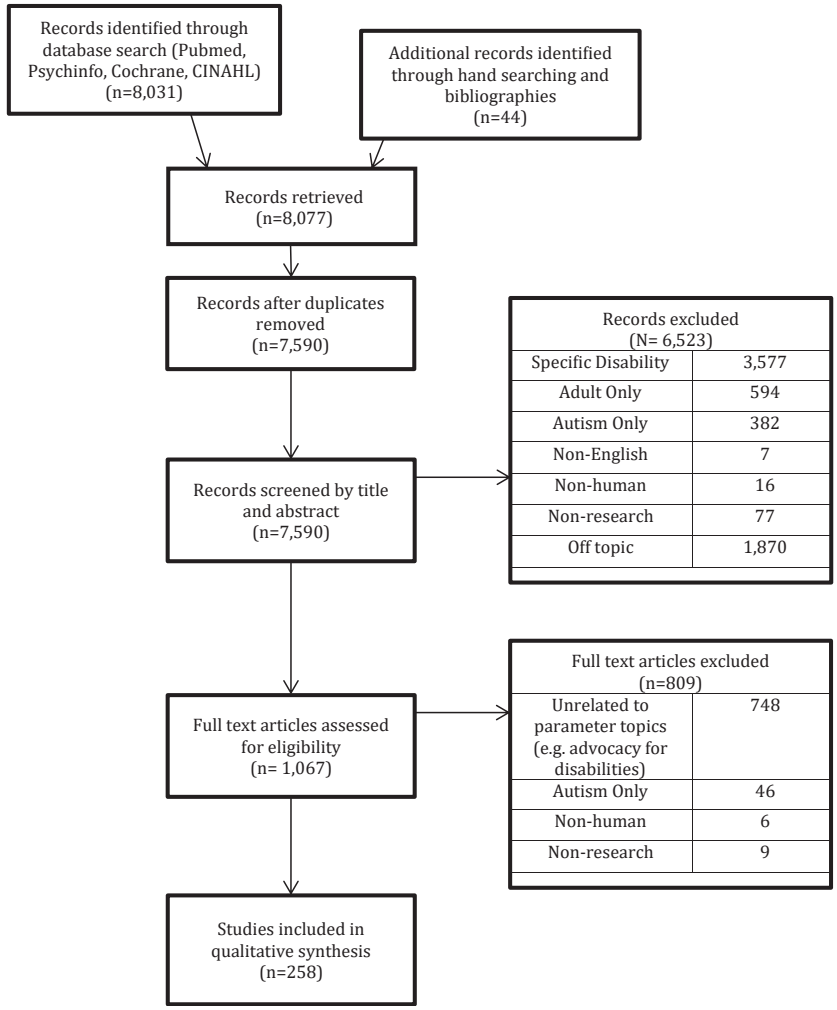
Changes in the terminology used for individuals with ID/IDD reflect society's perception of this population. In the 19th century, the now-unacceptable terms "cretin," "idiot," and "imbecile" were used, reflecting an era when these individuals were commonly housed in overcrowded asylums and institutions.[2] Many of these institutions were built during the height of the eugenics movement with the goal of preventing people with even mild ID/IDD from reproducing. Sterilization, often without consent or even the person's foreknowledge, was practiced during the eugenic era. In 1961, the American Association on Mental Retardation (AAMR) introduced the term "mental retardation" to replace earlier terms that had become pejorative.

By the 1970s, the de-institutionalization movement began to provide community care. Social Security Income for this population became available, and it was assumed that community-based care could absorb and adequately provide for their needs. A movement to assess and to treat co-occurring psychiatric disorders in individuals with ID/IDD was underway, but, unfortunately, few psychiatrists had training. By the 1980s, instruments for assessment of co-occurring psychiatric disorders were developed. Since then, evidence-based principles for assessment and treatment have been developed, and specific training incorporated into psychiatric curricula.[2]

The most recent conceptual paradigm shift began in 1992, when the American Association on Intellectual and Developmental Disabilities' (AAIDD) new definition placed greater emphasis on the disability construct by changing the earlier term "mental retardation" to "intellectual disability." The premise is that a disability occurs when the demands of the environment exceed an individual's ability. Thus, environmental modifications or social supports can ameliorate the extent to which a condition limits an individual's functioning.[3] This paradigm shift has helped frame intellectual disability not as a static condition, but one that can be enhanced by the provision of supports.[4]

In 2013, the American Psychiatric Association's (APA) fifth edition of the *DSM-5*[5] also revised the diagnostic term from "mental retardation" to "intellectual disability (intellectual developmental disorder)." However, unlike AAIDD's focus on the disability construct, the *DSM-5* focus is on the developmental disorder construct in keeping with the World Health Organization's (WHO) *International Classification of Diseases* (*ICD-11*). In *ICD-11,* these are recognized as disorders of neurodevelopment and "described as a metasyndrome occurring in the developmental period analogous to dementia or neurocognitive

FIGURE 1 Search Methodology



disorder in later life."[5] The APA revised the criteria for ID/IDD to focus on four levels of severity of impairment based on adaptive functioning across conceptual, social, and practical domains. Intelligence Quotient (IQ) scores are not used in *DSM-5* to define levels of severity.[5] The field and federal law have adopted "person first" language ("a person with intellectual disability"), and clinicians should incorporate this form of reference in their communications with and about individuals with ID/IDD.

ETIOLOGY

There are many causes of ID/IDD that reflect complex interactions of genetic predisposition, environmental insults, and developmental vulnerability. An etiology can be identified for the majority of individuals with severe ID and for a substantial subset of individuals with mild ID.[3] The classification of causes of ID has generally focused on both the type of risk factor and its timing, including prenatal, perinatal, and postnatal events.

GENETIC RISK FACTORS

Single-gene disorders or syndromes result from disruptive variants in an individual gene, which can be found only in the child (de novo variants) or can be inherited from the parents in a dominant, recessive, or X-linked fashion. The most common inherited cause of ID/IDD is Fragile X syndrome, resulting from a repeat variant in the *FMR1* gene on the X chromosome. Some single-gene disorders result in brain malformations, such as absence of cortical folds (lissencephaly) or abnormal layering of the cortex. Other single-gene disorders include neurocutaneous syndromes such as tuberous sclerosis or neurofibromatosis. Inborn errors of metabolism result from disruption of individual genes that encode enzymes that metabolize carbohydrates,

amino acids, or nucleic acids, as well as mitochondrial defects.

Chromosomal or copy number variant disorders result from either an extra copy or a missing copy of an entire chromosome or a chromosomal segment. The major chromosomal disorders are 3 autosomal trisomies (13, 18, and 21), with trisomy 21 (Down syndrome) being the most common genetic cause of ID/IDD.

Beyond single-gene, chromosomal, and copy number variant disorders that contribute a large amount of risk, genetic variants in many genes likely contribute a smaller amount of ID/IDD risk, particularly in children with mild ID/IDD, where multiple genetic and environmental risk factors may converge to lead to ID/IDD.

Prenatal and Perinatal Risk Factors

ID/IDD also has prenatal environmental risk factors, including malnutrition, vitamin or mineral deficiency, placental insufficiency, in utero exposures including alcohol, drugs, toxins, or teratogens, and maternal illness such as hypothyroidism. Fetal alcohol spectrum disorders are the leading cause of preventable developmental disabilities in the world.[6] Pre- and perinatal infections can also affect the developing fetus directly, including toxoplasmosis, syphilis, varicella-zoster, parvovirus B19, rubella, cytomegalovirus, and herpes. Although some of these infections cause mild maternal illnesses, they can have serious fetal consequences, and treatment of the mother may have limited impact on fetal outcomes. The timing of an environmental insult during fetal development is crucial in determining the type and severity of impact on the fetus.

During delivery, asphyxia is the most important risk factor for brain damage that may lead to ID/IDD. Prematurity and low birth weight, risk factors for asphyxia, as well as intracranial hemorrhage and hypoglycemia increase risk for brain damage. Placental insufficiency and intrauterine events during labor and delivery (ie, eclampsia, premature rupture of membranes) may lead to neonatal hypoxic−ischemic encephalopathy and ID/IDD.

Postnatal Risk Factors

Because ID/IDD may have onset throughout the developmental period, postnatal risk factors, potentially occurring as late as adolescence, can also lead to a diagnosis. Brain trauma, near-drowning, and other accidents that result in loss of consciousness can cause brain injury. Infections such as meningitis and encephalitis may also lead to permanent brain damage. Poisoning from lead and other substances contributes to ID/IDD risk, and severe environmental deprivation, child abuse, and neglect can also result in ID/IDD. Medical conditions such as brain tumors or intractable epilepsy also increase risk of ID/IDD, as can some surgical, radiation, or chemotherapy treatments. A child or adolescent without ID who experiences a change in cognitive functioning due to tumor, hypoxemia, or other central nervous system insult may be considered to have either traumatic brain injury (TBI) or ID, depending on the criteria of the state in which that individual resides. This determination may generate confusion, as those with acquired brain injury typically have a developmental pathway and set of needs that are different from those of individuals who have had lifelong ID/IDD.

EPIDEMIOLOGY

A recent meta-analysis estimated the overall prevalence of ID/IDD to be 10.37 per 1,000 population,[7] about 1%, similar to individual studies conducted in developed countries. The prevalence of ID/IDD in children was 18.3 per 1,000 population and was higher in males than in females. The primary risk factor was equally distributed across prenatal (including genetic), perinatal, and postnatal causes, although it was unknown in almost half of the cases. The prevalence in low-income (16.41/1,000 persons) and middle-income (15.94/1000) countries was much greater than in high-income countries (9.21/1,000). This difference has been hypothesized to be due to decreased access to, or the quality of, maternal and child health care facilities and perinatal screening methods.[8] Differences in the methodologies of prevalence studies may also contribute to the varying results, as studies in higher-income countries were more likely to use standard assessments and diagnostic systems.[7]

CLINICAL PRESENTATION AND COURSE

ID/IDD involves impairments in general mental abilities that affect adaptive reasoning in three domains: conceptual, social, and practical. Abilities in these domains determine individuals' effectiveness in functioning with everyday tasks at the level expected for their age and sociocultural background. Conceptual reasoning includes reading, writing, language, mathematics, knowledge, memory, problem solving, and judgment in novel situations. Social reasoning includes awareness of others' thoughts, feelings, and experiences; social judgment and understanding; interpersonal communication skills; and social problem solving. Practical reasoning includes self-management across life settings and includes personal care, responsibilities, transportation, finances, school and

work organization, and occupational skills.[4,5] Limitations in intellectual ability may impair learning from instruction and experience, practical understanding, reasoning, judgment in novel situations, and assessment of risk. Limitations in these areas, however, often coexist with strengths. For example, individuals with ID/IDD may have difficulty with conceptual reasoning (such as judgment in novel situations), but may have strengths in practical and social reasoning (such as recognition of others' thoughts and feelings and ability to attend to their own personal care). An appreciation of an individual's strengths and weaknesses facilitates individualization of supports.

Children generally come to clinical attention based upon observed delays in achieving developmental milestones, and as cognitive, social, and practical demands increase over time and reveal disparities between environmental expectations and the capacity of the individual. The mismatch in ability and expectations may be associated with frustration, adversely affect self-esteem, and lead to behavioral disturbances, which often lead to the assessment that reveals the presence of ID/IDD.

Onset of intellectual disability is defined in *DSM-5* as occurring during the developmental period.[5] The age and presentation at onset depends on the etiology and the severity of ID/IDD.[5] In the first 2 years of life, delayed motor, language, and social milestones may be identified in individuals with severe ID/IDD, whereas mild ID/IDD may not be identified until school age, when difficulties in academic learning become apparent.[5]

Although ID/IDD is generally lifelong, it is not a static disability. The level of cognitive impairments and adaptive skills may change over time, especially as the demands of the environment change (eg, as a youth leaves school and enters the workplace). Early and ongoing interventions may improve adaptive functioning throughout childhood and adulthood. In some cases, these result in significant improvement in intellectual functioning, such that the diagnosis of ID/IDD is no longer appropriate.[5] In addition, impairments can be reduced by provision of personalized services and supports to maximize adaptive ability.[3,4] Cognitive and adaptive impairments may also improve with treatment of co-occurring medical conditions (eg, epilepsy, hearing loss, or visual impairments) or worsen with progression of genetic disorders (eg, Rett syndrome).[5]

DIFFERENTIAL DIAGNOSIS OF ID/IDD

The diagnosis of an intellectual disability is made when the *DSM-5* criteria are met and other disorders have been adequately ruled out as the etiology. ID/IDD must be differentiated from specific learning disorders, communication disorders, major and mild neurocognitive disorders, autism spectrum disorder, and disorders that could affect performance for intelligence or adaptive skills when tested, such as affective disorders or psychosis. ID/IDD should not be assumed to be due to a genetic or medical condition. However, if a genetic or medical condition linked to ID/IDD is present, it should be noted in the diagnosis of ID/IDD.[5]

PSYCHIATRIC COMORBIDITIES IN CHILDREN WITH ID/IDD

Psychiatric disorders occur at least three times more often in children and adolescents with ID/IDD than in children with typical development.[9,10] Particularly high rates have been reported for oppositional defiant disorder (ODD), attention-deficit/hyperactivity disorder (ADHD), and anxiety disorders.[9,10] Autism spectrum disorder (ASD) also commonly co-occurs with ID,[9,10] as does fetal alcohol spectrum disorder (FASD), also known as neurodevelopmental disorder associated with prenatal alcohol exposure (ND-PAE).[5] In addition, serious behavioral problems have been reported to occur 2.5 to 4 times more frequently in children with ID/IDD compared to children with typical development.[11-13]

Although there is a dearth of rigorous studies examining how the presentation of psychiatric disorders may be altered in the context of atypical intellectual development, the course of some co-occurring psychiatric disorders is becoming better known, particularly for ADHD and anxiety.[14-16] Individuals with ID/IDD experience a developmental course of ADHD similarly to that of typically developing children, with more prominent hyperactivity symptoms earlier in development. Inattentive symptoms, which tend to decrease in the teen years in children with typical development, do not typically decrease in children with ID/IDD.[17] Co-occurring anxiety disorders in youths with ID/IDD vary in prevalence by age, as in children with typical development; however the reduction in the rate of separation anxiety disorder in children with ID/IDD occurs over a longer period of time than in children with typical development.[16]

Risk factors for co-occurring psychiatric disorders include the severity of cognitive, adaptive, and language impairments, socialization deficits, low family socioeconomic status, trauma, and having a single biological parent as a caregiver.[18] Certain genetic syndromes are associated with increased rates of psychiatric and behavioral disorders. Table 1[19] lists behavioral phenotypes and psychiatric features in certain genetic syndromes associated with ID/IDD.

ASSESSMENT

Screening

Clinical consensus and existing guidelines from the Social Security Act, the Individuals with Disabilities Education Improvement Act (IDEA) of 2004, and the American Academy of Pediatrics (AAP)[20,21] consistently recommend standardized developmental screening and support a variety of validated, parent-report screening tools for the early identification of developmental delays. The Ages and Stages Questionnaire (ASQ),[22] which is endorsed across specialties for clinical screening and research purposes,[23] and the Parent's Evaluation of Developmental Status (PEDS)[24] are two of the most frequently used measures.[25] Table 2 lists commonly used screening tools for developmental delay.

The American Academy of Pediatrics recommends that screening begin in the medical home as part of the standard 9-, 12-, 18-, 24-, and 30-month well-child visits, with sensitive and specific standardized developmental screening tools[21] that are able to detect children who display underdeveloped motor, language, and social skills. Some children display signs of ID/IDD later in their development, when academic demands exceed their capacities. Despite minimal literature on, or measures available for, systematic screening in later childhood and adolescence, primary care and mental health professionals should consider referring children and adolescents who have academic performance or behavioral challenges for standardized testing of intellectual functioning.

Diagnostic Evaluation

A positive screen for intellectual or developmental delay is followed by a diagnostic evaluation by a qualified practitioner (eg, a psychologist) to determine the degree of intellectual and adaptive impairment. Prior guidelines from the IDEA and the AAP[20,21] recommend that any child who screens positive for developmental delay should have standardized testing of intellectual functioning and assessment of adaptive functioning, using validated measures. Commonly used assessments of intelligence and adaptive functioning are listed in Table 3.

Child and adolescent psychiatrists (CAP) can be actively involved in the diagnosis of ID/IDD and in specifying severity as described in the diagnostic manual.[5] The *DSM-5* emphasizes the psychiatrist's role in the following: (1) identifying intellectual deficits (eg, in reasoning, problem solving, planning, abstract thinking, judgment, academic learning, learning from experience); (2) identifying adaptive functioning deficits in one or more areas of daily life (eg, communication, social participation, independent living) across multiple environments (eg, home, school, work, community); and (3) using the severity table to assess adaptive functioning in three domains (conceptual, social, and practical).[5]

The *DSM-5* intellectual deficit criterion is met when confirmed by both clinical assessment and individualized, standardized intelligence testing.[5] The *DSM-5* adaptive functioning deficit criterion is met when the individual fails to meet standards for personal independence and social responsibility.[5] Standardized scales to measure adaptive behaviors do not capture all functional domains; thus, adaptive functioning requires clinical assessment.[1,5]

The *DSM-5* notes that IQ test scores are "approximations of conceptual functioning but may be insufficient to assess reasoning in real life situations and mastery of practical skills."[5] With standardized intelligence testing, individual cognitive profiles based on neuropsychological testing are more useful for understanding intellectual disability than a single IQ score. The profile of IQ subtests can be more helpful than the IQ composite score, as it can reveal both cognitive strengths and weaknesses needing supports.[1] Performance on standardized testing can be underestimated in children from cultural and linguistic minorities, as testing instruments may not be as sensitive for these populations. Other factors at the time of testing that can have significant effects on the reliability of the results include motivation, cooperation, interest, temperament, behavior, physical health, mental health, the test setting, and whether the tester has a supportive attitude, as well as communication, sensory, and motor factors.

In addition, the results of intellectual testing are less reliable for individuals with more severe ID or language impairment, as fewer of such individuals were ascertained to establish score ranges. Clinical training and judgment are thus required to interpret the results of intellectual testing and assess performance.

In children under 5 years of age, IQ measures are not considered reliable, and the term "Global Developmental Delay" (GDD) is used, defined by significant limitations in two or more developmental domains.[5] The term "Unspecified Intellectual Disability" (Intellectual Developmental Disorder) is used in individuals over the age of 5 years when assessment of the degree of intellectual disability is rendered difficult or impossible because of associated sensory or physical impairment, communicative difficulties, locomotor disability, or the presence of severe problem behaviors or co-occurring psychiatric disorders.[5]

A diagnosis of ID/IDD requires continued reassessment, and federal law requires re-evaluation at least every 3 years in school-aged children.[26] Provisional diagnoses are sometimes necessary in the absence of reliable measurement

TABLE 1 Behavioral Phenotypes and Psychiatric Features in Specific Genetic Syndromes[a]

Diagnosis (Mutation)		
Diagnostic Testing	**Cognitive Features**	**Psychiatric Features**
Down syndrome (Trisomy 21) *Diagnostic Testing:* Karyotype	Mild—moderate to severe intellectual disability Strengths: grammar Weaknesses: expressive language Visual processing better than auditory	>50%: Hyperactivity, impulsiveness, inattention, and stubbornness 30%: Anxiety, depression 10%: Autism
Fragile X syndrome (FMR1; triplet repeat expansion) *Diagnostic Testing:* FMR1 PCR and Southern blot for CGG repeat length	Mild—moderate to severe intellectual disability Difficulty with abstract thinking, sequential cognitive processing, short-term memory, mathematics, and visual—motor processing	Attention dysfunction, hyperarousal, social anxiety, social cognition and communication challenges 25%—50%: Autism
Rett syndrome (MECP2) *Diagnostic Testing:* MECP2 sequencing; del/dep testing	Severe to profound intellectual disability Limited language acquisition and use	Stage I: Decreased interactions Stage II: Social withdrawal, irritability, autistic-like behaviors, sleep disturbance Stage III: Improvement in alertness, interactions, ongoing sleep disturbance Stage IV: Persistence of poor communication, irritability
Prader—Willi syndrome (15q11—q13; imprinting-loss of paternal contribution) *Diagnostic Testing:* Methylation PCR followed by FISH	Mild to borderline intellectual disability Strengths: visuospatial performance, reading and decoding, and long-term memory Weaknesses: short-term memory, auditory processing, socialization, mathematical skills, and sequential processing	Extreme hyperphagia, self-injurious behaviors (skin picking), OCD Social cognition deficits Cognitive inflexibility, explosiveness, poor affect regulation Depression/mood disorder/psychosis
Angelman syndrome (15q11—q13; imprinting-loss of maternal contribution) *Diagnostic Testing:* Methylation PCR followed by FISH; UBE3A sequencing	Severe to profound intellectual disability Minimal expressive speech, better receptive language	Social and happy; frequent inappropriate and unexpected laughter Positive interpersonal bias, social disinhibition with a diminished fear of strangers Fear of crowds and noise, hyperactivity/inattention, sleep disturbance
Williams syndrome (deletion 7q11.23) *Diagnostic Testing:* Locus-specific FISH or CGH microarray	Mild intellectual disability (75%) Strengths: auditory rote memory and language Weaknesses: severe visuospatial construction deficits and language	Adaptive behavior less than expected for IQ Superficial sociability Externalizing: inattention, impulsivity, attention seeking, hyperactivity, and temper tantrums Internalizing: obsessions/preoccupations, fears, anxiety, sadness/depression ADHD (>50%) Sleep disturbance

(continued)

TABLE 1 Continued

Diagnosis (Mutation)		
Diagnostic Testing	**Cognitive Features**	**Psychiatric Features**
Deletion 22q11.2 *Diagnostic Testing:* Locus-specific FISH or CGH microarray	Borderline to mild intellectual disability Verbal IQ is higher than performance Strengths: language abilities Weaknesses: receptive and high-order language skills, abstract reasoning, and visuospatial deficits	Emotional dysregulation ADHD Anxiety and phobias Poor social adaptation with withdrawal Autism 30%: Psychotic symptoms
Smith–Magenis syndrome (deletion 17p11.2) *Diagnostic Testing:* Locus-specific FISH or CGH microarray	Moderate intellectual disability (range from mild to severe) Weaknesses: short-term memory, visual-motor coordination, sequencing, and response speed Speech delay is common, with receptive language better than expressive	Sleep disturbance (inverted melatonin in circadian rhythms) Self-injurious behaviors Stereotypy: self-hug, lick-and-flip, mouthing objects, teeth grinding, body rocking, spinning Socially adult-oriented, demanding of adult attention, egocentric, delayed empathic skills
Lesch–Nyhan syndrome (HPRT deficiency) *Diagnostic Testing:* Urine urate/creatinine ratio (>2.0 suggestive) followed by HPRY enzyme activity determination; sequencing is available	Mild to moderate intellectual disability	Chronic, compulsive, self-injurious behaviors resulting in self-mutilation: biting, eye poking, fingernail pulling, psychogenic vomiting, arching, head snapping, head banging Language pattern: repeated ambivalent statements with anxiety and vulgarity Frequent compulsive aggression toward others (grabbing and pinching)

Note: ADHD = attention-deficit/hyperactivity disorder; CGH = comparative genomic hybridization; FISH = fluorescence in situ hybridization; IQ = Intelligence Quotient; OCD = obsessive-compulsive disorder; PCR = polymerase chain reaction.
[a]*Adapted from Siegel et al.*[19]

of cognitive function. Diagnoses of global developmental delay or unspecified intellectual disability should be reassessed if and when more precise evaluation becomes possible.[5] In addition, although ID/IDD is generally nonprogressive, the intensity and nature of needed supports and thus the severity level may change over time. Interventions aimed at environmental influences, communication, language, hearing, vision, motor or sensory function, and co-occurring medical or psychiatric conditions may allow for new skill acquisition and improvement in functioning, and thus the need for support may change. The *DSM-5* specifies that diagnostic assessments should determine whether improved adaptive skills are the result of a stable, generalized new skill acquisition (in which case the diagnosis of ID/IDD may no longer be appropriate) or whether the improvement is contingent on the presence of supports and ongoing interventions (in which case the diagnosis of ID/IDD would still be appropriate).

Etiological Assessment

Guidelines from the Agency for Healthcare Research and Quality (AHRQ),[27] the International Standard Cytogenomic Array Consortium (ISCAC),[28] the American Academy of Neurology (AAN),[29,30] and the AAP[31] recommend an evaluation to determine the etiology, once a diagnosis of ID/IDD, GDD, or unspecified ID/IDD has been made. Establishing the etiology of ID/IDD can help predict functional impact and prognosis, provide anticipatory

TABLE 2 Commonly Used Screening Tools for Developmental Delay

Tool	Age Range	Description	Psychometrics	Availability
Ages and Stages Questionnaire (ASQ-III)	1—66 mo	30-Item age-specific parent report measure Examines communication, gross motor, fine motor, problem solving, personal-social, self-regulation, compliance, language, adaptive behaviors, autonomy, affect, and interaction with people	High test—retest reliability (.92) and interrater reliability (0.93) for risk classification. Sensitivity 0.83—0.89 Specificity 0.80—0.92 across ages	Available for purchase at: http://products.brookespublishing.com/ASQ-3-Starter-Kit-P574.aspx
Parents' Evaluation of Developmental Status (PEDS)	1—95 mo	10-Item parent report measure. Examines global/cognitive, expressive language, receptive language, fine motor, gross motor, self-help, and social—emotional	Sensitivity 0.74—0.79 Specificity 0.70—0.80 across ages	Available for purchase at: http://www.pedstestshop.com/product-category/peds-products/
Bayley Infant Neurodevelopmental Screener (BINS)	3—24 mo	10- to 15-min screen for neurological impairment or developmental delay	Test—retest reliability 0.71—0.81 Strong internal consistency Interrater reliability 0.79—0.96	Available for purchase at: http://www.pearsonclinical.com/psychology/products/100000163/bayley-infant-neurodevelopmental-screener-bins-bins.html
Denver Prescreening Developmental Questionnaire (Denver PDQ-II)	2 wk to 6 y	Parent report measure. Positive results indicate follow-up testing with Denver-II by a professional. Examines personal and social, fine motor, gross motor, and language	Sensitivity 83% Specificity 43%	Available for free download at: http://denverii.com/pdq-iis-english-birth-to-6-yrs-instructions/
Infant/Toddler Checklist (ITC)	6—24 mo	Population-based screener designed to identify risk for language and communication impairment	Sensitivity 0.87—0.94; specificity 0.75—0.89	Available for free download at: http://www.brookespublishing.com/resource-center/screening-and-assessment/csbs/csbs-dp/csbs-dp-itc/

guidance, alert clinicians to associated medical and behavioral problems, and determine recurrence risks. Etiology may also inform treatment decisions and allow access to support groups and research studies.

A child and adolescent psychiatrist can perform an evaluation focused on etiology or can refer a child to a clinical geneticist, developmental pediatrician, or pediatric neurologist for further evaluation. The CAP can facilitate a multi-disciplinary approach by coordinating efforts with other medical professionals, therapists, case managers, in-home or residential support staff, and school staff. A detailed history and physical and neurological examination

are the most critical elements of the etiologic evaluation, with genetic testing being the next most helpful component. When considering an etiologic workup, it is important to note that almost all public and private health insurance payers reimburse for genetic testing, but it may be necessary to seek preapproval.[32]

The history includes prenatal and perinatal history, behavioral history, and a three-generational family history assessing for medical problems, learning disorders, psychiatric disorders, and ID/IDD. The physical examination notes physical features, growth, and minor abnormalities.[3,5,33] Because children with ID/IDD are at greater risk for vision and hearing impairment, all children with ID/IDD should be considered for screening for these problems. In particular, speech and language delay, a feature of ID/IDD, may be the result of hearing loss, and correction may improve developmental outcome. If family history, patient history, or physical findings suggest a specific etiology (ie, Down syndrome, hypothyroidism, lead exposure), the diagnosis can be confirmed with specific testing.

If no etiology is strongly suspected from the history and examination, the following stepwise approach to additional testing is recommended. Inborn errors of metabolism (IEMs) are an uncommon but sometimes treatable cause of ID/IDD. As some metabolic disorders are not included in standard newborn screening, existing guidelines from the AAN[30] and the AAP[31] recommend that focused metabolic testing such as serum amino acids and urine organic acids be considered in individuals with suggestive clinical features (eg, developmental regression, episodic decompensation), physical examination findings (eg, hepatosplenomegaly, coarse facial features), or history (parent consanguinity, family history, homogenous population groups). Existing guidelines from the ISCAC,[28] the AAN,[30] and the AAP[31] recommend consideration of an electroencephalogram (EEG) if seizures or paroxysmal events are suspected, and brain magnetic resonance imaging (MRI) if there are historical findings (ie, intrapartum asphyxia), physical findings (cerebral palsy, microcephaly, macrocephaly, abnormalities of cranial contour), or abnormal neurologic findings (focal motor findings, pyramidal signs, extrapyramidal signs, epilepsy).

The American College of Medical Genetics,[34] the ISCAC,[28] AAN,[29] and AAP[31] recommend a chromosomal microarray in all individuals with ID of undetermined etiology. Microarray is currently the genetic test with the highest diagnostic yield in children with unexplained ID/IDD, with an abnormal result reported in 7.8% of subjects with GDD/ID/IDD and in 10.6% of those with syndromic features, on average.[28,29] The degree of intellectual disability/developmental delay does not predict the diagnostic yield.[35] Figure 2 provides a genetic testing pathway.[36]

Existing guidelines from the AAN[28] and the AAP[31] recommend that all individuals (male and female) with ID/IDD of undetermined etiology also have specific testing for Fragile X syndrome, and that girls with severe impairment may be appropriate for testing for *MECP2* mutations, regardless of whether the specific clinical features of Rett syndrome are present. FMR1 testing for Fragile X syndrome has a combined yield of at least 2% in male and female subjects with mild GDD/ID/IDD, and *MeCP2* mutations are found in 1.5% of girls with moderate/severe GDD/ID/IDD and in less than 0.5% of males with GDD/ID.[29]

For patients with a family history strongly suggestive of X-linked inheritance, the AAP guidelines[31] suggest a complete X-linked intellectual disability (XLID) panel comprising common nonsyndromic XLID genes, with exome/genome sequencing as a viable alternative as costs continue to decrease. Mutations in X-linked genes may explain up to 10% of all cases of GDD/ID. Testing of XLID genes has a yield of 42% in males from definitely X-linked families and of 17% in males from possibly X-linked families.[29]

Assessment of Factors Contributing to Psychiatric/Behavioral Symptoms

Previous guidelines from the AACAP[1] recommend that the assessment of psychiatric or behavioral symptoms in the context of ID/IDD encompasses multiple potential contributing factors including medical, genetic, behavioral, communication, cognitive, sensorial, environmental, and psychosocial factors. The overall goal of a treatment plan is full habilitation and achievement of the best quality of life possible for an individual by addressing comorbid problems and reducing the effect of functional impairments to limit the extent of disability. The following factors can contribute to the development and maintenance of psychiatric and behavioral symptoms in children and adolescents with ID/IDD.

Medical and Genetic. Medical and genetic factors can present as, or exacerbate, emotional or behavioral problems. Consider the possibility of common pediatric illnesses and causes of pain such as that from ear infections, headaches, menstrual cycles, injuries, constipation, gastroesophageal reflux, and dental problems when evaluating emotional or behavioral problems.[36] In particular, individuals with limited language and more severe ID/IDD may show physical discomfort through their behavior. In addition, genetic disorders are often associated with specific medical problems (eg, Down syndrome

TABLE 3 Common Measures of Intellectual Functioning and Adaptive Functioning in Children and Adolescents

Instrument and current version	Age Range	Description:	Availability
Measures of Intellectual Functioning			
Bayley Scales of Infant and Toddler Development (BSID-III)	2–49 mo	Subtests: language, receptive and expressive, gross motor, fine motor, cognitive problem-solving ability, and sustained attention	Available for purchase at: http://www.pearsonclinical.com/childhood/products/100000123/bayley-scales-of-infant-and-toddler-development-third-edition-bayley-iii.html
Stanford–Binet Intelligence Scale (SB5)	2–85 y	Gives an FSIQ. Subtests: verbal reasoning, abstract/visual reasoning, quantitative memory, and short-term memory	Available for purchase at: http://www.wpspublish.com/store/p/2951/stanford-binet-intelligence-scales-fifth-edition-sb-5
Wechsler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R)	2 y 6 mo to 7 y 3 mo	Gives an FSIQ score. Subtests: verbal, processing, performance, and general language.	Available for purchase at: http://www.pearsonclinical.com/psychology/products/100000422/wechsler-preschool-and-primary-scale-of-intelligence–third-edition-wppsi-iii.html
Wechsler Intelligence Scale for Children–IV (WISC-IV)	6-16 yrs	Gives an FSIQ score. 15 Subtests; 4 core areas: verbal comprehension, perceptual reasoning, working memory, and processing speed.	Available for purchase at: http://www.pearsonclinical.com/psychology/products/100000310/wechsler-intelligence-scale-for-children-fourth-edition-wisc-iv.html
Leiter International Performance Scale –3(Leiter –3)	3–75+ y	Give an NVIQ. Two groupings of subtests: cognitive and attention/memory, 10 subtests total. Measures fluid reasoning, visualization, memory, attention, cognitive interference	Available for purchase at: http://www.wpspublish.com/store/p/2840/leiter-international-performance-scale-third-edition-leiter-3
Mullen Scales of Early Learning	Birth to 68 mo	Measures cognitive ability and motor development; 5 scales: gross motor, visual reception, fine motor, expression language, receptive language	Available for purchase at: http://www.pearsonclinical.com/childhood/products/100000306/mullen-scales-of-early-learning.html
Measures of Adaptive Functioning			
Vineland Adaptive Behavior Scales–II	Birth to adulthood	Survey Interview, Parent/Caregiver Rating Forms, Expanded Interview Form, Teacher Rating Form. Assesses communication, daily living skills, socialization, motor skills, and maladaptive behaviors	Available for purchase at: http://www.pearsonclinical.com/psychology/products/100000668/vineland-adaptive-behavior-scales-second-edition-vineland-ii-vineland-ii.html

(*continued*)

TABLE 3 Continued

Measures of Intellectual Functioning			
Instrument and current version	**Age Range**	**Description:**	**Availability**
Scales of Independent Behavior—Revised	Infancy-80+ years	Interview or checklist. Assesses 14 areas of adaptive behavior and 8 areas of problem behavior.	Available for purchase at: http://www.hmhco.com/hmh-assessments/other-clinical-assessments/sib-r
AAIDD's Diagnostic Adaptive Behavior Scale (DABS)	4–21 y	Under development. Standardized assessment including three domains: conceptual skills, social skills, and practical skills.	Not yet available
Adaptive Behavior Assessment Scale (ABAS II)	Birth to 89 y	Parent/Primary Caregiver form or Teacher/daycare provider form. Assesses three domains of adaptive behavior: conceptual, social, and practical	Available for purchase at: http://www.pearsonclinical.com/psychology/products/100000449/adaptive-behavior-assessment-system-second-edition-abas-second-edition.html

Note: *FSIQ = Full Scale Intelligence Quotient; NVIQ = Nonverbal Intelligence Quotient.*

carries an increased risk of thyroid, cardiac, vision, and hearing problems). Metabolism, stress response, and immune and inflammatory mechanisms can also be affected by the underlying ID/IDD syndrome, and may contribute to psychiatric or behavioral exacerbations. For example, in children with ID/IDD due to metabolic disorders, physiologic stress can cause psychiatric or behavioral symptoms.[37]

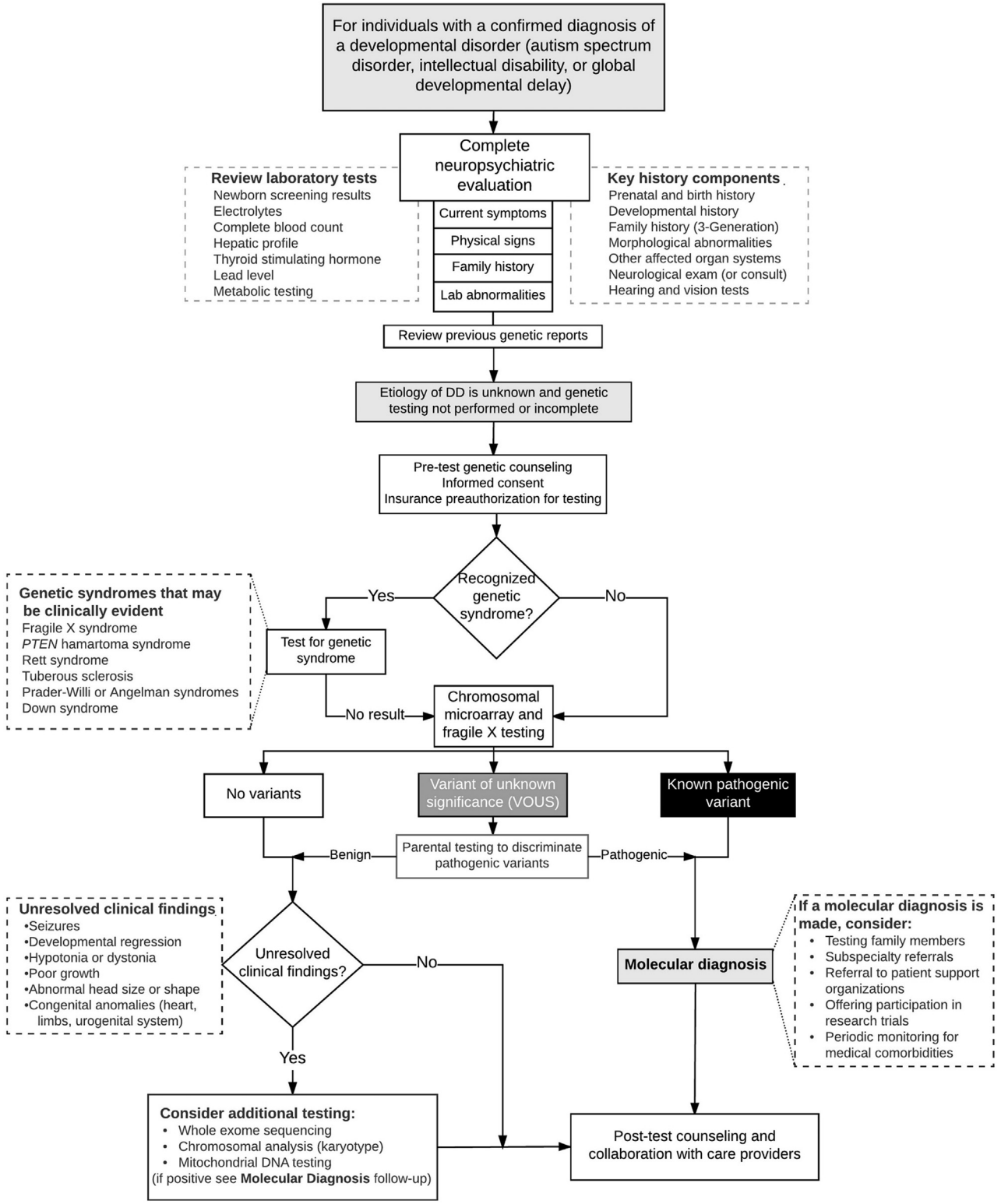
Individuals with ID/IDD are at an increased risk for seizures, and a substantial subset of individuals with severe and profound ID/IDD have a seizure disorder.[3] Post-ictal symptoms may include dysphoria, irritability, and other types of behavioral symptoms, although these typically are transient. A substantial minority of children with ID/IDD are deaf or hard of hearing, and approximately 30% of children with visual impairments have ID/IDD.[3] Children with hearing deficits or visual impairment have higher rates of anxiety and challenging behaviors, and difficulties with psychosocial adjustment and pragmatic language.[3] Children with motor impairments, such as spina bifida and cerebral palsy, have higher rates of inattention and hyperactivity.[3] Collaboration with primary care and other medical providers can assist with assessment of these potential contributing factors.

Medication. Side effects of psychotropic and other medications may contribute to psychiatric or behavioral symptoms, particularly sedating or activating medications. For example, stimulants, muscle relaxants, antiepileptics, calcium channel blockers, phosphodiesterase inhibitors, and centrally acting antiemetics have a high risk of psychiatric or behavioral side effects.[37]

Behavioral. Behaviors typically have one or more functions that serve to maintain the behavior. The most common functions are to escape (avoid demands), gain attention or gain access to preferred items/people, or internal reinforcement (eg self-stimulatory or pain-reducing) and can inadvertently be developed and maintained by the child's environment.[38] A behavioral assessment seeks to elucidate the function(s) of behavioral symptoms by understanding the setting events, antecedents, and consequences that maintain the behavior, and is typically performed by a psychologist or behavior analyst with training in applied behavioral analysis (ABA).

Communication. Limited ability to spontaneously and efficiently communicate, be it verbally or through alternative or augmentative communication (AAC) methods, can frustrate an individual and potentially cause or exacerbate psychiatric or behavioral symptoms.[39] Practitioners evaluating psychiatric or behavioral symptoms can consider

FIGURE 2 Diagnostic Genetic Testing Algorithm for Youth With Developmental Disorders



Note: *Recommendations for genetic testing in people with autism spectrum disorder (ASD), global developmental delay (GDD), and intellectual disability (ID) per the American College of Medical Genetics,[1] the American Academy of Child and Adolescent Psychiatry,[3] the American Academy of Neurology,[6] and the American Academy of Pediatrics.[4] Published with permission from Muhle et al.[36]*

whether the child has consistent access to an adequate communication system across settings, and can consult with a speech language pathologist as needed.

Cognitive, Sensory, and Occupational. Individuals with ID/IDD are at risk for being placed in settings where the demands exceed their cognitive abilities, which can lead to psychiatric or behavioral symptoms. Consideration of demand−ability matching is an important component of assessing problems in home, school, and community settings. Consideration of strengths and weakness as revealed by cognitive, academic, and adaptive skill testing can inform the level of support needed by individuals in different settings.

An occupational therapist can assess the role that motor impairments, sensory hyper- or hyporeactivity, and challenges in daily living skills such as dressing, bathing, and eating may be playing in exacerbation of psychiatric or behavioral symptoms. Social workers and case managers may also be able to assist with adaptive skill assessments and identify areas of needed support.

Environmental and Psychosocial. Individuals with ID/IDD can be sensitive to changes in their environment and can have difficulty adapting to change, particularly individuals with more severe ID/IDD. Consideration should be given to how variations in routine such as changing schools, residence, or staff may contribute to psychiatric or behavioral symptoms. The educational/habilitation program that is provided for the child and whether it meets the child's needs should be assessed, as inappropriate educational placements/demands are a major cause for the emergence of psychiatric and behavioral symptoms. Stressful life events predicted emergency department use for behavioral health complaints in individuals with ID/IDD, including the following: a move to a new house/residence; a serious problem with family, friend, or caregiver; problems with police or other authorities; sustained unemployment; recent trauma/abuse; and drug or alcohol problem.[40] Sleep disturbance is associated with behavioral and psychiatric disorders and was found to be 2.8 times more likely in youths with ID/IDD than an age-matched group of typically developing youths.[41]

Psychosocial challenges, including changing family roles, individuation, relational difficulties with other individuals, caregiver stress, exhaustion, or psychopathology, and trauma and abuse can also trigger psychiatric and behavioral symptoms. Individuals with ID/IDD are at increased risk for being bullied, particularly if they have ADHD.[42] Individuals with ID/IDD are also at an elevated risk for trauma and abuse throughout their life.[43] A study of substantiated child maltreatment cases found that 11.3% of the sample had ID/IDD.[44]

Assessment of Psychiatric Comorbidities

Previous guidelines from the AACAP[1] recommend a comprehensive psychiatric evaluation for individuals with ID/IDD who come to clinical attention for psychiatric or behavioral symptoms. The psychiatric evaluation includes collection of information on the child's current behavior, consideration of behaviors typical of the child's developmental age (as opposed to chronologic age), and comparison to the child's baseline behavior.

When interviewing caregivers and other providers, questions may need to be adapted to capture all relevant information (eg, seeking observational information about a child with limited verbal ability).[37] Questions asked of the child can be simplified and extra time allowed for the child to process the information and to articulate a response. Avoid leading questions and monitor for comprehension to avoid the child simply giving a rote response (eg, "yes") or just repeating the interviewer's last words (echolalia).

Knowledgeable informants are essential for identifying symptoms and functioning across settings, as well as constructing a picture of the child's baseline strengths and weaknesses in terms of cognitive and executive functioning, emotional expressivity, receptive and expressive language skills, and typical behavior. Caregivers can qualify the symptoms in terms of change from baseline (eg, new behavior, worsening intensity or frequency of previous behavior, new contexts in which behaviors occur), and note whether there is any discrepancy across settings or with different caregivers. Discrepancies may arise when families or school systems accommodate behaviors to minimize disruption, or provide different levels of support that reduce symptoms in one setting more than another. To evaluate the underlying impairment, consider how the presentation would change if stressors were removed or if the accommodations or supports were modified or unavailable.

Reasonable expectations for learning and behavior and an understanding of the child's level of cognitive development is guided by the child's developmental level, not chronological age. This helps to avoid pathologizing developmentally appropriate behavior in a child with developmental delays. Psychiatric diagnoses consist only of symptoms that are in excess of, or are atypical for, the child's developmental level and are causing impairment. Clinicians should remain alert to the possibility of "diagnostic overshadowing," which is the failure to recognize a co-occurring psychiatric disorder because the symptoms are attributed to ID/IDD.[45] Diagnostic overshadowing can also

TABLE 4 Assessment Tools for Co-occurring Psychiatric Disorders in Children With Intellectual Disability

Assessment Tool	Target Population	Description	Availability
Broad-Based Measures			
Reiss Scales for Children's Dual Diagnosis (RSCDD)	4–21 y Mild to profound ID Norms for children with ID	Empirically driven and developed from *DSM-III-R* taxonomy 60 Items distributed on 10 subscales: anxiety disorder; anger/self-control; attention deficit; autism/PDD; conduct disorder; depression; poor self-esteem; psychosis; somatoform behaviors; withdrawn/isolated; total score Provides information on how scores correspond to *DSM-IV* diagnosis when appropriate.	Available for purchase at: http://www.idspublishing.com/scales/
Developmental Behavior Checklist (DBC)	4–18 y version Adult version Full range of ID. Norms available by ID level.	96 Items distributed on 5 subscales Total score; disruptive/antisocial; self-absorbed; communication disturbance; anxiety; social relating; and depressive (adults only) Symptom cluster information available to calculate depression and attention-deficit/hyperactivity scores for the parent version Separate versions for parents and teachers Short versions available Daily monitoring version available Good psychometric properties and empirical support	Available for purchase at: https://www.monash.edu/medicine/scs/psychiatry/research/developmental/clinical-research/dbc
Nisonger Child Behavior Rating Form (NCBRF)	3–16 Intellectual or developmental disabilities and normally developing children	10 Social competence items distributed on 2 subscales. 66 Problem behavior items distributed over 6 subscales. Parent and teacher versions Good psychometric properties but not as much empirical support as the DBC	Available for free download at: http://psychmed.osu.edu/ncbrf.htm
Aberrant Behavior Checklist (ABC)	5–51 y+ Full range of ID (particularly severe to profound) Separately normed community and residential versions	56-Question caregiver-completed survey. Subtests: (I) Irritability, Agitation, Crying; (II) Lethargy, Social Withdrawal; (III) Stereotypic Behavior; (IV) Hyperactivity, Noncompliance; and (V)	Available for purchase at: http://www.slosson.com/onlinecatalogstore_i1002727.html?catId=51452

(continued)

TABLE 4 Continued

Assessment Tool	Target Population	Description	Availability
		Inappropriate Speech Developed to access medication and treatment effects; test−retest used to detect medication response Has been used for screening children and adolescents	
Diagnostic Manuals			
Diagnostic Manual −Intellectual Disability (*DM-ID-2*)		Developed using an expert consensus process by the National Association of the Dually Diagnosed in association with the APA, to adapt the criteria of the *DSM-5* to the intellectually disabled population.	Available at bookstores and online.
Diagnostic Criteria for Psychiatric Disorders for Use with Adults with Learning Disabilities/ Mental Retardation		Complements the *ICD-10*−DCR but has yet to be modified for children. Addresses problem behaviors and behavioral phenotypes. Replaces some self-report items with observable items	Available at bookstores and online.

occur when IDD/IDD symptoms are attributed to another psychiatric disorder or a seizure disorder.

Consideration of behavioral phenotypes can inform the psychiatric evaluation of an individual with ID/IDD. For example, hyperphagia is a behavioral phenotype of Prader−Willi syndrome, and compulsive self-injury is a behavioral phenotype of Lesch−Nyhan syndrome. Table 1 outlines behavioral phenotypes associated with common genetic syndromes that typically include ID/IDD.

Psychiatric Symptom Measures

To facilitate accurate assessment, measures validated in children and adolescents with ID/IDD can be used when available, and practitioners should be aware that measures developed with a typically developing sample may not be valid for youth with ID/IDD. In addition, the *DSM* is a categorical system that often has to be adapted in its application to people with ID/IDD. The *DSM* categories rely primarily on self-report of complex intellectual processes and do not fully address problem behaviors or behavioral phenotypes. Although not diagnostic on their own, validated measures can support diagnostic decisions, characterize the nature and breadth of specific symptoms, and serve as a baseline for tracking symptoms over time.

Selection of an instrument is determined by the objective of the assessment and measure characteristics (ie, psychometric properties and population parameters).[46] Informant-based scales can be classified by their content (broad or specific), intended population (age and level of functioning), and whether the scale was developed from diagnostic criteria from an established mental health taxonomy (ie, the *DSM*) or from factor analysis of behavioral descriptors in empirical studies.[46] Self-report questionnaires are frequently unreliable in the ID/IDD population[47]; as such, measures with parent and teacher versions can be more useful.

Three broad, empirically driven, informant-based measures to assess for psychiatric and behavioral problems in children with ID/IDD have been developed: the Developmental Behavior Checklist (DBC),[48] the Nisonger Child Behavior Rating Form (NCBRF),[49] and the Reiss Scales for Children's Dual Diagnosis (RSCDD).[50] The DBC has parent and teacher versions, contains 96 items distributed on 5 subscales, and has good psychometric properties and empirical support.[46,48] The NCBRF has parent and teacher versions, and contains 10 social competence items distributed on 2 subscales and 66 problem behavior items distributed over 6 subscales. The

NCBRF has good psychometric properties but not as much empirical support as the DBC.[46,49] The RSCDD is an extension of the Reiss Screen for Maladaptive Behavior (RSMB),[51] which was both empirically driven and developed from *DSM-III-R* taxonomy for adolescents and adults with mild to profound ID. The RSCDD contains 60 items distributed on 10 subscales.

There is a paucity of psychiatric disorder–specific measures with good empirical support. The strongest support for measuring anxiety in children with ID/IDD is with the DBC and the NCBRF subscales measuring anxiety.[16] There is some support for the reliability and validity of the Child Behavior Checklist's Anxious/Depressed subscale[52] for children with mild ID/IDD, but the overlap of the two domains in the subscale limits utility in supporting an anxiety diagnosis. Masi *et al.*[53] examined multiple measures for accuracy in diagnosing anxiety and depression in individuals with ID and found that the Psychopathology Instrument for Mentally Retarded Adults (PIMRA) and the Child Behavior Checklist (CBCL) had good convergent validity. See Table 4 for a summary of assessment tools for psychopathology.

The *Diagnostic Manual–Intellectual Disability: A Clinical Guide for Diagnosis of Mental Disorders in Persons with Intellectual Disability* (*DM-ID*)[54] was developed using an expert consensus process by the National Association of the Dually Diagnosed (NADD) in association with the APA, as a complement to the *DSM-IV-TR.* Updated to accompany the *DSM-5,* the *DM-ID-2*[55] facilitates accurate psychiatric diagnosis in children and adults with ID/IDD and provides a thorough discussion of the issues involved in applying *DSM* diagnostic criteria for psychiatric disorders to persons with intellectual disability. The *DC-LD: Diagnostic Criteria for Psychiatric Disorders for Use with Adults with Learning Disabilities/Mental Retardation* (*DC-LD*)[56] complements the *ICD-10-DCR,* but has yet to be modified for children. The *DC-LC* uses a hierarchical approach that addresses problem behaviors and behavioral phenotypes and replaces some self-report items with observable items.[57]

Informed Consent/Assent

Previous guidelines from the AACAP[58] recommend that informed parent/guardian consent for treatment should be obtained, along with patient assent when possible, in consideration of the child's or adolescent's developmental age. Informed consent may be challenging to assess and to obtain because of language, social, and cognitive limitations. Historically, it was assumed that individuals with ID/IDD lacked the ability to provide consent (ie, were considered incompetent), and thus were not involved in treatment decisions, were denied participation in research, or were inappropriately enrolled in research (eg, Willowbrook studies of hepatitis). Denying individuals with ID/IDD the opportunity to participate in well-considered and ethical research limits the knowledge available about treatments that may benefit them.

More recent arguments[59] support helping individuals with ID/IDD understand the consent process for medical decision making. This stance supports their participation according to their developmental ability to understand the information and to provide consent if they have retained their own decision-making capacities, or assent if their legal guardian will provide consent. Ethically, this approach reflects an increasing emphasis on patient autonomy, individual decision making, and respecting the rights and preferences of persons with ID/IDD. Decisional capacity is not a global entity, but rather is the ability to understand that a specific choice is to be made, to understand information about the choice (factual understanding), to understand the specific situation (eg, how the intervention or study will affect him/her), and to be able to rationally manipulate that specific information (ie, calculate risks, benefits, and weigh these in a reasonable manner).

TREATMENT

Evidence Base for Treatment Statements

In this Parameter, all treatment statements are graded as a Clinical Option [OP], reflecting their derivation from emerging rather than definitive empirical evidence (eg, few or inconsistent randomized controlled trials [rct], nonrandomized controlled trials [ct], observational studies [ob], case series/reports [cs], extrapolations from adult studies).

Statement 1: Psychosocial interventions can be considered to target comorbid psychiatric disorders or specific psychiatric symptoms in children and adolescents with ID/IDD. [OP]

Treatments

Psychotherapy/Cognitive–Behavioral Therapy. Because of a paucity of methodologically rigorous studies on the effectiveness of psychotherapeutic and cognitive–behavioral therapy (CBT) modalities in children with ID/IDD, it is reasonable when treating psychiatric symptoms and disorders in this population to consider studies of interventions that included adults with ID/IDD. Prout and Nowak-Drabik published a meta-analysis of psychotherapy effectiveness studies conducted over the prior 30 years in people with ID/IDD (26% were studies of youths, 62% adults, 11% mixed/unclear ages); the overall effect size was 1.01.[60] A systematic review by the Cochrane Collaboration identified six CBT trials in adults with ID/IDD and aggressive

behavior (three anger management trials including two group-based, and one each of relaxation, mindfulness, and problem solving), which reported some improvements; but the quality of the evidence was rated as low in all but one of the studies.[61] Studies of CBT in youth with ID/IDD are scarce, although cognitive bias modification training in 69 socially anxious youth with mild ID/IDD showed a reduction in negative interpretation bias,[62[rct]] and slow-paced breathing appeared to enhance stress management in a small study of 14 adolescents with ID/IDD.[63[ct]] Despite the minimal evidence, it should not be assumed that an individual with ID/IDD could not benefit from a psychotherapeutic approach, with accommodations for their developmental age and communication ability, particularly in the case of cognitive behavioral therapy.[46,64] Clinicians can consider collaborating with a therapist with experience working with children with ID/IDD.

Communication Interventions. A meta-analysis of 54 studies of alternative and assistive communication (AAC) interventions for challenging behavior, 65% of which were in non-ASD children, adolescents, and adults with ID/IDD, found a mean pooled effect size of 0.88 for AAC interventions.[65] Intervention at a younger age and the use of Functional Communication Training (FCT), in which children are trained to initiate communication using carefully selected communicative strategies to replace problem behaviors that serve the same function, were associated with higher effect sizes.[65] Although individuals with ID/IDD can have significant social communication impairments, a meta-analysis of teaching theory of mind skills to children, adolescents, and adults with ID/IDD[66] found little evidence of skill generalization or maintenance across time or settings.

Applied Behavioral Analysis. In a descriptive analysis of 101 behavioral intervention studies using a single-subject design, Doehring *et al.*[67] found that studies using Applied Behavioral Analysis (ABA) were generally effective for behavioral problems in youths 6 to 18 years old with ID/IDD (91% of participating youths) and/or ASD (48% of youths). The ABA interventions focused on increasing adaptive, communication, and social skills, antecedent interventions, reinforcement strategies, functional communication training, and extinction procedures. The use of differential reinforcement (eg reinforcing desired behaviors) in school and home settings was found to be one of the more successful interventions. There is far less research support for non−ABA-based interventions for problem behaviors in youth with ID/IDD. In one study, the Positive Parenting Program (Triple P) was piloted with parents of children with ID/IDD, and was associated with a decrease in parental distress, maladaptive parenting, and child behavioral issues.[68[ob]]

Social Support. Families may need psychoeducation and support throughout the lifespan, although there is a lack of empirical support for these approaches. Families may also need help in obtaining insurance benefits, community supports, school services, or governmental supports such as accessing the state developmental disability agency, and can be assisted in this by a case manager, educational advocate, or local disability advocacy organization.

Other Treatment Modalities. Albeit important domains to consider, there is currently a paucity of research studies on therapies in children with ID/IDD that are sensory-oriented, trauma-focused, or that employ cognitive training. It has been suggested that computerized attention training may enhance some aspects of attention (selective attention) in children with ID/IDD, but a single study produced small-to-medium effect sizes and awaits refinement and replication.[69[rct]]

Statement 2: Psychotropic medications can be considered to target comorbid psychiatric disorders or specific psychiatric symptoms in children and adolescents with ID/IDD. [OP]

Studies under this recommendation and in Table 5[70-81] are randomized controlled trials (RCTs) or open-label extensions of RCTs from 1999 to 2019 (since the last practice parameter) with 10 or more children with ID/IDD. Case series, studies in adults, and studies of subjects with both ASD and ID/IDD are not included. The most rigorous RCTs are of risperidone and methylphenidate, although smaller studies have examined other medications. The research evidence supporting the use of these medications tends to focus on general targets (eg "disruptive behaviors") rather than specific psychiatric disorders, and to include multiple types of behaviors or symptoms (ie, irritability, aggression, hyperactivity) the pathogenesis of which could be quite different. As opposed to ASD, there are no medications that are US Food and Drug Administration (FDA) approved specifically for use in individuals with ID.[82]

For psychopharmacology trials in children with ID/IDD prior to 1999, please see the prior AACAP Practice Parameter[1] and a review of pharmacologic management of psychiatric and behavioral symptoms in ID/IDD.[83] For psychopharmacology trials in adults with ID/IDD, see the European Association for Mental Health in Mental Retardation (EAMHMR) practice guidelines.[84] For psychopharmacology trials in children with Autism Spectrum Disorder, see the most recent AACAP Practice Parameter.[85]

TABLE 5 Randomized Controlled Trials With More Than 10 Children With Intellectual Disability Since 1999

Agent	Study	Target Symptoms	Mean Dose / Length	Demographics	Common Side Effects	Results/ Comments
Atypical Antipsychotics						
Risperidone	Buitelaar *et al.*[70,a] Randomized, double-blind, placebo-controlled, parallel design	Outwardly directed aggressive behaviors (eg, severe verbal aggression, threatening behavior, property destruction, physical violence)	2.9 mg/d 6 wk	38 Children 12–18 y Boys: 33 (85%) IQ range: 60–90 Subaverage IQ: 10 (26%) Borderline IQ: 14 (37%) Mild ID: 14 (37%) Inclusion: *DSM-IV* diagnoses: CD, ODD, or ADHD Exclusion: *DSM-IV* diagnoses: mood disorders, psychotic disorders, substance abuse disorder	Transient tiredness (58%), hypersalivation, nausea, slight weight gain Mean weight gain: 3.5% of body weight Extrapyramidal symptoms were absent or very mild	Measures: CGI-S, OAS-M, ABC, EPRS Significant improvements found on all scales in the risperidone group from baseline to endpoint Significant between group differences at end-point found for CGI-S
	Van Bellinghen and De Troch[71,a] Randomized, double-blind, placebo-controlled, parallel design.	Various "behavioral disturbances" (eg, hostility, aggressiveness, irritability, agitation, or hyperactivity)	1.20 mg/d 4 wk	13 Children 6–14 y Boys: 5 (38%) (Risperidone: 6; placebo: 7) IQ range: 66–85 Borderline IQ: 13 (100%)	No significant difference between risperidone and placebo groups with respect to mean weight gain or extrapyramidal side effects	Measures: ABC, CGI-I, CGI-S, VAS, PAC Significant improvement in behavior problems in risperidone group compared to placebo as measured by the ABC subscales for irritability and hyperactivity, the VAS, and the PAC scores for social relationships and occupational attitudes. CGI showed 83% improvement in the risperidone group and none in the placebo group
	Aman, *et al.*[72,a] Randomized, double-blind, placebo-controlled, parallel design; multi-center (N = 11)	Disruptive behaviors NCBRF score of 24 or more on the Conduct Problem subscale.	1.16 mg/d 6 wk Note: Concomitant stimulant medication allowed	118 Children 5–12 y Boys: 74 (63%) (Risperidone: 55; placebo: 63) IQ range: 34–84 Borderline IQ: 60 (50%) Mild ID: 38 (32%)	Somnolence (51%), headache (29%), vomiting (20%), dyspepsia (15%), weight increase (15%), asymptomatic	Measures: NCBR-F, ABC, CGI-I, BPI, VAS, EPRS Significant improvement in behavior problems with risperidone group compared with placebo group as measured by all

(*continued*)

TABLE 5 Continued

Agent	Study	Target Symptoms	Mean Dose / Length	Demographics	Common Side Effects	Results/ Comments
				Moderate ID: 20 (17%) Vineland ABS score: 84 or less Inclusion *DSM-IV* diagnoses: Disruptive Behavior Disorders (DBDs; eg, CD, ODD, or DBD NOS with or without ADHD). Exclusion *DSM-IV* diagnoses PDD, psychotic disorder.	elevated serum prolactin (13%), increased appetite (11%), rhinitis(11%) Mean weight gain: risperidone: 4.8 lb; placebo: 2 lb No significant between-group difference in the severity of EPS	NCBR-F subscales as well as ABC subscales for irritability, lethargy/social withdrawal, and hyperactivity; the BPI aggressive/destructive behavior subscale; and VAS CGI showed 77% improvement in the risperidone group and 33% in the placebo group
	Findling *et al.*[73] Open-label extension study of Aman, *et al.*[72]	Disruptive behaviors	1.5 mg/d 48 wk	107 Children 5—12 y Boys: 86 (80.4%) IQ range: 36—84 Borderline IQ: 51 (47.6%) Mild ID: 36 (33.6%) Moderate ID: 20 (19%)	Most common: somnolence (33%) headache (33%), rhinitis (28%), and weight gain (21%). Mean weight gain: 12.1 lb (half due to normal growth) No significant changes in extrapyramidal symptoms Transient asymptomatic increases in prolactin	Measures: NCBR-F, ABC, CGI-I, BPI, VAS 47% Completed the trial Improvement with risperidone was maintained over the 48-wk extension period
	Snyder *et al.*[74,a] Randomized, double-blind, placebo-controlled, parallel design	Conduct and disruptive behaviors (eg, aggression, impulsivity, defiance of authority figures, property destruction)	0.98 mg/d 6 wk Note: concomitant stimulant medication allowed	110 Children 5—12 y Boys: 83 (76%) (Risperidone: 53; placebo: 57) Borderline IQ: 53 (48%) Mild ID: 42 (38%) Moderate ID: 15 (14%) Vineland ABS score: 84 or less	Somnolence (41.5%), headache (17%), appetite increase (15.1%), dyspepsia (15.1%), asymptomatic increased prolactin (11.3%), weight gain (7.5%).	Measures: NCBR-F, ABC, CGI-I, BPI, VAS, ESRS Significant improvement in behavior problems in risperidone group compared with placebo group on the NCBR-F Conduct Problem Subscale, all ABC subscales (including

(*continued*)

TABLE 5 Continued

Agent	Study	Target Symptoms	Mean Dose / Length	Demographics	Common Side Effects	Results/ Comments
		NCBRF Conduct Problem subscale score: 24 or more		Co-occurring ADHD: 80% Inclusion *DSM-IV* diagnoses: DBDs (ie, CD, ODD, or DBD NOS) with or without ADHD Exclusion *DSM-IV* diagnoses: PDD, psychotic disorder	Mean weight gain: 4.8 lb No significant between-group difference in the severity of extrapyramidal symptoms	irritability, and hyperactivity subscales), the BPI aggressive/destructive behavior subscale; and the VAS. CGI showed 77% improvement in risperidone group and 25% in placebo group. Note: effect of risperidone unaffected by IQ, diagnosis, presence or absence of ADHD, stimulant use, or somnolence.
	Turgay *et al.*[75] Open-label extension study of Snyder *et al.*[74]	Disruptive behaviors	1.38 mg/ d throughout study 48 wk	77 Children 5–12 y Boys: 57 (74%) IQ range: 36–84 Borderline IQ; 39 (50.6%) Mild ID: 26 (33.8%) Moderate ID: 12 (15.4%) Vineland ABS score: 84 or less Co-occurring ADHD: 79%	Somnolence (52%), headache (38%), weight gain (36%) EPS (26%) Mean weight gain: 18.7 lb (half attributable to normal growth) No significant changes from baseline ESRS scores. Significant asymptomatic elevation in prolactin levels from baseline but within normal limits	Measures: NCBR-F, ABC, CGI-I, BPI, VAS 78% Completed the trial Improvement was maintained over the 48-wk extension period Note: effect of risperidone unaffected by IQ
Stimulants						
Methyl-phenidate (MPH)	Pearson *et al.*[76,a] Placebo-controlled, double-blind,	Hyperactivity, inattention, aggression,	0.1 5 mg/kg, 0.30 mg/kg, 0.60 mg/kg bid	24 Children 8–13 y Boys: 18 (75%) IQ: 56.5 (SD 10.24) Mild ID: 17 (71%)	Loss of appetite and sleeping problems at higher doses of MPH	Measures: ADD-H CTRS, Conners Teacher and Parent Rating Scale, ABC, RBPC, and PIC-R

(*continued*)

TABLE 5 Continued

Agent	Study	Target Symptoms	Mean Dose / Length	Demographics	Common Side Effects	Results/ Comments
	cross-over treatment	asocial behavior		Moderate ID: 7 (29%) Inclusion *DSM-III-R* diagnosis: ADHD Exclusion *DSM-III-R* diagnosis: all other diagnoses		The most significant improvements occurred at 0.60 mg/kg methylphenidate dose for teacher ratings of inattention, hyperactivity, aggression, and asocial behavior No significant improvements relative to placebo occurred at the 0.15 mg/kg dosage Of note, nearly all significant medication-related behavioral improvements were noted by teachers
	Simonoff *et al.*[77,a] Randomized, double-blind, placebo-controlled	Hyperactivity	Individually titrated low (0.5 mg/kg), medium (1.0 mg/kg), high (1.5 mg/kg) dosing 16 wk	122 Children 7−15 y Boys: 85 (70%) IQ range: 30−69 Inclusion *ICD-10* diagnosis: Hyperkinetic disorder Exclusion *ICD-10* diagnoses: dementing, psychotic, bipolar, severe obsessive-compulsive disorder, or severe Tourette syndrome	Sleep difficulty (21%) and loss of appetite (15%)	Measures: Conners Rating Scale Short Version ADHD index and hyperactivity scale from parent and teacher; ABC hyperactivity subscale; CGI-I Methylphenidate was effective in reducing ADHD symptoms with moderate effect sizes of 0.39−0.52. None of the moderators (IQ, autistic symptoms, ADHD severity) had an effect on parent- or teacher-rated Conners ADHD index
Comparison of Stimulants and Atypical Antipsychotics						
Stimulant vs. Risperidone	Aman *et al.*[78,a] Randomized, double-blind, placebo-controlled post hoc analysis of	DBD and ADHD	Risperidone: 0.02 mg/kg/d to 0.06 mg/kg/d Stimulants: MPH: 10−60 mg	155 Children 5−12 y from two 6-wk trials IQ range: 36−84 (Placebo + no stimulant: 39) Male: 29 (74%) Borderline IQ: 17 (44%)	Risperidone + stimulant group had a higher frequency of somnolence, increased appetite, and	Measures: NCBRF, ABC Risperidone-treated subjects had significant reductions in both disruptive behavior and hyperactivity scores compared to placebo, regardless of concomitant

(*continued*)

TABLE 5 Continued

Agent	Study	Target Symptoms	Mean Dose / Length	Demographics	Common Side Effects	Results/ Comments
	Aman *et al.*[72] and Synder *et al.*[74]		Dextroamphetamine: 10–30 mg Pemoline: 30–56.25 mg 6 wk Stimulant medication was ongoing and stablized before children were randomized to placebo or risperidone	Mild ID: 12 (31%) Moderate ID: 10 (26%) (Placebo + stimulant: 38) Male 35 (92%) Borderline IQ: 14 (37%) Mild ID: 18 (47%) moderate ID: 6 (16%) (Risperidone alone: 43) Male: 35 (81%) Borderline IQ: 25 (58%) Mild ID: 11 (26%) Moderate ID: 7 (16%) (Risperidone + stimulant: 35) Male: 30 (86%) Borderline IQ: 19 (54%) Mild ID: 11 (31%) Moderate ID: 5 (14%) Score of 24 or greater on the Conduct Problem subscale of the NCBRF Inclusion *DSM-IV* diagnoses: DBD + ADHD Exclusion *DSM-IV* diagnoses: PDD or psychotic disorder	weight gain than children on stimulant alone. Risperidone + stimulant group had less somnolence, headaches, and vomiting and more rhinitis and increased appetite than children on risperidone alone. Stimulant groups gained as much weight as those not receiving stimulants (mean weight gain 32.84 kg) irrespective of combined use with risperidone or placebo Asymptomatic elevated prolactin was similar for risperidone + simulant and risperidone-alone groups.	stimulant use The addition of risperidone to a stimulant resulted in significantly better control of hyperactivity than was achieved with stimulant alone
	Filho *et al.*[79,a] Randomized, single-blind, parallel-group trial	Hyperactivity/ impulsivity, inattention, ODD symptoms	MPH: 25 mg/d vs. risperidone: 2.9 mg/d	46 Children 6–16 y (MPH: 24 children) Boys: 18 (75%) Moderate ID: 24 (100%) IQ 48.4 (SD 5.1) (Risperidone: 22 children)	MPH: weight loss, decreased appetite, insomnia Risperidone: weight gain, somnolence	Measures: SNAP-IV, NCBRF, SERS, and UKU. Both groups had reduced hyperactivity/impulsivity and inattention as well as ODD symptoms during the trial,

(*continued*)

TABLE 5 Continued

Agent	Study	Target Symptoms	Mean Dose / Length	Demographics	Common Side Effects	Results/ Comments
				Boys: 16 (76%) Moderate ID: 24 (100%) IQ: 46.8 (SD 4.7) Inclusion *DSM-IV* dx: ADHD Exclusion *DSM-IV* dx: PDD, psychotic disorders		more pronounced for risperidone
Other Medications						
Clonidine	Agarwal *et al.*[80,a] Randomized, double-blind, placebo-controlled, cross-over design	Hyperactivity, impulsivity, and inattention	Fixed doses of 4, 6, 8 μg/kg/d 12 wk	10 Children 6–15 y Mean IQ: 48.2 Mild ID: 4, Moderate ID: 5 Severe ID: 1 Inclusion *ICD-10* diagnoses: Hyperkinetic disorder, conduct disorder Exclusion *ICD* diagnoses: PDD or psychotic disorder	Drowsiness (50%) Drop in SBP (70% at 4 μg had a 3% mean drop, 100% at 6 μg had a 7% mean drop, 100% at 8 μg had a 9% mean drop)	Measures: PSQ, Hillside BRS, CGI, DRTES Scale Hyperactivity and impulsivity improved with increasing doses from 4 to 8 μg Improvement in inattention occurred only with the 4- and 6-μg doses The 8-μg dose did not further improve inattention
Melatonin	Niederhofre *et al.*[81,a] Placebo-controlled, randomized, double-blind	Insomnia	0.1 or 0.3 mg nightly 30 min before bed 9 wk	20 Adolescents 14–18 y Boys: 10 (50%) IQ <70	None notable	Sleep data obtained by polysomnography on the last 3 nights of each treatment period. A 0.3-mg dose improved sleep efficiency, acting principally in the middle third of the night; it also elevated plasma melatonin levels to normal. The lowest dose (0.1 mg) also improved sleep

Note: ABC = Aberrant Behavior Checklist; ADD-H CTRS = Attention-Deficit/Hyperactivity Comprehensive Teacher Rating Scale; ADHDRS-IV = Attention-Deficit/Hyperactivity Disorder Rating Scale; AMS = Abnormal Movement Scale; BPI = Behavior Problems Inventory; CGI-I = Clinical Global Impressions–Improvement Scale; CGI-S = Clinical Global Impressions–Severity Scale; DRTES = Dosage Record Treatment Emergent Symptom Scale; dx = diagnosis; EPRS = Extrapyramidal Symptom Rating Scale; Hillside BRS = Hillside Behavior Rating Scale; IQ = Intelligence Quotient; NCBR-R = Nisonger Child Behavior Rating Form; OAS-M = Modified Overt Aggression Scale; PAC = Personal Assessment Checklist; PIC-R = Personality Inventory for Children–Revised; PSQ = Parent Symptom Questionnaire; RBPC = Revised Behavior Problem Checklist; SBP = systolic blood pressure; SERS = Barkley's Side Effects Rating Scale; SNAP = Swanson, Nolan, and Pelham; UKU = Udvalg for Kliniske Undersogeslser; VAS = visual analogue scale.
[a]*Randomized controlled trial.*

Most medication trials specifically exclude children with ID/IDD, as opposed to including them and reporting any differential responses based on the presence and severity of ID/IDD. Although a medication's effects may be influenced by medical and neurological disorders associated with ID, there continues to be no clear evidence that ID/IDD itself changes the mechanism of action of psychotropic medications. Children with ID/IDD may be more sensitive to side effects of medication and thus conservative dosing is generally recommended; although this may reflect shifts in dose-response as opposed to changes in the mechanism of action of a medication. In selecting a medication, clinicians should consider the evidence base for psychopharmacology in youth with ID/IDD and consult the literature on children with typical development when there is a lack of evidence for guidance.

Irritability and Aggression

Multiple studies in youth with ID/IDD have shown risperidone to improve symptoms of irritability and aggression as well as additional problem behaviors associated with conduct disorder (CD) and oppositional defiant disorder (ODD).[70-72,74[rct]] The positive findings usually started within 2 weeks of initiation and were sustained in 2 open-label, 48-week extension studies.[73,75[ob]] In the risperidone studies, the most common side effects were headache and somnolence. The extrapyramidal symptom profile of the medication group was comparable to placebo, and no changes were detected on electrocardiography. However, the medication group experienced more weight gain and had asymptomatic increases in prolactin. Descriptions of concurrent behavioral therapy were limited to whether it was allowed[70,71] or not allowed[71] for the duration of the study. Although these studies indicate that risperidone can improve irritability, aggression, and other behaviors associated with behavior disorders in children and adolescents with ID/IDD, because of its side effect profile risperidone is best considered after assessments of whether potential contributors to irritability and aggression could be addressed by nonpharmacological means.

Hyperactivity and Inattention

Stimulants. Guidelines on the pharmacologic management of ADHD for children with typical development recommend methylphenidate (MPH) as the first line agent[86]; however, most medication trials of methylphenidate exclude children with ID/IDD. Two RCTs of MPH in children with ID/IDD have been performed (N = 122[77]; N = 24[76]), the larger of which found methylphenidate to be effective in about 40% of children with ID/IDD and an *ICD-10* diagnosis of hyperkinetic disorder, with an effect size of 0.39 to 0.52.[74[rct]] This effect size is consistent with that in the other RCT in youths with ID[76[rct]], but is lower than the effect size reported in studies of typically developing children (eg, the Multimodal Treatment of Attention Deficit Hyperactivity Disorder Study (MTA), which reported an effect size of 0.8−0.9).[87[rct]] The adverse effects experienced by children with ID/IDD in these RCTs were similar to those observed in children with typical development with ADHD, primarily appetite suppression and sleep problems.[76,77,] The study by Simonoff *et al.*[77] did not find the efficacy of MPH to be moderated by level of ID, the presence or absence of autistic symptoms, or the severity of ADHD symptoms. These studies suggest that despite a lower effect size in children with ID versus children with typical development, MPH can be considered in children with ADHD and ID/IDD regardless of severity of ID/IDD or ADHD or presence of ASD.

Atypical Antipsychotics. Two large RCTs of risperidone that targeted irritability and aggression in children with ID/IDD and disruptive behavior disorders (DBDs) also reported improvements in hyperactivity as a secondary outcome.[72,74[rct]] These trials permitted children with co-occurring ADHD to continue their stimulant medication. A post hoc analysis combining these studies and selecting the patients with ADHD (in addition to ID and DBD) suggested that the addition of risperidone to a stimulant resulted in better control of hyperactivity than was achieved with stimulant treatment alone, without causing an increase in adverse events.[78[rct]] In a comparison study of risperidone and MPH for treatment of ADHD in children 6 to 16 years of age, Filho *et al.*[79[rct]] found both medications to reduce inattention and hyperactivity, in addition to ODD symptoms, with more pronounced effects for risperidone. Despite the potential efficacy of risperidone for the treatment of ADHD, due to risperidone's side effect profile, MPH remains the first-line agent.

Other Agents. A single study reported improvement in ADHD symptoms in children with ID/IDD taking an α-2 agonist, clonidine.[80[rct]] It is reasonable to assume that other α-2 agonists, such as guanfacine, could be similarly efficacious, although there are no trials of guanfacine in more than 10 children with ID/IDD. Potential side effects with α-2 agonists of depression, sleep disturbance, sedation, cardiac disturbances, and cognitive dulling should be taken into consideration.[1]

Anxiety and Depression. There have been no new studies since 1999 on pharmacologic treatment of anxiety or depressive disorders in children with ID/IDD, and the

treatment approach continues to be similar to that for children without ID/IDD. For depression and anxiety in children with ID/IDD, selective serotonin reuptake inhibitors (SSRIs, ie, fluoxetine and sertraline) continue to be the treatment of choice because of their evidence for efficacy in typically developing youths. α-Agonists (clonidine, guanfacine) and β-blockers are sometimes used for management of anxiety. However, there are no trials investigating the use of these medications for this indication in children with ID/IDD. Benzodiazepines are not generally recommended for the treatment of chronic anxiety in children with ID/IDD, because of concern for potential heightened sensitivity to behavioral side effects such as disinhibition.[88]

Mania and Psychosis. There have been no new studies since 1999 on pharmacologic treatment of bipolar or psychotic disorders or schizophrenia in children with ID/IDD, and treatment continues to be similar to that for children without ID/IDD, namely, use of mood stabilizers (ie, valproic acid, lithium, etc). For the treatment of psychotic disorders, newer atypical antipsychotics (ie, risperidone and aripiprazole) are generally preferred over older, first-generation antipsychotics (ie, haloperidol) because of possible increased sensitivity to extrapyramidal symptoms in the ID/IDD population.[89]

Sleep Disorders. One RCT has reported melatonin to be effective in improving sleep in adolescents with ID/IDD.[81[rct]] The long-term use of benzodiazepine hypnotics and antihistamines should be approached cautiously because of potential side effects, including disinhibition.

Medication Selection and Monitoring. The choice of a psychotropic medication in a child with ID/IDD should proceed from diagnosis of a *DSM-5* psychiatric disorder and should be part of a comprehensive treatment plan. Prescribing medication for a behavioral problem, such as aggression, self-injury, or property destruction, should be minimized if possible because the behavior may be due to a variety of disorders or other factors (ie, ADHD, anxiety, medical issues, communication deficits). Medication targeting a behavioral problem is best limited to individuals who pose a risk of injury to self or others, have severe impulsivity, are at risk for losing access to an important service (ie, foster home, school, or residential placement), or if other treatments have failed. Psychotropic medications should not be used as a substitute for appropriate services. In patients with medical comorbidities, such as a seizure disorder, opportunities for simplification and consolidation of medication regimens should be considered (ie, choosing an anticonvulsant drug with desired psychotherapeutic effects). As-needed (PRN) medication use for behavioral problems has the potential for overuse, and clear and specific indications for use should be provided and the frequency monitored.

Previous guidelines from the AACAP[57] recommend that once medication is initiated for a child with ID/IDD, the clinician should monitor response. The most commonly used outcome measure in psychopharmacology studies in ID/IDD is the Aberrant Behavior Checklist,[90] with subscales on hyperactivity, social withdrawal, lethargy, stereotypy, and irritability. The Conners Clinical Index (Conners CI)[91] and the Swanson, Nolan, and Pelham Questionnaire (SNAP)[92] measures are also frequently used in children with ID and ADHD. Some of the assessment measures in the Psychiatric Symptom Measures section of this document can also be used to monitor response to treatment. If measures are not available, use of metrics, such as number of outbursts per day, may improve the ability to track symptoms.

Children with ID/IDD may be more sensitive to side effects of medication, such as irritability, cognitive impairment, or sedation. Because children with ID/IDD may be less able to communicate subjectively experienced adverse events, particular care should be taken to elicit adverse effects when prescribing and monitoring medications, such as through the use of visual aids to enhance communication. Scales measuring involuntary movements such as the Abnormal Involuntary Movement Scale (AIMS)[93] can be used. Because many patients with ID/IDD engage in stereotypic movements that may be difficult to differentiate from involuntary movements secondary to medication, careful pretreatment documentation of baseline movements is important.

Statement 3: Specialized treatment providers and settings can be considered in treatment-refractory cases. [OP]

Although it is within the purview of CAPs to render high-quality psychiatric care for youths with ID/IDD, there may be cases in which referral to a psychiatrist, psychiatric treatment program, or developmental–behavioral pediatrician specializing in the ID/IDD population may be beneficial. In addition, although studies of specialized schools, residential settings, and other, more intensive treatment programs are generally lacking, there is preliminary evidence for improvement in irritability (aggression, self-injury, and tantrums) in children with ID/IDD or ASD, and decreased length of stay and readmission rates, associated with hospitalization in specialized child psychiatric

units.[94[ob],95[ob],96[ob]] Some institutions offer advanced training for psychiatrists in developmental neuropsychiatry.

PARAMETER LIMITATIONS

AACAP Practice Parameters are developed to assist psychiatrists in psychiatric decision making. These Parameters are not intended to define the standard of care or to guarantee successful treatment of individual patients, nor should they be deemed inclusive of all proper methods of care or exclusive of other methods of care directed at obtaining the desired results. These Parameters do not usurp sound clinical judgment. The ultimate judgment regarding the care of a particular patient must be made by the psychiatrist in light of all the circumstances, values, and preferences presented by the patient and his/her family, the diagnostic and treatment options available, and the accessible resources.

This Practice Parameter was developed by Matthew Siegel, MD, Kelly McGuire, MD, MPA, Jeremy Veenstra-VanderWeele, MD, Katherine Stratigos, MD, Bryan King, MD, and the American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (CQI) members: Christopher Bellonci, MD, Munya Hayek, MD, Helene Keable, MD, Carol Rockhill, MD, PhD, MPH and AACAP CQI Co-Chairs Oscar G. Bukstein, MD, MPH, and Heather J. Walter, MD, MPH.

AACAP Practice Parameters are developed by topic experts under the direction of the AACAP CQI with review by representatives from multiple constituent groups, including additional topic experts, AACAP members, relevant AACAP committees, the AACAP Assembly of Regional Organizations, and the AACAP Council. Final approval of a Practice Parameter as an AACAP Official Action is conferred by the AACAP Council.

The primary intended audience for the AACAP Practice Parameters is child and adolescent psychiatrists; however, the information presented also could be useful for other medical or behavioral health clinicians.

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