Practice Parameter for the Assessment and Treatment of Children and Adolescents With Autism Spectrum Disorder

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Autism spectrum disorder is characterized by patterns of delay and deviance in the development of social, communicative, and cognitive skills that arise in the first years of life. Although frequently associated with intellectual disability, this condition is distinctive in its course, impact, and treatment. Autism spectrum disorder has a wide range of syndrome expression and its management presents particular challenges for clinicians. Individuals with an autism spectrum disorder can present for clinical care at any point in development. The multiple developmental and behavioral problems associated with this condition necessitate multidisciplinary care, coordination of services, and advocacy for individuals and their families. Early, sustained intervention and the use of multiple treatment modalities are indicated. J. Am. Acad. Child Adolesc. Psychiatry, 2014;53(2):237–257. **Key Words:** autism, Practice Parameters, guidelines, developmental disorders, pervasive developmental disorders

Since the first Practice Parameter for the Assessment and Treatment of Children, Adolescents, and Adults with Autism and Other Pervasive Developmental Disorders[1] was published, several thousand research and clinical articles have appeared and the diagnostic criteria for autism have changed. This Parameter revision provides the opportunity to update the previous version and incorporate new research. Because the extant body of research was performed under the *DSM-IV-TR* diagnostic schema, the evidence will be presented using that terminology. This Parameter is applicable to evaluation of children and adolescents (≤17 years of age) but often will have some relevance to adults. This document presumes basic familiarity with aspects of normal child development and child psychiatric diagnosis and treatment. Unless otherwise noted, the term *child* refers to adolescents and younger children, and *parents* refers to the child's primary caretakers regardless of whether they are the biological or adoptive parents or legal guardians.

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METHODOLOGY

The first version of this Parameter was published in 1999. For this revision, the literature search covered the period from 1991 to March 19, 2013 using the PubMed, PsycINFO, Cochrane, and CINAHL (EBSCO) databases. The initial searches were inclusive and sensitive. Search terms were a combination of MeSH headings and keywords, and the MeSH headings were adjusted to terms used by PsycINFO and CINAHL by using their thesauri.

In PubMed the MeSH terms *autistic disorder, childhood development disorders—pervasive, Asperger*,* and *Rett** and the keyword *autism* were searched. The initial search yielded 20,807 results. Then, the results were limited to English, human, *all child (0 to 18 years)*, and 1991 to March 19, 2013. Additional limits included classic article, clinical trial, comparative study, controlled clinical trial, evaluation studies, guideline, historical article, meta-analysis, practice guideline, multicenter study, randomized controlled trial, review, twin study,

and validation studies. The refined PubMed search yielded 3,613 articles.

In the PsycINFO database subject headings (focused) of *autism, autistic thinking, pervasive developmental disorders, retts syndrome, aspergers,* and keyword *autism* were searched. The initial search returned 24,875 articles and was then limited to English, *childhood: birth to age 12yrs, adolescence: age 13-17 yrs, peer reviewed journal,* and 1991 to March 19, 2013. The refined PsycINFO search yielded 9,583 articles.

In the Cochrane Database of Systematic Reviews, keywords of *autism, autistic, rett*, asperger**, or (*pervasive and disorder** and *develop**) were searched without additional limits. The Cochrane search yielded 95 articles. An additional 517 articles were retrieved from the CINAHL database, after excluding Medline articles, by searching *autistic disorder, autism, asperger syndrome, child development disorders, pervasive,* and *rett syndrome*.

A total of 13,808 articles were identified and exported to the EndNote reference management program. After removing duplicate references, the resulting yield from the comprehensive search was 9,581 articles.

The titles and abstracts of all articles were reviewed. Studies were selected for full text review based on their place in the hierarchy of evidence (e.g., randomized controlled trials), quality of individual studies, and generalizability to clinical practice. The search was augmented by review of articles nominated by expert reviewers and further search of article reference lists and relevant textbook chapters. A total of 186 articles were selected for full text examination.

CLINICAL PRESENTATION AND COURSE

Autism was first described in 1943 by Kanner[2] who reported on 11 children with an apparently congenital inability to relate to other people but who were quite sensitive to change in the nonsocial environment. Kanner emphasized that the lack of interest in people was in stark contrast to the profound social interest of normal infants. He also observed that when language developed at all, it was marked by echolalia, pronoun reversal, and concreteness. The children also exhibited unusual, repetitive, and apparently purposeless activities (stereotypies). Autism was initially believed to be a form of childhood psychosis, but, by the 1970s, various lines of evidence made it clear that autism was highly distinctive. By 1980, autism was officially recognized as a diagnosis in *DSM-III*.[3]

Under *DSM-IV-TR*, the diagnosis of autism required disturbances in each of 3 domains: social relatedness, communication/play, and restricted interests and activities with onset by 3 years of age.[4] The disturbance in social relatedness is striking and includes marked impairment in nonverbal communication, peer relationships, and social-emotional reciprocity. Impairments in communication include a delay or total lack of spoken language (without an attempt to compensate through other means) or, for verbal individuals, a marked difficulty in the ability to sustain or initiate conversation, stereotyped and repetitive (or idiosyncratic) language, and lack of developmentally appropriate make-believe or social play. Impairment in interests and activities includes encompassing preoccupations, adherence to apparently nonfunctional routines or rituals, stereotypies and motor mannerisms, and persistent preoccupation with parts of objects.

There is variability in the age at which children may present the features essential for this diagnosis.[5] Preschool children with autism typically present with marked lack of interest in others, failures in empathy, absent or severely delayed speech and communication, marked resistance to change, restricted interests, and stereotyped movements. Common parental concerns include a child's lack of language, inconsistencies in responsiveness, or concern that the child might be deaf. In children with autism, social and communication skills usually increase by school age; however, problems dealing with change and transitions and various self-stimulatory behaviors (sometimes including self-injury) also may become more prominent during this time.[6] In adolescence, a small number of individuals with autism make marked developmental gains; another subgroup will behaviorally deteriorate (e.g., tantrums, self-injury, or aggression). Children and adolescents with autism have an increased risk for accidental death (e.g., drowning).[7] Predictors of ultimate outcome include the presence of communicative speech by 5 years of age and overall cognitive ability (IQ). Evidence that earlier detection and provision of services improves long-term prognosis makes early diagnosis particularly important.[8]

The *DSM-IV-TR* category of pervasive developmental disorders included autistic disorder, Rett's disorder, Asperger's disorder, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified (PDD-NOS). Rett's disorder was described by Andreas

Rett in 1966 in a series of girls with unusual hand washing/wringing stereotyped mannerisms. In most cases, Rett's disorder is caused by mutations in the gene *MeCP2* (methyl-CpG-binding protein 2).[9] Head circumference and development are normal at birth and during infancy. Before 4 years of age, head growth decelerates, purposeful hand movements are lost, and characteristic stereotyped hand movements (wringing or washing) develop.[10] The central role of *MeCP2* mutations in this disorder makes it clear that boys may carry the same mutations that lead to the full syndrome in girls, but with differing clinical manifestations ranging from fatal encephalopathy[11] to progressive but nonfatal developmental disorder[12] to nonspecific X-linked intellectual disability.[13]

Childhood disintegrative disorder (CDD) was first described by Theodor Heller in 1908.[14] This condition is characterized by a period of at least 2 years of normal development, followed by a marked deterioration and clinically significant loss of at least 2 skills in the areas of receptive or expressive language, social skills, toileting skills, play, or motor skills.[14] The onset of CDD is highly distinctive, typically occurring at 3 to 4 years of age and can be gradual or abrupt. Sometimes parents report that the child experienced a period of anxiety or dysphoria before onset of CDD symptoms. Once established, CDD resembles autism in clinical features,[14] but the outcome is poor. The child typically becomes mute or, at best, regains limited speech.

Asperger's disorder was described in 1944 but not officially recognized until *DSM-IV*. Unlike children with autism, individuals with Asperger's disorder do not present with delays in language acquisition or with unusual behaviors and environmental responsiveness during the first years of life. Consequently, parents often have no concerns about their child's early development.[15] Asperger originally described children who were precocious in learning to talk but who then talked in a formal, pedantic, 1-sided way, often about a topic of circumscribed interest.[16] Social difficulties arise due to this idiosyncratic, 1-sided social style. The outcome in Asperger's disorder generally appears to be better than that for autism, although this may, in part, relate to better cognitive and/or verbal abilities.[8,15]

The term pervasive developmental disorder not otherwise specified (PPD NOS) (also sometimes termed atypical PDD or atypical autism) encompasses subthreshold cases on the autism spectrum, e.g., cases in which full criteria for one of the explicitly defined PDDs are not met, but the child has problems in social interaction and some difficulties in communication or restricted patterns of behavior. Although studies are limited, individuals with PDD-NOS typically have been characterized as less impaired, having fewer repetitive behaviors, and having a better prognosis than persons with autism.[16]

DSM-IV-TR to *DSM-5*

Because there was little evidence to support reliable and replicable diagnostic differences among the various *DSM-IV-TR* PDDs,[17] the *DSM-5* workgroup on neurodevelopmental disorders subsumed the prior categories under the new diagnosis of autism spectrum disorder (ASD) in the *DSM-5*. Diagnostic domains were reduced from 3 to 2, focusing on social communication and interaction deficits and restricted, repetitive patterns of behaviors and interests. The strict requirement for onset before 3 years of age was changed to onset in the early developmental period, the occurrence of potential sensory abnormalities was incorporated, and a severity scale for impairments in each of the 2 core domains was included. Diagnostic reporting now includes specifiers that may enhance descriptive subtyping of the population, including specifiers for the presence or absence of intellectual impairment, language impairment, catatonia, and known medical, genetic, or environmental factors. The new criteria allow for a history of symptoms that may not be present currently, recognizing that through intervention or normal development some children with autism no longer present some symptoms later in life. It will be some years before the implications of these changes for autism prevalence and other facets of assessment and treatment can be fully assessed.

EPIDEMIOLOGY

Many studies, mostly conducted outside the United States, have examined the prevalence of autism or, less commonly, ASD or PDDs.[17] Of the approximately 36 surveys of autism available, prevalence estimates for autistic disorder range from 0.7 in 10,000 to 72.6 in 10,000.[18] The variability in estimates reflects different factors, including changes in definition. When the 18 surveys conducted since the introduction of the *DSM-IV* criteria are considered, estimates ranging from 10 in 10,000 to 16 in 10,000, with a median prevalence of 13 in 10,000, are obtained.[18] The most recent study by the Centers for Disease

Control and Prevention estimated the prevalence of ASD in the United States as 11.3 in 1,000.[19] Contrary to popular perception, data from 7 surveys suggest that rates of Asperger's disorder are in fact *lower* than for typical autism (2.6 in 10,000 or one fifth as common as typical autism).[18]

Recent observations of higher rates of autism have led to concern that the prevalence of this disorder may be increasing. Various factors may contribute to an apparent increase,[20] such as differences in diagnostic criteria and diagnostic practices, the age of children screened, and the location of the study (see Fombonne[18] for discussion).

Autism is approximately 4 times more common in males than in females, but females with autism tend to have more severe intellectual disability. Although the original report by Kanner[2] suggested a predominance of autism in more educated families, subsequent work has not shown this. Current approaches to the diagnosis of ASD appear to work well internationally and cross-culturally,[3] although cultural aspects of the condition have not received much attention.[21] Within the United States, there may be underdiagnosis in some circumstances (e.g., in disadvantaged inner-city children).[22]

ETIOLOGY

Neurobiology

Electroencephalographic (EEG) abnormalities and seizure disorders are observed in as many as 20% to 25% of individuals with autism.[23] The high rates of epilepsy suggest a role for neurobiologic factors in autism.[13,24,25] The number of areas affected by autism suggests that a diverse and widely distributed set of neural systems must be affected. Although various theories have posited potential loci for difficulties, definitive data are lacking. Postmortem studies have shown various abnormalities, particularly within the limbic system.[25] Functional magnetic resonance imaging procedures have identified difficulties in tasks involving social and affective judgments and differences in the processing of facial and nonfacial stimuli.[26] Structural magnetic resonance imaging has shown an overall brain size increase in autism, and diffusion tensor imaging studies have suggested aberrations in white matter tract development.[27] One of the most frequently replicated neurochemical findings has been the elevation of peripheral levels of the neurotransmitter serotonin. The significance of this finding remains unclear. A role for dopamine is suggested given the problems with overactivity and stereotyped mannerisms and the positive response of such behaviors to neuroleptic medications.[28]

During the past decade, much concern has focused on vaccines as a possible postnatal environmental cause for ASD, with the concern focused on the possibility that the measles-mumps-rubella vaccine may cause autism or that thimerosal (a mercury-containing preservative now removed from all single-dose vaccines) might do so.[29] The preponderance of available data has not supported either hypothesis (see Rutter[30] for a review). However, a possible role of the immune system in some cases of autism has not been ruled out.[31]

Neuropsychological correlates of ASD include impairments in executive functioning (e.g., simultaneously engaging in multiple tasks),[32] weak central coherence (integrating information into meaningful wholes),[33] and deficits in theory-of-mind tasks (taking the perspective of another person).[34]

Familial Pattern and Genetic Factors

The high recurrence risk for autism in siblings and even higher concordance for autism in identical twins has provided strong support for the importance of genetic factors.[30] Higher rates of autism are consistently noted in siblings of affected children. Recurrence risk has typically been cited at 2% to 10%, but a recent prospective longitudinal study has reported a rate of 18.7% when the broad autism spectrum is considered.[35] Identified risk factors for ASD appear to include closer spacing of pregnancies, advanced maternal or paternal age, and extremely premature birth (<26 weeks' gestational age).[36-38] In addition, high rates of learning/language problems and social disability and a possible increase in the risk for mood and anxiety disorders has been noted in family members.

It is now clear that multiple genes are involved in autism.[30,39] Over the past several years, studies have supported a role for common (present in >5% of the general population) and rare genetic variations contributing to autism.[40] The rate of progress in gene discovery has been increasing rapidly over the past several years and these results are already beginning to influence clinical practice with regard to genetic testing, as noted below.[41]

DIFFERENTIAL DIAGNOSIS

ASD must be differentiated from specific developmental disorders (including language disorders),

sensory impairments (especially deafness), reactive attachment disorder, obsessive-compulsive disorder, intellectual disability, anxiety disorders including selective mutism, childhood-onset schizophrenia, and other organic conditions.

A diagnosis of autism is made when the requisite *DSM-5* symptoms are present and other disorders have been adequately ruled out. In autism it is typical for parents to report that there was no period of normal development or that there was a history of unusual behaviors (e.g., the child seemed too good and undemanding as an infant). Less commonly, a period of apparently normal development is reported before a regression (loss of skills). The topic of regression in autism remains an active area of current investigation. Developmental regression is typical in Rett syndrome but also can be observed in other conditions (e.g., childhood-onset schizophrenia or degenerative CNS disorders).

Developmental language disorders have an impact on socialization and may be mistaken for an ASD. The distinction is particularly difficult in preschool children. However, 2 behaviors have been reported to consistently differentiate autistic children from language-impaired peers at 20 and 42 months of age, namely pointing for interest and use of conventional gestures.[42] Similarly, differentiating mild to moderate developmental delay from ASD may be difficult, particularly when evaluating the younger child (see Chawarska and Volkmar[42] for a detailed discussion). One study identified some items on the Autism Diagnostic Interview that differentiated between these 2 groups at 24 months, especially directing attention (showing) and attention to voice (Table 1).[43-56] At 36 months, 4 items correctly classified all subjects: use of other's body, attention to voice, pointing, and finger mannerisms. From 38 to 61 months, children with autism were more likely to show impaired nonverbal behaviors (such as eye contact) to regulate social interaction. In childhood, there may be diagnostic overlap between ASD and attention-deficit/hyperactivity disorder, making the differential diagnosis difficult.[57,58]

Children with reactive attachment disorder may exhibit deficits in attachment and therefore inappropriate social responsivity, but these usually improve substantially if adequate caretaking is provided. Obsessive-compulsive disorder has a later onset than ASD, is not typically associated with social and communicative impairments, and is characterized by repetitive patterns of behavior that are ego dystonic. Symptoms that characterize anxiety disorders, such as excessive worry, the need for reassurance, the inability to relax, and feelings of self-consciousness, are also seen in ASD, particularly in higher functioning individuals. However, the 2 conditions can be differentiated by the prominent social and communicative impairments seen in ASD but not anxiety disorders, and the developed social insight of children with anxiety disorders, which is not seen in ASD. Differentiating childhood schizophrenia from autism can be difficult, because they are characterized by social impairments and odd patterns of thinking. However, florid delusions and hallucinations are rarely seen in autism.

COMORBIDITIES

Given difficulties in communication (e.g., mutism) and cognitive impairment, issues of comorbidity in ASD can be quite complex. The process of diagnostic overshadowing (the tendency to fail to diagnose other comorbid conditions when a more noticeable condition is present) may occur.[59] Attempts to determine comorbidity prevalence in ASD have been hampered by methodologic issues, although most studies have shown increased rates of anxiety and attentional disorders.[60]

In most epidemiologically based samples of persons with autistic disorder, approximately 50% exhibit severe or profound intellectual disability, 35% exhibit mild to moderate intellectual disability, and the remaining 20% have IQs in the normal range.[18] For children with autistic disorder, verbal skills are typically more impaired than nonverbal skills. For children with Asperger's disorder, the reverse pattern is sometimes observed and the profile of nonverbal learning disability may be present.[61] Clearly, intellectual impairment is not an essential diagnostic feature of autism, and thus it is necessary and important for the diagnosis of intellectual disability to be made.

A range of behavioral difficulties can be observed in ASD, including hyperactivity, obsessive-compulsive phenomena, self-injury, aggression, stereotypies, tics, and affective symptoms. The issue of whether these qualify as additional disorders is complex.[3] Affective symptoms are frequently observed and include lability, inappropriate affective responses, anxiety, and depression. Impairments in emotion regulation processes can lead to under- and

TABLE 1 Summary of Selected Assessment Instruments for Autism Spectrum Disorder[a]

Scale (see legend)	Uses	Age Range	Method of Administration	Population Studied	Scale characteristics	Reference
ABC	screening	children	parent rated	AD	57 items, scale 1-4	Krug *et al.*, 1980[43]
CARS	screening	children	clinician rated	AD	15 items, scale 1-4	Schopler *et al.*, 1980[44]
M-CHAT	screening	toddlers	parent rated	AD	23 items, yes/no	Robins *et al.*, 2001[45]
CSBS-DP-IT-Checklist	screening	toddlers	parent rated	AD	24 items	Wetherby *et al.*, 2008[46]
ASQ	screening	child/adult	parent rated	AD/AspD	40 items, yes/no	Berument *et al.*, 1999[47]
AQ	screening	child/adult	self or parent rated	AspD	50 items, scale 0-3	Baron-Cohen *et al.*, 2001[48]
CAST	screening	4-11 years	parent rated	AspD	37 items, yes/no	Scott *et al.*, 2002[49]
ASDS	screening	5-18 years	parent or teacher rated	AspD	50 items, yes/no	Myles *et al.*, 2000[50]
GADS	screening	3-22 years	parent or teacher rated	AspD	32 items, scale 0-3	Gilliam, 2001[51]
ASDI	screening	child/adult	interview + clinician rated	AspD	50 items, yes/no	Gillberg *et al.*, 2001[52]
SRS	screening	4-18 years	parent or teacher rated	AspD	65 items, scale 1-4	Constantino *et al.*, 2003[53]
ADI	diagnostic	child/adult	interview + clinician rated	AD/AspD	see text	Lord *et al.*, 2003[54]
DISCO	diagnostic	child/adult	interview + clinician rated	AD/AspD	see text	Wing *et al.*, 2002[55]
ADOS	diagnostic	child/adult	semi-structured interactive session	AD/AspD	see text	Lord *et al.*, 1994[56]

Note: ABC = Autism Behavior Checklist; AD = autism disorder; ADI = Autism Diagnostic Interview–Revised; ADOS = Autism Diagnostic Observation Schedule; AQ = Autism Quotient; ASDI = Asperger Syndrome Diagnostic Interview; ASDS = Asperger Syndrome Diagnostic Scale; AspD = Asperger's disorder; ASQ = Autism Screening Questionnaire; CARS = Childhood Autism Rating Scale; CAST = Childhood Autism Screening Test; M-CHAT = Checklist for Autism in Toddlers; CSBS-DP-IT-Checklist = Communication and Symbolic Behavior Scales Developmental Profile Infant-Toddler Checklist; DISCO = Diagnostic Interview for Social and Communication Disorders; GADS = Gilliam Asperger's Disorder Scale; Parent = primary caregiver; SRS = Social Responsiveness Scales.

[a]Note that these instruments may need to be revised to provide evidence of validity for DSM-5 ASD and supplement but DO NOT REPLACE clinical diagnosis.

over-reactivity.[62] Overt clinical depression is sometimes observed and this may be particularly true for adolescents with Asperger's disorder.[15] Case reports and case series have suggested possible associations with bipolar disorders and tics and Tourette's syndrome. Bullying involvement, including victimization and perpetration, occurs more frequently in general educational settings.[63]

Attentional difficulties also are frequent in autism, reflecting cognitive, language, and social problems.[64] The historical prohibition on making an additional diagnosis of attention-deficit/hyperactivity disorder in those with ASD has been removed in the *DSM-5*. Notably, a subset of children with ASD with elevated scores for hyperactivity showed a 49% response rate in a large randomized controlled trial of methylphenidate treatment.[64]

EVIDENCE BASE FOR PRACTICE PARAMETERS

In this Parameter, recommendations for best assessment and treatment practices are stated in accordance with the strength of the underlying empirical and/or clinical support.

- Clinical standard [CS] is applied to recommendations that are based on rigorous empirical evidence (e.g., meta-analyses, systematic reviews, individual randomized controlled trials) and/or overwhelming clinical consensus.
- Clinical guideline [CG] is applied to recommendations that are based on strong empirical evidence (e.g., nonrandomized controlled trials, cohort studies, case-control studies) and/or strong clinical consensus.
- Clinical option [OP] is applied to recommendations that are based on emerging empirical evidence (e.g., uncontrolled trials or case series/reports) or clinical opinion but lack strong empirical evidence and/or strong clinical consensus.
- Not endorsed [NE] is applied to practices that are known to be ineffective or contraindicated.

The strength of the empirical evidence is rated in descending order as follows:

- [rct] Randomized controlled trial is applied to studies in which subjects are randomly assigned to at least 2 treatment conditions.
- [ct] Controlled trial is applied to studies in which subjects are nonrandomly assigned to at least 2 treatment conditions.
- [ut] Uncontrolled trial is applied to studies in which subjects are assigned to 1 treatment condition.
- [cs] Case series/report is applied to a case series or a case report.

ASSESSMENT

Recommendation 1. The developmental assessment of young children and the psychiatric assessment of all children should routinely include questions about ASD symptomatology [CS].

Screening should include inquiries about the core symptoms of ASD, including social relatedness and repetitive or unusual behaviors. Screening instruments have been developed that may be helpful to the clinician. Some of these instruments are completed by clinicians and others by primary caregivers (Table 1).[43-56] Screening is applicable to young children and to infants, when the diagnosis may first be considered. In some instances, screening may be relevant to older children, e.g., those who are more intellectually able and whose social disability is therefore more likely to be detected later.

Recommendation 2. If the screening indicates significant ASD symptomatology, a thorough diagnostic evaluation should be performed to determine the presence of ASD [CS].

Currently, biological diagnostic markers are not available and diagnosis rests on careful examination of the child. A standard psychiatric assessment should be followed,[65] including interviews with the child and family and a review of past records and historical information. The history and examination should be conducted with careful consideration of *DSM-5* diagnostic criteria. Although the *DSM-5* criteria are intended to be independent of age and intellect, the diagnosis of autism in infants and very young children is more challenging, and some features (e.g., stereotyped movements) may develop later.[5] Systematic attention to the areas relevant to differential diagnosis is essential. Information on the nature of changes over the course of development, e.g., in response to intervention, is helpful. The history should include a review of past and current educational and behavioral interventions and information regarding family history and relevant psychosocial issues. Consideration of possible comorbid diagnoses is an important focus of assessment.

Observation of the child should focus on broad areas of social interaction and restricted, repetitive behaviors. The child's age and developmental level may dictate some modification in assessment procedures. Clinicians should be sensitive to ethnic, cultural, or socioeconomic factors that may affect assessment.

Various instruments for the assessment of ASD have been developed (Table 1[43-56], see Coonrod and Stone[66] for a review). As a practical matter, all these instruments vary in their usefulness for usual clinical practice. Some require specific training. The use of such instruments supplements, but does not replace, informed clinical judgment.[3]

Recommendation 3. Clinicians should coordinate an appropriate multidisciplinary assessment of children with ASD [CS].

All children with ASD should have a medical assessment, which typically includes physical examination, a hearing screen, a Wood's lamp examination for signs of tuberous sclerosis, and genetic testing, which may include G-banded karyotype, fragile X testing, or chromosomal microarray. In a community sample of children with ASD, diagnostic yields were 2.5% for karyotype testing, 0.57% for fragile X testing, and 24% for chromosomal microarray.[67] Chromosomal microarray has been recommended by medical geneticists as the standard of care for the initial evaluation of children with developmental disabilities and/or ASDs.[68] These tests currently detect known abnormalities clearly associated with increased rates of ASD (e.g., 15q11-13 maternal duplications and duplications and deletions of chromosome 16p11.2) and genetic variations of uncertain significance. Recent data from a study of families with only a single affected child have shown that lower IQ is not a strong predictor of a positive chromosomal finding.[69] Any abnormal or indeterminate result from such a study warrants referral for further genetic evaluation and counseling. The yield of genetic testing in the presence of clinical suspicion is currently in the range of at least one third of cases.[70]

Unusual features in the child (e.g., history of regression, dysmorphology, staring spells, family history) should prompt additional evaluations. The list of potential organic etiologies is large but falls into the categories of infectious (e.g., encephalitis or meningitis), endocrinologic (e.g., hypothyroidism), metabolic (e.g., homocystinuria), traumatic (e.g., head injury), toxic (e.g., fetal alcohol syndrome),[4] or genetic (e.g., chromosomal abnormality). Certain developmental disorders, most notably Landau-Kleffner syndrome, also should be ruled out. In this condition, a highly distinctive EEG abnormality is present and associated with development of a marked aphasia.[71] Genetic or neurologic consultation, neuroimaging, EEG, and additional laboratory tests should be obtained when relevant, based on examination or history (e.g., testing for the *MeCP2* gene in cases of possible Rett's disorder).[72]

Psychological assessment, including measurements of cognitive ability and adaptive skills, is indicated for treatment planning and helps to frame observed social-communication difficulties relative to overall development. The results of standard tests of intelligence may show considerable scatter. Unusual islets of ability ("splinter skills") may be present. For children with autism, these sometimes take the form of unusual ability ("savant skills"), e.g., the ability to produce intricate drawings or engage in calendar calculations. For higher functioning children, areas of special interest are often present and the single-minded pursuit of these interests may interfere with the child's ability to learn. Psychological tests clarify areas of strength and weakness useful in designing intervention programs and may need to include instruments valid for a nonverbal population.[7]

Communication assessment, including measurements of receptive and expressive vocabulary and language use (particularly social or pragmatic), is helpful for diagnosis and treatment planning.[73] Occupational and physical therapy evaluations may be needed to evaluate sensory and/or motor difficulties.[74] Sleep is an important variable to assess in individuals with ASD.[75] When members of multiple disciplines are involved in assessment, it is optimal that coordination occur among the various professionals.

TREATMENT

Recommendation 4. The clinician should help the family obtain appropriate, evidence-based, and structured educational and behavioral interventions for children with ASD [CS].

Structured educational and behavioral interventions have been shown to be effective for many children with ASD[76] and are associated with better outcome.[8] As summarized in the National Research Council report,[76] the quality of the research literature in this area is variable,

with most studies using group controls or single-subject experimental methods. In general, studies using more rigorous randomized group comparisons are sparse, reflecting difficulties in random assignment and control comparisons. Other problems include lack of attention to subject characterization, generalization of treatment effects, and fidelity of treatment implementation. Despite these problems, various comprehensive treatments approaches have been shown to have efficacy for groups of children, although none of the comprehensive treatment models has clearly emerged as superior.[76]

Behavioral

Behavioral interventions such as Applied Behavioral Analysis (ABA) are informed by basic and empirically supported learning principles.[77] A widely disseminated comprehensive ABA program is Early Intensive Behavioral Intervention for young children, based on the work of Lovaas *et al.*[78] Early Intensive Behavioral Intervention is intensive and highly individualized, with up to 40 hours per week of one-to-one direct teaching, initially using discrete trials to teach simple skills and progressing to more complex skills such as initiating verbal behavior. A meta-analysis found Early Intensive Behavioral Intervention effective for young children but stressed the need for more rigorous research to extend the findings.[79] Behavioral techniques are particularly useful when maladaptive behaviors interfere with the provision of a comprehensive intervention program. In such situations, a functional analysis of the target behavior is performed, in which patterns of reinforcement are identified and then various behavioral techniques are used to promote a desired behavioral alternative. ABA techniques have been repeatedly shown to have efficacy for specific problem behaviors,[80] and ABA has been found to be effective as applied to academic tasks,[81[ut]] adaptive living skills,[82[ut]] communication,[83[ut]] social skills,[84[ut]] and vocational skills.[85[ct]] Because most children with ASD tend to learn tasks in isolation, an explicit focus on generalization is important.[86]

Communication

Communication is a major focus of intervention and typically will be addressed in the child's individualized educational plan in coordination with the speech-language pathologist. Children who do not yet use words can be helped through the use of alternative communication modalities, such as sign language, communication boards, visual supports, picture exchange, and other forms of augmentative communication. There is some evidence for the efficacy of the Picture Exchange Communication System, sign language, activity schedules, and voice output communication aids.[87[rct],88-90] For individuals with fluent speech, the focus should be on pragmatic language skills training. Children and adolescents with fluent speech may, for example, be highly verbal but have severely impaired pragmatic language skills that can be addressed through explicit teaching. Many programs to enhance social reciprocity and pragmatic language skills are currently available (Table 2; see Reichow and Volkmar[91] for an extensive review).[92-103]

Educational

There is consensus that children with ASD need a structured educational approach with explicit teaching.[76] Programs shown to be effective typically involve planned, intensive, individualized intervention with an experienced, interdisciplinary team of providers, and family involvement to ensure generalization of skills. The educational plan should reflect an accurate assessment of the child's strengths and vulnerabilities, with an explicit description of services to be provided, goals and objectives, and procedures for monitoring effectiveness. Although the curricula used vary across programs, they often share goals of enhancing verbal and nonverbal communication, academic skills, and social, motor, and behavioral capabilities. In some instances, particularly for younger children, a parent-education and home component may be important. Development of an appropriate individualized educational plan is central in providing effective service to the child and family. Efficacy has been shown for 2 of the structured educational models, the Early Start Denver Model[104[rct]] and the Treatment and Education of Autism and related Communication handicapped Children program,[105[ct]] but significant challenges remain in disseminating knowledge about effective interventions to educators.

Other Interventions

There is a lack of evidence for most other forms of psychosocial intervention, although cognitive behavioral therapy has shown efficacy for anxiety and anger management in high functioning youth with ASD.[106[rct],107[rct]] Studies of sensory oriented interventions, such as auditory integration

TABLE 2 Methods Available for the Delivery of Social Reciprocity/Pragmatic Language-Oriented Interventions

Developmental Level	Method	Notes	Reference
Infant/preschool (play based)	guided participation	adult coaching and mediation by trained peers	Schuler and Wolfberg, 2002[92]
	Do-Watch-Listen-Say	careful selection of play materials to foster participation; organization of environment to facilitate participation and cooperation	Quill, 2000[93]
	play organizers	neurotypical peers taught to encourage sharing, helping, and praising to facilitate play; some evidence of generalization	Strain *et al.*, 1977[94]
	buddy skills	teaches neurotypical peers to stay with, play with, and talk to their "buddies"; some evidence of improvement in the frequency of social communication that was generalized to other interactions	Goldstein and Wikstrom, 1996[95]
School age	social stories	state a problem and give the child an acceptable response to it; usually focuses on maladaptive behaviors; little evidence of generalization and maintenance	Gray, 2000[96]
	social skills groups	see text	Kamps *et al.*, 1997[97]
	peer network/circle of friends	typical peers taught to initiate and model appropriate social interactions; results have shown improvement in interaction and generalization to new settings	Kamps *et al.*, 1997[97]; Whitaker *et al.*, 1998[98]
Adolescence	peer network/circle of friends	see above	Whitaker *et al.*, 1998[98]; Paul, 2003[99]
	visual schedule/verbal rehearsal	using written and pictorial representations of expected activities and behavior	Klin and Volkmar, 2000[100]; Hodgdon, 1995[101]
	social skills group	see text	Paul, 2003[99]
	social thinking	addresses underlying social cognitive knowledge required for expression of related social skills; promotes teaching the "why" behind socialization	Crooke *et al.*, 2007[102]
	training scripts	scripts are provided that give the opportunity to ask questions in response to others = initiation of conversation	Klin and Volkmar, 2000[103]

training, sensory integration therapy, and touch therapy/massage, have contained methodologic flaws and have yet to show replicable improvements.[108,109] There is also limited evidence thus far for what are usually termed developmental, social-pragmatic models of intervention, such as Developmental-Individual Difference-Relationship Based/Floortime, Relationship Development Intervention, Social Communication Emotional Regulation and Transactional Support, and Play and Language for Autistic Youths, which generally use naturalistic techniques in the child's community setting to develop social communication abilities. Children with ASD are psychiatrically hospitalized at substantially higher rates than the non-ASD child population.[110] The efficacy of this intervention is unknown, although there

TABLE 3 Randomized Controlled Trials of Psychotropic Medications in Children and Adolescents With Autism Spectrum Disorder (ASD)

Agent	Study	Target Symptoms	Dose	Demographics	Significant Side Effects	Primary Outcome(s)
α_2 Agonists						
Clonidine	Jaselskis *et al.*, 1992[116]	hyperactivity, irritability, inappropriate speech, stereotypy	0.15-0.20 mg divided 3×/d	8 children 5-13 y old	hypotension, drowsiness	statistically and clinically relevant decrease in ABC Irritability subscale
Guanfacine	Handen *et al.*, 2008[117]	hyperactivity, inattention	1-3 mg divided 3×/d	7 children with ASD 5-9 y old	drowsiness, irritability	45% with >50% decrease in ABC Hyperactivity subscale
Antipsychotics						
Aripiprazole	[b]Marcus *et al.*, 2009[118]	irritability, hyperactivity, stereotypy, social withdrawal, inappropriate speech	5, 10, or 15 mg/d fixed dose	218 children 6-17 y old	somnolence, weight gain, drooling, tremor, fatigue, vomiting	56% positive response[a] for aripiprazole 5 mg vs. 35% on placebo; significant improvement in Irritability, Hyperactivity, and Stereotypy subscales
	[b]Owen *et al.*, 2009[119]	irritability, hyperactivity, stereotypy, social withdrawal inappropriate speech	5-15 mg/d flexibly dosed	98 children 6-17 y old	somnolence, weight gain, drooling, tremor, fatigue, vomiting	52% positive response[a] for aripiprazole vs. 14% on placebo; significant improvement in Irritability, Hyperactivity, and Stereotypy subscales
Haloperidol	Anderson *et al.*, 1984[120]	multiple behavioral symptoms, global functioning	0.5-4 mg/d	40 children 2-7 y old	sedation, irritability, extrapyramidal symptoms (>25%)	behavioral symptoms improved with significant decrease in 8 of 14 items of CPRS
	Anderson *et al.*, 1989[121]	multiple behavioral symptoms, global functioning	0.25-4 mg/d	45 children 2-7 y old	sedation, extrapyramidal symptoms	behavioral symptoms improved with significant decrease in 7 of 14 items of CPRS
Olanzapine	[b]Hollander *et al.*, 2006[122]	global functioning, aggression, compulsions, irritability	7.5-12.5 mg/d	11 children 6-14 y old	weight gain, sedation	50% of those on olanzapine much or very much improved in global functioning vs. 20% on placebo
Risperidone	RUPP, 2002[123]	irritability, hyperactivity, stereotypy, social withdrawal, inappropriate speech	0.5-3.5 mg/d	101 children 5-17 y old	weight gain, increased appetite, fatigue, drowsiness, drooling, dizziness	69% had positive response[a] on risperidone vs. 12% positive response[a] on placebo; significant positive findings for hyperactivity and stereotypy

TABLE 3 Continued

Agent	Study	Target Symptoms	Dose	Demographics	Significant Side Effects	Primary Outcome(s)
	[b]Shea *et al.*, 2004[124]	irritability, hyperactivity, stereotypy, social withdrawal, inappropriate speech	0.02-0.06 mg/kg/d	79 children 5-12 y old	weight gain, somnolence,	64% improvement in ABC Irritability subscale on risperidone vs. 31% improvement on placebo; significant positive finding for hyperactivity
	McDougle *et al.*, 2005[125]	social and communication impairment, repetitive behavior and stereotypy	0.5-3.5 mg/d	101 children 5-17 y old	weight gain, increased appetite, fatigue, drowsiness, drooling, dizziness	significant response[c] for repetitive behavior and stereotypy on risperidone
Risperidone vs. haloperidol	[b]Miral *et al.*, 2008[126]	behavior, social, sensory, language	0.01-0.08 mg/kg/d	30 children 8-18 y old	EPS, weight gain, gynecomastia	risperidone reported superior to haloperidol only on ABC total score, no subscales reported
Mood stabilizers						
Valproic acid	Hellings *et al.*, 2005[127]	irritability	20 mg/kg/d, average level 75-78	30 subjects 6-20 y old	increased appetite, skin rash	no significant difference for ABC Irritability subscale
	[b]Hollander *et al.*, 2005[128]	repetitive behavior	500-1,500 mg/d	12 children 5-17 y old, 1 adult 40 y old	irritability, aggression	statistically significant decrease in repetitive behavior on CY-BOCS
	Hollander *et al.*, 2010[129]	global irritability	dosed to mean level of 89.8 μg/mL	27 children 5-17 y old	skin rash, irritability	62.5% positive response for irritability on CGI on divalproex vs. 9.09% on placebo
Lamotrigine	[b]Belsito *et al.*, 2001[130]	irritability, social behavior	5 mg/kg/d	28 children 3-11 y old	insomnia, hyperactivity	no significant difference in irritability or social behavior on multiple instruments
Levetiracetam	[b]Wasserman *et al.*, 2006[131]	irritability, global functioning	20-30 mg/kg/d	20 children 5-17 y old	aggression	no significant difference in global functioning or irritability
Norepinephrine reuptake inhibitors						
Atomoxetine HCl	[b]Harfterkamp *et al.*, 2012[132]	hyperactivity, inattention	1.2 mg/kg/d	97 children 6-17 y old	nausea, anorexia, fatigue, early wakening	significant difference in the ADHD-RS for active treatment group; no difference in CGI-I

TABLE 3 Continued

Agent	Study	Target Symptoms	Dose	Demographics	Significant Side Effects	Primary Outcome(s)
	[b]Arnold *et al.*, 2006[133]	hyperactivity, inattention	20-100 mg divided 2×, mean 44 mg/d	16 children 5-15 y old	upper GI symptoms, fatigue, racing heart	57% positive response[a] for parent-rated ABC Hyperactivity subscale vs. 25% on placebo
Serotonin reuptake inhibitors						
Citalopram	King *et al.*, 2009[134]	repetitive behavior	2.5-20 mg/d, mean 16 mg/d	149 children 5-17 y old	hyperactivity, insomnia, inattention, impulsivity, diarrhea, stereotypy	no significant difference in repetitive behavior on CGI-I and CY-BOCS PDD
Fluoxetine	Hollander *et al.*, 2005[135]	repetitive behavior	2.4-20 mg/d, mean 9.9 mg/d	39 children 5-17 y old	none significant	statistically significant decrease in repetitive behavior on CY-BOCS Compulsions scale
Clomipramine	Gordon *et al.*, 1993[136]	stereotypy, repetitive behavior, compulsions	25-250 mg/d, mean 152 mg/d	12 children 6-18 y old	insomnia, constipation, twitching, tremors	decrease in repetitive behavior on CPRS
	Remington *et al.*, 2001[137]	stereotypy, irritability, hyperactivity	100-150 mg/d, mean 128.4 mg/d	31 subjects <20 y old	lethargy, tremors, tachycardia, insomnia, diaphoresis, nausea	no significant difference in stereotypy, irritability, or hyperactivity for clomipramine on ABC
Stimulants						
Methylphenidate	RUPP, 2005[138]	hyperactivity	7.5-50 mg/d divided 3×/d	58 children 5-14 y old	decreased appetite, insomnia, irritability, emotionality	49% positive responders[a] for hyperactivity vs. 15.5% on placebo
	Pearson *et al.*, 2013[139]	hyperactivity, inattention	10-40 mg each morning, methylphenidate extended release	24 children 7-12 y old	decreased appetite, insomnia	significant decrease in hyperactivity and inattention on multiple teacher and parent measurements
	Handen *et al.*, 2000[140]	hyperactivity	0.3-0.6 mg/kg/dose, 2-3×/d	13 children 5-11 y old	social withdrawal, irritability	8 of 13 children with >50% decrease in hyperactivity on Teacher Conners Hyperactivity subscale
	Quintana *et al.*, 1995[141]	hyperactivity	10-20 mg 2×/d	10 children 7-11 y old	irritability, anorexia, insomnia	decrease in ABC Hyperactivity subscale by 8 points over placebo
Miscellaneous						
Amantadine	[b]King *et al.*, 2001[142]	hyperactivity, irritability	2.5-5.0 mg/kg/d	39 children 5-19 y old	insomnia	no statistical difference in parent ABC Hyperactivity or Irritability subscales, statistical improvement in clinician Hyperactivity and Inappropriate Speech subscales

TABLE 3 Continued

Agent	Study	Target Symptoms	Dose	Demographics	Significant Side Effects	Primary Outcome(s)
Cyproheptadine (in combination with haloperidol)	Akhondzadeh *et al.*, 2004[143]	ABC total score, CARS	Titrated up to 0.2 mg/kg/d	40 children 3-11 y old	none significant, trend toward increased appetite	statistically significant difference in ABC total score and CARS diagnostic screening tool, with unknown clinical significance
Donepezil	Chez *et al.*, 2003[144]	"autistic behavior," expressive-receptive communication	1.25-2.5 mg/d	43 children 2-10 y old	diarrhea, stomach cramping, irritability	"autistic behavior" statistically, improved on CARS diagnostic screening tool with unknown clinical significance
Naltrexone	Willemsen-Swinkels *et al.*, 1995[145]	"social behavior," irritability	single 40-mg dose	20 children 3-7 y old	sedation, increased stereotypy	no effect on social behavior; significant decrease on ABC Irritability subscale vs. placebo
	[b]Kolmen *et al.*, 1995[146]	hyperactivity, communication initiation	1 mg/kg/d	13 children 3-8 y old	transient sedation	no significant difference in communication initiation
	[b]Feldman *et al.*, 1999[147]	communication	1 mg/kg/d	24 children, 3-8 y old	transient sedation	no significant difference in multiple communication measurements
	Campbell *et al.*, 1993[148]	CGI, CPRS, discriminant learning, hyperactivity	0.5-1 mg/kg/d	18 children 3-8 y old	increased aggression and stereotypy	no significant difference on CGI or CPRS or discriminant learning; positive trend for hyperactivity
	Campbell *et al.*, 1990[149]	hyperactivity, discriminant learning, self-injurious behavior	0.5-1 mg/kg/d	41 children 3-8 y old	none significant	significantly decreased hyperactivity; no effect on discriminant learning; positive trend for self-injurious behavior
Pentoxifylline (in combination with risperidone)	Akhondzadeh *et al.*, 2010[150]	irritability, hyperactivity, stereotypy, social withdrawal, inappropriate speech	200-600 mg/d	40 children 4-12 y old	sedation, GI effects, increased appetite	significant improvement on ABC Irritability and Social Withdrawal subscales

Note: ABC = Autism Behavior Checklist; ADHD-RS = Attention-Deficit/Hyperactivity Disorder Rating Scale; CY-BOCS = Children's Yale-Brown Obsessive Compulsive Scale; CARS = Childhood Autism Rating Scale; CPRS = Children's Psychiatric Rating Scale; EPS = extrapyramidal side effects; GI = gastrointestinal; PDD = pervasive developmental disorder; RUPP = Research Units on Pediatric Psychopharmacology.

[a]*A positive response in this study was defined as a >25% reduction in the ABC subscale and a Much Improved or Very Much Improved rating on the Clinical Global Impression–Global Improvement (CGH).*

[b]*Study identified as funded by pharmaceutical industry.*

[c]*A positive response in this study was defined as a greater than 25% decrease in ABC (CY-BOCS) compulsions score and a much improved or very much improved rating on the CGH.*

is preliminary evidence for the efficacy of hospital psychiatry units that specialize in the population.[111]

Recommendation 5. Pharmacotherapy may be offered to children with ASD when there is a specific target symptom or comorbid condition [CG].

Pharmacologic interventions may increase the ability of persons with ASD to profit from educational and other interventions and to remain in less restrictive environments through the management of severe and challenging behaviors. Frequent targets for pharmacologic intervention include associated comorbid conditions (e.g., anxiety, depression) and other features, such as aggression, self-injurious behavior, hyperactivity, inattention, compulsive-like behaviors, repetitive or stereotypic behaviors, and sleep disturbances. As with other children and adolescents, various considerations should inform pharmacologic treatment.[112] Risperidone[113[rct]] and aripiprazole[114[rct]] have been approved by the Food and Drug Administration for the treatment of irritability, consisting primarily of physical aggression and severe tantrum behavior, associated with autism. There is a growing body of controlled evidence for pharmacologic intervention,[115] and a summary of randomized controlled trials of medication in children with ASD is included (Table 3).[116-150] Combining medication with parent training is moderately more efficacious than medication alone for decreasing serious behavioral disturbance and modestly more efficacious for adaptive functioning.[151[rct],152[rct]] Individuals with ASD may be nonverbal, so treatment response is often judged by caregiver report and observation of specific behaviors. Although this may help document the effectiveness of the selected medication, one must remember that an overall goal of treatment is to facilitate the child's adjustment and engagement with educational intervention. Several objective rating scales also are available to help monitor treatment response.[153]

TABLE 4 Resources for Parents

ASPEN TM, Inc. (Asperger Syndrome Education Network) (http://www.aspennj.org)	A regional nonprofit organization providing families and those individuals affected with Asperger syndrome and related disorders with information, support, and advocacy.
Autism Society of America (http://www.autism-society.org)	The mission of the Autism Society of America is to promote lifelong access and opportunities for persons within the autism spectrum and their families to be fully included, participating members of their communities through advocacy, public awareness, education, and research related to autism.
Autism Speaks (http://www.autismspeaks.org)	Autism Speaks is an autism science and advocacy organization dedicated to funding research into the causes, prevention, treatments, and a cure for autism; increasing awareness of autism spectrum disorders; and advocating for the needs of individuals with autism and their families.
Division TEACCH (Treatment and Education of Autism and related Communication handicapped Children, University of North Carolina at Chapel Hill) (www.teacch.com)	The TEACCH Web site includes information about their program, educational and communication approaches to teaching individuals with autism, their research and training opportunities, and information and resources on autism.
LDAA (Learning Disabilities Association of America) (http://www.ldanatl.org)	The LDAA site includes information and resources on many learning disabilities, including learning disabilities involving a significant social component, such as autism and Asperger syndrome.
OASIS (Online Asperger Syndrome Information and Support) (http://www.asperger.org)	General information on Asperger syndrome and related disorders, including resources and materials, announcements of major pertinent events and publications, and being the major "intersection" for communication among parents, clinicians, educators, and individuals with social disabilities.
Yale Child Study Center (www.autism.fm)	Information on autism, Asperger syndrome, and related disorders, lists of resources organized by state, and parent support organizations and advocacy agencies.

Recommendation 6. The clinician should maintain an active role in long-term treatment planning and family support and support of the individual [CG].

Children's and families' need for help and support will change over time. The clinician should develop a long-term collaboration with the family and realize that service utilization may be sporadic. For very young children, issues of diagnosis and identification of treatment programs often will be most important. For school-age children, psychopharmacologic and behavioral issues typically become more prominent. For adolescents, vocational and prevocational training and thoughtful planning for independence/self-sufficiency is important. As part of this long-term engagement, parents and siblings of children with ASD will need support (Table 4). Although raising a child with autism presents major challenges, rates of parental separation and divorce are not higher among parents of children with ASD than those with non-ASD children.[154]

Recommendation 7. Clinicians should specifically inquire about the use of alternative/complementary treatments and be prepared to discuss their risk and potential benefits [CS].

Although most alternative or complementary treatment approaches have very limited empirical support for their use in children with ASD, they are commonly pursued by families.[155] It is important that the clinician be able to discuss these treatments with parents, recognizing the motivation for parents to seek all possible treatments. In most instances, these treatments have little or no proved benefit but also have little risk.[7] In a few instances, the treatment has been repeatedly shown not to work (e.g., intravenous infusion of secretin[156] and oral vitamin B6 and magnesium[157[rct]]), or randomized controlled evidence does not support its use (e.g., the gluten-free, casein-free diet,[158] ω-3 fatty acids,[159] and oral human immunoglobulin).[160[rct]] Some treatments have greater potential risk to the child directly (e.g., mortality and morbidity associated with chelation[161[cs]]) or from side effects owing to contaminants in "natural" compounds or indirectly (e.g., by diverting financial or psychosocial resources). For a detailed review of alternative treatments, see Jacobson *et al.*[162] and Levy and Hyman.[163] Although more controlled studies of these treatments are needed, it is important that the family be able to voice their questions to health care providers. Families may be guided to the growing body of work on evidence-based treatments in autism.[164]

PARAMETER LIMITATIONS

AACAP Practice Parameters are developed to assist clinicians in psychiatric decision making. These Parameters are not intended to define the sole standard of care. As such, the Parameters should not be deemed inclusive of all proper methods of care or exclusive of other methods of care directed at obtaining the desired results. The ultimate judgment regarding the care of a particular patient must be made by the clinician in light of all of the circumstances presented by the patient and his or her family, the diagnostic and treatment options available, and available resources. &

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The AACAP Practice Parameters are developed by the AACAP CQI in accordance with American Medical Association policy. Parameter development is an iterative process between the primary author(s), the CQI, topic experts, and representatives from multiple constituent groups, including the AACAP membership, relevant AACAP committees, the AACAP Assembly of Regional Organizations, and the AACAP Council. Details of the Parameter development process can be accessed on the AACAP Web site. Responsibility for Parameter content and review rests with the author(s), the CQI, the CQI Consensus Group, and the AACAP Council.

The AACAP develops patient-oriented and clinician-oriented Practice Parameters. Patient-oriented Parameters provide recommendations to guide clinicians toward best assessment and treatment practices. Recommendations are based on the critical appraisal of empirical evidence (when available) and clinical consensus (when not) and are graded according to the strength of the empirical and clinical support. Clinician-oriented Parameters provide clinicians with the information (stated as principles) needed to develop practice-based skills. Although empirical evidence may be available to support certain principles, principles are based primarily on clinical consensus. This Parameter is a patient-oriented Parameter.

The primary intended audience for the AACAP Practice Parameters is child and adolescent psychiatrists; however, the information contained therein also may be useful for other mental health clinicians.

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