21

Autism Spectrum Disorders

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Autism spectrum disorder (ASD) is a chronic and highly debilitating neurodevelopmental disorder. Like all neurodevelopmental disorders (e.g., intellectual disabilities, communication disorders), ASD begins early in life and is characterized by deficits that impair the individual's functioning across multiple life domains (e.g., academic and social). ASD is usually identified and diagnosed during childhood, and most affected individuals retain the diagnosis through adulthood (Cederlund, Hagberg, Billstedt, Gillberg, & Gillberg, 2008). The major diagnostic features of ASD are *social communication/interaction deficits* (i.e., impaired reciprocity, absent or delayed nonverbal interpersonal communication, and impoverished social relationships) and *presence of restricted, repetitive behaviors or interests* (e.g., insistence on keeping rigid routines, hyper/hyporeactivity to sensory stimuli) See McPartland and Dawson (2014) for a review of the history of the diagnosis and changes to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM; American Psychiatric Association, 2013) and the International Classification of Diseases (ICD; World Health Organization, 1992).

Diagnosis of Autism Spectrum Disorder

ASD is more common than childhood diabetes—affecting approximately one in 88 children (Centers for Disease Control and Prevention, 2012). Some of the earliest markers of the disorder include behavioral regression or loss of language skills; lack of cooing, babbling, or other socially meaningful gestures by around 12 months of age; no single-word speech by 16 months; and a lack of two-word spontaneous phrase speech by 24 months of age (Johnson & Myers, 2007). Screening for possible ASD usually involves behavioral questionnaires completed by the caregiver, such as the Social Communication Questionnaire (Rutter, Bailey, & Lord, 2005) or Social Responsiveness Scale-2 (Constantino & Gruber, 2012). A full diagnostic evaluation for ASD should include measures specifically designed and validated for ASD assessment, such as the Autism Diagnostic Observation Schedule (Lord et al., 2012), as well as cognitive testing and assessment of adaptive behavior. Ideally, a full diagnostic assessment will be conducted (e.g., with semi-structured clinical interview, teacher reports of behavior) to adequately consider the possibility of other disorders that may be comorbid or that might better explain the presenting concerns, rather than only an ASD-specific evaluation. It is also recommended that other professionals (e.g., speech therapist, genetics specialist) be involved to the extent possible, as ASD often co-occurs with other conditions, such as fragile X.

Challenges Ahead

An in-depth account of the history of ASD is beyond the scope of this chapter. For detailed commentaries on the history of ASD and debate about nosology, the reader is referred to Davis, White, and Ollendick (2014). What we now label "ASD" has been recognized, in assorted forms and by various terms, since at least 1943, with Kanner's (1943) account of 11 children who demonstrated idiosyncratic language, impaired socialization, and repetitive behaviors. The label "infantile autism" first became part of our clinical nosology 40 years later with the publication of the third edition of the DSM (DSM-III; American Psychiatric Association, 1980) and, since this initial introduction, the scope of the diagnosis has broadened and

its nuances have been elaborated—largely as a function of ever-growing research and public interest, as well as increased clinical recognition and diagnosis.

The most fundamental, and hotly debated, change in the diagnostic criteria for autism from the fourth edition of the DSM (DSM-IV; American Psychiatric Association, 1994a) to DSM-5 was the elimination of all sub-diagnoses (i.e., pervasive developmental disorder–not otherwise specified; Asperger's disorder; childhood disintegrative disorder; autistic disorder) and development of one umbrella diagnostic label of "ASD." The diagnosis of ASD thus replaced the category of pervasive developmental disorders, under which autistic disorder, pervasive developmental disorder–not otherwise specified, and Asperger's disorder fell. Second, the criteria for the diagnosis were restructured. Since the 1970s (e.g., Rutter, 1978), ASD comprises a triad of impairments in the domains of social impairment, communicative deficits, and restricted interests and repetitive behaviors. In DSM-5, these three domains are reduced into two broad domains: social communication features and restricted interests features. Language delay is no longer diagnostic; it is identified as an *associated* condition. Third, for diagnosis, an individual must present persistent deficits in every subdomain of the first domain (social communication impairment). In DSM-IV, there were no broad criteria in which each sub-domain had to be present, making the predecessor a more polythetic approach, which has undoubtedly contributed to some of the vast clinical heterogeneity seen in ASD. Although there are other changes in DSM-5, such as greater recognition of the possibility that problems might not be apparent until later childhood or even adulthood, these three represent the most substantive modifications.

The clinical and scientific consequences of these changes are, as yet, unknown. Clinically, individuals with diagnoses now subsumed under ASD (e.g., Asperger's disorder) who were evaluated using DSM-IV criteria, are to be 'grandfathered' in, meaning that the DSM-5 diagnosis of ASD applies without need for reevaluation. Newly diagnosed individuals who would have previously been diagnosed with Asperger's disorder now would likely be diagnosed with ASD. Nonetheless, there are likely to be some challenges ahead with respect to resource allocation and services. The composition of study samples over time will likely differ considerably, assuming inclusion criteria for new studies are based on current nosology. This will affect our ability to make comparisons over time across the samples in studies. This issue, of course, has been faced in the past with DSM revisions. However, the impact might be greater and harder to navigate with ASD because of the scope of the diagnostic changes seen in DSM-5 and the quick pace with which new research is being conducted and produced now and over the past 5 years in this field (e.g., Reichow & Volkmar, 2011). There is much greater attention given to possible comorbidities with ASD (e.g., social anxiety disorder, attention-deficit/hyperactivity disorder), which should ease differential diagnostic decisions when both disorders truly do present in the same person. There is also more consideration for the severity of the disorder related to daily adaptive behavior. This should yield more clinically informative data than the "sub-diagnosis" approach that may have obfuscated diagnosis with adaptive behavior (e.g., high-functioning being synonymous with either pervasive developmental disorder–not otherwise specified or Asperger's disorder). The more unified the criteria, the more likely the diagnosis will yield more homogeneous samples and will thus increase our ability to identify disease biomarkers and establish efficacious treatments with discernable underlying change mechanisms. In summary, although there are several weighty concerns (e.g., under-identification and poor sensitivity), the changes seen in DSM-5 are empirically based and are consistent with changes seen for other psychiatric disorders—moving toward a more consistent, individualized, and dimensional approach to diagnostic categorization.

Etiology

Historically, autism was first thought to be the result of highly educated parents, especially mothers, who lacked warmth in their parenting. These parents were described as "refrigerator mothers" (Kanner, 1954; Rapin, 2011). Advances in the field, including development in fields such as psychopharmacology and neuroscience, led researchers to examine autism with a biological lens (Rapin, 2011). Owing to increased prevalence and awareness of ASD, research on its etiology has advanced considerably over the previous 40 years. Among the initial forays of genetic research in ASD in the 1970s and 1980s was research on associated genetic conditions, such as fragile X syndrome, epilepsy, and tuberous sclerosis (Freitag, 2007; Rapin, 2011), as well as twin studies and family heritability studies (Rapin, 2011). As new genetic, neuroscience, and epigenetic and environmental methods emerged, additional information concerning ASD etiology has continued to accumulate.

Biogenetic Factors Research concerning familial aggregation of ASD and its symptomatology has provided indicators of the nature of its etiology. Twin studies of heritability in ASD, in which monozygotic and dizygotic twins are compared, have found that up to 90% of ASD has a genetic cause (see Rutter, 2005, for a review). Owing to the likely genetic underpinnings of ASD, much research has used a sample of younger siblings of children diagnosed with ASD to investigate the developmental trajectories in ASD. This research on siblings of children diagnosed with ASD has shown that approximately 15–20% of siblings of a child with ASD are also diagnosed with the disorder, and an additional segment of siblings not diagnosed with ASD at age 3 years nonetheless present with delays in motor and language functioning (Messinger et al., 2013; Rogers, 2009). Research on first-degree relatives of individuals with ASD, mostly with adult samples, has also shed light on the existence

of subclinical traits of ASD in what has been termed the broader autism phenotype (Klusek, Losh, & Martin, 2014; Sasson, Lam, Parlier, Daniels, & Piven, 2013). Social difficulties, difficulties with sociocommunicative interactions, restricted and repetitive interests and behaviors, and personality traits such as rigidity comprise the broader autism phenotype, a term used to describe genetic relatives of individuals with ASD who may have characteristics associated with ASD but who nonetheless do not have ASD diagnoses. First-degree and extended relatives of individuals with ASD have been found to score higher on measures of broader autism phenotype symptomatology, lending evidence to the genetic basis of ASD (Pickles et al., 2000; Piven & Palmer, 1999; Sasson et al., 2013). In addition to research on family members, broader autism phenotype characteristics also present in subclinical levels among the general population (Hurley, Losh, Parlier, Reznick, & Piven, 2007).

Overall, research on the genetic basis of ASD has highlighted the heterogeneous nature of the disorder. Only a minority of cases of ASD are due to a single gene mutation; the majority of cases appear to be due to multiple genetic mutations, including duplications and deletions, which can be inherited from parents' genetic information or due to mutations at the individual's conception (Geschwind, 2008). Thus far, studies have had divergent findings in genetic linkage studies, where individual genes have only accounted for up to 1–2% of cases of ASD (Geschwind, 2008).

Brain-Based Factors Much of the previous research on the etiology of ASD has focused upon neurological methodologies, such as functional and functional connectivity magnetic resonance imaging (fMRI and fcMRI; McFadden, Minshew, & Scherf, 2011). Overall, imaging data suggest that ASD affects no single area of the brain, but instead is pervasive in altering functioning. Structural differences are seen as well; while there is no uniform pattern of structural difference, a majority of individuals with ASD show abnormally increased gray and white matter growth in the first several years of life, which is then followed by a dampened growth trajectory that results in average brain volume, gray matter, in adolescence and adulthood (McFadden et al., 2011). fcMRI studies, in particular, have demonstrated that individuals with ASD have less white matter connectivity, especially in the frontal regions and connecting across the brain, and greater connectivity within the parietal, visual, and temporal cortices (Just, Cherkassky, Keller, & Minshew, 2004). This pattern of under- and overconnectivity may serve to explain some commonly reported of ASD phenotypic expression—specifically superior visual or auditory processing, alongside impairment of executive functioning and other higher-order processes (McFadden et al., 2011; Philip et al., 2012). Many of the domains of functioning most impaired in ASD, such as social cognition, theory of mind, pragmatic and idiomatic language, and conceptual thinking, are those which require coordination of several distinct neural regions; conversely, intra-regional skills such as simple motor movements, formal and rule-based language, and memory are sometimes areas of strength for people with ASD (McFadden et al., 2011). A review of studies comparing individuals with ASD and neurotypical individuals found differences in neural areas associated with motor processing, visual processing, executive functioning, both simple and complex social processing tasks, cortical connectivity, and a lack of preference for social stimuli, as indicated in under-activation of the fusiform face area during tasks which require participants to attend to faces and eyes (Philip et al., 2012; Russo et al., 2007; Vissers, Cohen, & Geurts, 2012).

Psychosocial/Environmental Factors Although the current consensus is that ASD is predominantly genetic in origin, increasing attention has been paid to the identification of potential environmental factors that may contribute to ASD, largely due to the increased prevalence and awareness of ASD among the general population.

Another growing area of research in the environmental risk factors for ASD is epigenetics, the study of how gene expression is altered. Many factors, including parental age, infection during pregnancy, and exposure to environmental toxins during pregnancy or postnatally, have been examined in large-scale epidemiology studies as factors that could potentiate expression of underlying genetic vulnerability. Overall, studies suggest that no single environmental factor is strongly associated with later ASD diagnosis, and instead each environmental agent may contribute synergistically to existing genetic vulnerabilities to result in ASD (Hertz-Picciotto, 2011; Persico & Bourgeron, 2006).

The most well-known, and hotly debated, environmental agent linked to ASD has been vaccines, in particular the preservative used in the measles, mumps and rubella vaccine, thimerosol. Thimersol, which contains mercury, was studied as a potential environmental factor associated with the increase in ASD diagnoses (Gerber & Offit, 2009; Stehr-Green, Tull, Stellfeld, Mortenson, & Simpson, 2003). However, research on a global scale, including regions where usage of preservatives in vaccines differs, has shown that vaccines and thimerosol in particular do not have an effect on ASD prevalence (Stehr-Green et al., 2003). Nevertheless, some parents have chosen to forego vaccinating their children because of continuing concerns about the association of vaccines to ASD, which has contributed to the reemergence of preventable diseases (Poland, 2011).

Culture and Gender There is some evidence for the effects of gender and cultural factors on identification of ASD. For example, age at diagnosis has been found to vary on several culturally relevant factors, including socioeconomic

status and geographic location. Worldwide, while recognition of ASD has continued to increase, there remains scant large-scale data concerning prevalence or research on ASD in many areas outside of North America, Western Europe, and some Asian nations, and the similarity of core symptoms and onset of ASD worldwide remains an assumption (Grinker, Yeargin-Allsopp, & Boyle, 2011).

Within the nations where ASD is more intensively studied, cultural factors are still a significant indicator of when an individual is diagnosed with ASD. Initially, ASD was thought to be limited to children of highly educated parents and some researchers posit that ASD is more common among children of engineers and scientists (Baron-Cohen, 2004). Socioeconomic status does appear to effect identification (specifically *when* an individual is initially identified and diagnosed), though not actual occurrence of ASD. A study of Pennsylvania families in which a child was diagnosed with ASD found that the average age at diagnosis was higher for children with fewer language difficulties and less severe symptom presentations, but that children located in rural areas and children of families in poverty or near-poverty levels were significantly older (0.4 and 0.9 years, respectively) when they received their diagnosis (Mandell, Novak, & Zubritsky, 2005). Racial and ethnic identity have been found to be associated with age of initial diagnosis, with individuals of ethnic minorities receiving their diagnosis at least 1 year later than White children, although this finding has not been demonstrated consistently in the literature (Mandell, Listerud, Levy, & Pinto-Martin, 2002; Mandell et al., 2005). Factors such as geographic location and socioeconomic status may interact with race and ethnicity (Mandell et al., 2002). Given the importance of early identification and treatment in achieving optimal outcomes among individuals with ASD, these cultural differences in access to services and obtaining an initial diagnosis can have considerable consequences.

Since Kanner's (1943) first observations of children diagnosed with autism, ASD has been found to occur more frequently in males than females. The ratio of males to females diagnosed has ranged from 3:1 to 7:1, with some studies finding that fewer females are diagnosed, especially at the higher-functioning edge of the spectrum. Females with ASD are also more likely to be diagnosed later in life than males (see Kreiser & White, 2013, for a review). However, researchers have also argued that this discrepancy in diagnosis across gender may reflect methodological biases in our assessment practices and lack of sensitivity in the diagnostic criteria to gender-based differences in manifest expression, in addition to actual sex-based etiological differences (Kreiser & White, 2013; van Wijngaarden-Cremers et al., 2014). These arguments center on a different presentation of ASD in females, particularly those who are higher functioning. This "female prototype" of ASD is characterized by better mask sociocommunicative deficits, alongside fewer stereotyped behaviors and routines and restricted interests that appear less unusual than is often seen in males with ASD; ambivalent social motivation; and increased internalizing (e.g., poor emotion regulation) symptoms (Kreiser & White, 2013; Trubanova, Donlon, Kreiser, Ollendick, & White, 2014).

Treatment of ASD

We must consider evidence-based treatment for ASD a bit differently than we might normally for most other mental health disorders, owing to the pervasiveness of impairment and the multi-faceted impairments and symptoms for which a person with ASD seeks (or, more often, is referred for) treatment. More to the point, treatment is usually sought for problems that are co-occurring (e.g., aggression) rather than core ASD symptoms (Joshi et al., 2010). Given the chronicity of the disorder, interventions are not intended to cure ASD or treat to remission. Yet another factor that complicates identification of evidence-based treatments for ASD is the tremendous phenotypic heterogeneity present among people diagnosed with ASD (e.g., in verbal and cognitive ability, in severity of core and secondary symptoms). As such, identification of evidence-based treatments for ASD is a complicated and nuanced endeavor. Herein, we describe the most commonly used pharmacological and psychosocial interventions, and also summarize interventions that are contraindicated.

Biological/Pharmacological Interventions It is estimated that over half of children and adults diagnosed with ASD are treated pharmacologically (Mandell et al., 2008). Once medicated, young people with ASD tend to remain on psychotropic medication during adolescence and into adulthood (Esbensen, Greenberg, Seltzer, & Aman, 2009). Additionally, pharmacological intervention for ASD tends to target specific secondary problems (e.g., anxiety, irritability) rather than ASD core symptoms such as social disability (Malone, Maislin, Choudhury, Gifford, & Delaney, 2002).

Most pharmacological treatment for people with ASD is done off-label, as the only medications approved by the U.S. Food and Drug Administration for individuals with ASD are risperidone and aripiprazole, both of which are indicated for reduction of irritability (including extreme aggression). The most commonly prescribed drug classes are neuroleptics (atypical antipsychotics), antidepressants, and stimulants (Mandell et al., 2008). Repetitive and stereotyped behaviors are often treated with serotonin-reuptake inhibitors, although careful monitoring of adverse effects is encouraged (Vahabzadeh, Buxton, McDougle, & Stigler, 2013). There is general agreement that severe irritability (e.g., temper tantrums, physical aggression) can be treated effectively pharmacologically, although not without considerable adverse effects such as increased appetite and weight gain, tremors, and fatigue (e.g., Owen et al., 2009; Scahill, Koenig, Carroll, & Pachler, 2007). Supplementation of pharmacological treatment with a

psychosocial approach (e.g., parent training) may be most effective (Aman et al., 2009), although there has been little research on the use of such integrated approaches. Most of the clinical research in this field has been done with children and adolescents and, as a result, we know fairly little about pharmacological treatment for adults with ASD. There is active research on other compounds, and it is likely that the next decade will see considerable improvements with respect to our knowledge of pharmacological interventions. For example, recent research on the use of oxytocin for social impairments in people with ASD has produced very promising findings (Sikich et al., 2013; Wudarczyk, Earp, Guastella, & Savulescu, 2013).

Psychosocial Interventions There are many thorough, informative reviews on evidence-based treatments in this area, of which the most recently published (Wong et al., 2013) used five databases and generated over 29,000 articles published in just a 21-year period (1990–2011). In the most recent report from the National Professional Development Center on ASD, 27 evidence-based practices (defined as those to be shown effective through high-quality scientific research) were identified (Wong et al., 2013). The National Autism Center (2009), likewise, has developed empirically informed guidelines for treatment of ASD in the schools, dividing approaches into those that are "emerging" (22 treatments identified as such) and those that are "established" (11 treatments identified as such; e.g., antecedent packages, modeling). These large-scale reviews have applied stringent criteria by which to rate the quality of the reviewed treatment research, and are extremely useful resources. The *Encyclopedia of Autism Spectrum Disorders* (Volkmar, 2013) and the fourth edition of the *Handbook of Autism and Pervasive Developmental Disorders* (Volkmar, Paul, Rogers, & Pelphrey, 2014) are also valuable resources, offering summaries of empirically based interventions, and critical analyses of the extant research. In this chapter, we do not cover all of the comprehensive and focused psychosocial treatments for ASD. Rather, we concentrate on approaches that can be used to target a range of clinical foci: behavioral/cognitive-behavioral interventions, skills training interventions, and educational interventions. Several intervention approaches that have been well-researched and supported for treatment of core deficits in ASD, such as augmentative communication systems (e.g., Picture Exchange Communication System; Sulzer-Azaroff, Hoffman, Horton, Bondy, & Frost, 2009) and comprehensive educational and treatment programs (e.g., Treatment and Education of Autistic and related Communication handicapped Children; Panerai, Ferrante, & Zingale, 2002) are not covered.

Behavioral/Cognitive-Behavioral Interventions Of the approaches used with people who have ASD, applied behavior analysis is the most widely used and empirically supported treatment (e.g., Lovaas, 2003). Common targets of applied behavior analysis include teaching new skills such as verbal speech, mathematics, and daily living skills, and reducing undesirable or dangerous behaviors such as self-injurious behaviors, tantrums, and disruptive behaviors such as leaving the classroom or screaming when instruction is taking place. Intensive applied behavior analysis (i.e., up to 40 hours per week) is usually recommended for children under the age of 4 years who are diagnosed with ASD (e.g., Eikeseth, Smith, Jahr, & Eldevik, 2007; Harris & Handleman, 2000; National Research Council, 2001), and there is evidence that the effect of applied behavior analysis is strongest when dosage is high and the client is very young (Smith, 2010). Applied behavior analysis is based on the principles of operant condition, or stimulus-response learning (Skinner, 1938). The particular technique of discrete trial training, often used with young children with ASD, relies on structured prompting and reinforcing of specific, targeted skills and behaviors. Applied behavior analysis is a highly structured, data-based approach. Individual (one-on-one) instruction is the mainstay and, as such, the approach is costly.

Behavior therapy and cognitive-behavioral therapy (CBT) are often used to treat secondary (non-core) problems in people with ASD as well (see Scarpa, White, & Attwood, 2013). Approximately 40% of higher-functioning people with ASD have co-occurring problems with anxiety (van Steensel, Bogels, & Perrin, 2011). CBT is a supported treatment for anxiety in children and adolescents with ASD (e.g., Chalfant, Rapee, & Carroll, 2007; White et al., 2013). Although depression is also common in ASD, estimated to effect up to 40% of children and adolescents with ASD (Strang et al., 2012), there is insufficient research to know if CBT is effective for treatment of depression in ASD. Many children with ASD are referred because of problems with aggressive behavior (Johnson & Myers, 2007). For such cases, a functional assessment is first used to identify the factors that maintain or exacerbate the behaviors (Matson, 2009), followed by behaviorally based interventions such as parent training (e.g., teaching the parent to consistently deliver consequences and use visual schedules to ease transitions) and environmental modifications (e.g., reducing noise level to decrease baseline arousal) (e.g., Johnson & Myers, 2007; Research Units on Pediatric Psychopharmacology, 2007).

In implementing a CBT treatment program with a client with ASD, modifications in both content and approach are often made to improve response. Although it has not been scientifically established which (if any) modifications are necessary for CBT to be helpful for people with ASD, we review some of the most common modifications. Behavioral experiments and practices (e.g., practice, self-talk) tend to be emphasized over the more cognitively oriented tasks (e.g., emotion recognition, thought challenging) of CBT (Lang, Regester, Lauderdale, Ashbaugh, & Haring, 2010), although there is evidence that children

with ASD do not uniformly show deficiency in cognitive skills likely to influence response to CBT intervention. Lickel and colleagues (2012) showed that children with ASD performed similar to typically developing peers on tasks of thought/feeling/behavior discrimination and cognitive-affective inference, but significantly poorer on tasks of emotion recognition. With young clients, parents are often more involved in treatment than what might be typical of individual CBT for a child without ASD. Given the chronicity of ASD and the pervasiveness of impairments, it can be helpful to educate the family about the disorder and the treatment, encourage familial support in the intervention, and have them involved in outside-session practices (e.g., Sofronoff, Attwood, & Hinton, 2005; White et al., 2010). Consistent, directive feedback as opposed to indirect or subtle feedback, and intensive practice (e.g., of new skills) are often emphasized as well. Finally, incorporation of teaching aides that involve the client's interests or address some aspect of his learning style (e.g., using visual supports with a client who processes auditory information slowly) can help maintain interest and motivation (White et al., 2010).

Skills Training Interventions The hallmark characteristic of ASD is pervasive social disability (American Psychiatric Association, 2013). Interventions are often aimed at improving general social competence in children, adolescents, and adults with ASD. Although interventions to improve social functioning in youth with ASD have generally demonstrated promising results (Reichow & Volkmar, 2011; Wang & Spillane, 2009), the supporting evidence has not been consistently strong, and common methodological limitations (e.g., primarily parent report measures and use of wait-list comparison conditions) have hampered comparative evaluation across programs. There are, however, many intervention models to target social disability in ASD, with an emerging base of empirical support including applied behavior analysis-based behavior modification, peer as interventionist and tutor models, social stories, computer-based training games, and video-modeling, many of which we discuss below. There are also resources available on how to design individualized programming to address the social problems of children with ASD (e.g., White, 2011).

Social skills training in a group format is common and, although research indicates such an approach is feasible and often helpful (effective), there has been little research comparing active (i.e., not just a wait-list control) programs and very few controlled trials (White, Keonig, & Scahill, 2007). There are many commercially available curricula developed specifically for young people with ASD (e.g., Baker, 2003; Bellini, 2008; Laugeson & Frankel, 2010; McAfee, 2002). Regardless of the approach or curriculum taken, it is generally suggested that parents (or other family members) be involved and that practice be frequent across contexts, to help promote generalization of learned skills (White et al., 2007).

Video-based and computer-delivered interventions to improve social functioning have also been evaluated empirically (e.g., Hopkins et al., 2011; Tanaka et al., 2010). Such technology is appealing as an intervention modality for many reasons, including capacity to increase "dose" of intervention without necessarily adding cost, allowing tighter controls over the intervention parameters in a "safe" environment, and an-oft reported affinity for technology and computers among many individuals affected by ASD. Although this field is still early in its development, there is considerable promise of feasibility (Wainer & Ingersoll, 2011). We are sure to see much more research on the efficacy of such programs in the next few years.

Educational Interventions Educational interventions aim to improve learning, and decrease behaviors that interfere with learning and functioning, within the typical school (general education) setting. A full review of the scope of educational interventions is beyond the scope of this chapter. We do not, for instance, review available comprehensive special education programs for learners with ASD (e.g., TEACCH; Mesibov, Shea, & Schopler, 2005; Learning Experiences and Alternative Program for Preschoolers and their Parents; Strain & Hoyson, 2000). Instead, we focus on more targeted intervention approaches typically implemented within the regular education classroom.

When a behavior is identified as detrimental to the student's progress, the first step is usually a functional assessment of the behavior to identify the goal(s) of the behavior, as well as precipitants (antecedents) and maintaining processes (consequences; Gresham, Beebe-Frankenberger, & MacMillan, 1999; Gresham et al., 2004). The functional assessment begins with data gathered from teachers and support staff (e.g., via interview) and careful, direct observation of the learner. Once hypotheses are formulated to try to explain the occurrence of the behavior, target variables (e.g., amount of stimulation in environment, teacher's choice of response to the behavior) are systematically altered while the behavior is closely tracked. Through this systematic, experimental process one can reduce (or increase, if that is the intent) the behavior. Functional assessments, within an inclusive school setting, can be useful for a range of difficulties, such as aggression, elopement, and work refusal (e.g., Williams, Johnson, & Sukhodolsky, 2012).

Antecedent-based approaches are implemented prior to the occurrence of a target problem behavior (National Autism Center, 2009). Such approaches, sometimes referred to as stimulus control or environmental modification, are often sufficient to address the behavior. Within this broad class of intervention approaches we include use of visual cues and prompts, chaining procedures (teaching skills in small steps hierarchically), and errorless learning (prompting the behavior, followed by reinforcement, before error or non-response can occur).

Consequence-based strategies modify what happens after the behavior. For example, if it is determined that escape from task demands is what is maintaining and reinforcing a target behavior (e.g., screaming during class), the student may be sent to a different room with his or her work to complete, rather than being sent out of the classroom empty-handed. Academically, embedded instruction is often used, in which strategies to address the child's identified skill deficiencies are integrated into the general education routine (e.g., during transitions between activities; see, e.g., Johnson, McDonnell, Holzwarth, & Hunter, 2004). Peer-mediated approaches, such as peer tutoring, have been found to be effective in improving the academic skills of students with ASD (e.g., Kamps, Barbetta, Leonard, & Delquadri, 1994). Peer support interventions can also more generally promote academic engagement of students with ASD (McCurdy & Cole, 2014; for more comprehensive summaries of educational interventions for students with ASD, see Odom, Boyd, Hall, & Hume, 2014; Martins, Harris, & Handleman, 2014).

Interventions That Are Contraindicated by Research The myriad controversial intervention approaches for ASD can be grouped into two broad categories: Those that lack sufficient scientific validation or have weak/equivocal support; and those that are not supported (i.e., contraindicated). Auditory integration therapy, vitamin supplements, and special diets fall in the former camp, in that they lack empirical support to suggest efficacy (Sinha, Silove, Wheeler, & Williams, 2005; Smith, 2008). In other words, such approaches may be effective for some individuals, but either the research to date has been highly equivocal in its support or there has been insufficient research to make a determination on impact. In addition to the limited empirical support for efficacy, the considerable cost of many of these interventions should be considered and discussed with clients and their families as a potential risk during the informed consent and education prior to commencing treatment, given that the resources could be used for more effective or efficient treatments. Facilitated communication, where another individual physically assists someone without verbal communication in using a keyboard or assistive technology device to communicate, and secretin therapy fall in the latter camp, among those treatments that have been disproven, pose risk and are, as such, contraindicated (American Psychological Association, 1994; Jacobson, Foxx, & Mulick, 2005). Secretin, a hormone that aids with digestion, is probably one of the best-studied agents in ASD treatment research, but it not effective in treating ASD (Smith, 2008; Williams, Wray, & Wheeler, 2012).

The field of ASD treatment research is developing at a rapid pace, but there is still much to learn about for whom supported treatments work best (e.g., moderators and individual differences) and through what mechanisms interventions exert influence. Clinicians are encouraged to keep an open mind because many families do considerable online research and want to learn about treatment alternatives, but they should also be cautious consumers. When an evidence-based treatment is not viable or not available, or when the client has not responded to the primary evidence-based treatment or is otherwise not able to participate, fully informed consent is critical at the outset of the therapeutic relationship and the clinician must balance costs and risks of the approach with careful data-based monitoring of treatment outcomes.

Summary

Given the complexities of the presentation of ASD and its treatment, a case example may be instructional. "Tyler", a 9-year-old male, is a third grade student. He presented as a bright boy who reportedly enjoys reading and playing video games. His parents were concerned with his performance in school. He reports that his classes are "easy," but his grades were lower than would be expected based on assessed intellectual ability. He was referred to a community-based practitioner for an evaluation for attention-deficit hyperactivity disorder.

During the clinical interview, Tyler's mother reported that he does not have any close friends, and that he spends most of his time reading about military history, his favorite topic, or playing video games with his younger brother. His mother reported that his knowledge of the American Civil War is immense, and that he has enjoyed visiting battle sites and discussing details of the battle with anyone who shows interest. Throughout the assessment, Tyler displayed compliant behavior, but little eye contact with the examiner. Additionally, he appeared to have difficulty engaging in conversation with the examiner. Results from cognitive and school-based achievement measures indicate that Tyler's functioning is above average in most domains, although his processing speed score on the Wechsler Intelligence Scale for Children IV was significantly lower than his other scores. His academic achievement scores were even with his cognitive scores.

As a result of these findings and clinical observations of social difficulties, the examiner administered the Autism Diagnostic Observation Schedule-2, Module 3 (Lord et al., 2012) to Tyler. This is a semi-structured clinical observation task; in Module 3, used with verbally fluent children, the examiner asks the child questions and has the child complete tasks. Tyler's obtained scores fell above the autism spectrum cutoffs. The Autism Diagnostic Interview-Revised (Lord, Rutter, & Le Couteur, 1994) was administered to Tyler's mother, to assess his current behavior and developmental history, which indicated continuing difficulties in social, communication, and restricted interest and repetitive behavior domains since early childhood. Based upon these findings, Tyler was diagnosed with autism spectrum disorder without accompanying intellectual and language impairment.

Following his diagnosis, intervention concerning his school performance and social skills was recommended.

Treatment included individual therapy centering on developing social skills and managing anxiety concerning interacting with his peers. Specifically, the therapist helped Tyler identify classmates who had some of the same interests in military history and video games, and didactic instruction and practice on how to initiate and participate in reciprocal conversations. In school, Tyler qualified for access to extra supports in the form of one class period of direct tutoring each day, which allowed for instruction on time management and organization of his homework assignments so that homework completion was increased.

This case example highlights some of the pertinent issues commonly seen among individuals with ASD. Tyler's symptomatological presentation is one of a high-functioning individual on the spectrum; while his cognitive and academic achievement abilities were unimpaired with respect to global domains of functioning, he demonstrated difficulties in maintaining social reciprocity in conversation, including a lack of effective eye contact, establishing and maintaining typical friendships with peers, and a restricted interest in military history. Tyler's initial referral for attention-deficit hyperactivity disorder and older age at initial diagnosis are also common among individuals who are higher functioning on the spectrum, as their early developmental delays, if present at all, can be slight (such as a slight delay in speech or motor skills) and thus can go unnoticed until the social milieu exceeds their abilities. Comorbid difficulties in attention and anxiety when interacting with peers are also commonly seen among children and adolescents with ASD, and are imperative to evaluate when designing effective interventions.

The pace of research being produced related to etiology, assessment, and treatment of ASD is strong. As such, we can anticipate tremendous advancements made in our understanding of the disorder as well as clinical care and policy. ASD is a heterogeneous neurodevelopmental disorder. Although genetic in origin, there is no single identifying pathway to diagnosis. Treatment research to date has largely focused on educational, comprehensive programming for very young children, but recently more attention has been given to treatment options for both core and secondary problems in adolescents and adults with ASD. This is critical, as ASD is both pervasive and chronic.

Resources

Books

Amaral, D. G., Dawson, G., & Geschwind, D. G. (2011). *Autism spectrum disorders*. New York, NY: Oxford University Press.

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White, S. W. (2011). Practitioner's guide to social skills training in children with asperger's syndrome and high functioning autism. New York, NY: Guilford.

Online Resources

- National Institute of Mental Health. Autism spectrum disorder: http://www.nimh.nih.gov/health/topics/autism-spectrum-disorders-pervasive-developmental-disorders/index.shtml?utm_source=rss_readers&utm_medium=rss&utm_campaign=rss_full (accessed May 7, 2015).
- Centers for Disease Control and Prevention. Autism spectrum disorder: http://www.cdc.gov/ncbddd/autism/research.html (accessed May 7, 2015).
- Organization for Autism Research: http://www.researchautism.org (accessed May 7, 2015).
- Autism Speaks: http://www autismspeaks.org (accessed May 7, 2015).
- Global and Regional Asperger Syndrome Partnership (GRASP): http://grasp.org (accessed May 7, 2015).

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