CHAPTER 1

The Evolution of Autism as a Diagnostic Concept

Fred R. Volkmar and Roald A. Øien

More than 75 years have passed since Leo Kanner's classic (1943) description of the syndrome he called infantile autism. Over this period of time, many significant changes have occurred in our understanding of the condition and narrowed, or broadened, view of it. As we will discuss in this chapter, at present we paradoxically have two competing views—a narrow view more akin, in some ways, to Kanner's original paper and a broader view that reflects more generally an awareness that autism does shade off into normalcy (via the "broad autism phenotype"; Ingersoll & Wainer, 2014). The broader view has become, in many ways, more consistent with our evolving understanding of the genetics of autism.

These issues are of great relevance to the diagnosis of autism in infants and very young children. In Kanner's time, and for decades after that, it was not usual to see very young children who either seemed to have autism or to be at high risk for it. Indeed, on my (F.R.V.) first entry into the field in 1980, seeing a 4-year-old was equated with seeing a very young child with autism. Other chapters in this volume document the dramatic upsurge of interest in the first and earliest manifestations of the condition. Through studies of high-risk populations and use of new methods, considerable progress has been made in our attempt to see autism in its earliest form—before subsequent intervention and life circumstances and ongoing development have altered its course. In this chapter, we review the development of the concept from Kanner's original description to our current broad view of the autism spectrum, with particular emphasis on the diagnosis of ASD in infants and young children.

Issues in Diagnosis and Classification

Classification systems have different intended uses and purposes, including enhanced communication for clinical work, for facilitation research,

or for policy planning (Volkmar, Sukhodolsky, Schwab-Stone, & First, 2017). The consistent use of the same criteria for purposes of research has vastly increased the value of results obtained since, as a result, the critically important issues of generalization can be addressed forthrightly. In some countries, like the United States, use of specific diagnostic labels may also produce essential entitlements to services or treatments. This is especially so in the United States where the various states have a plethora of services and programs available (Doehring & Becker-Conttrill, 2013). Diagnostic systems that try to be both all-encompassing and useful for research as well as clinical purposes face unique challenges; such a task has been undertaken in the various DSMs since 1980. As noted elsewhere (e.g., see Jackson & Volkmar, 2019), alternatives are available, particularly the World Health Organization's approach of having different books for clinical and research use. Of course, for clinical work, establishing a working diagnosis is only one part of a broader diagnostic process that aims to decrease impairment and increase life choices and satisfaction. Other considerations also arise for comprehensive diagnostic approaches. Issues of reliability and validity are essential. That is, are there some independent validators that increase our concept in the basic diagnostic construct, and can the proposed criteria be meaningfully used by clinicians from different backgrounds and with different levels of experience?

Many tensions exist, for example, between clinical and research uses, between "lumping and splitting," and so forth. It is possible, for example, to increase diagnostic precision dramatically, but if the concept is too narrow, it cannot be generally applied. Conversely, it is possible that for research purposes a very narrow concept may facilitate identification of underlying pathophysiological processes. Another issue of importance for autism is the degree to which impairment (including, for children, interference in learning or development) must be viewed as an essential feature of the condition. Indeed, in some ways, Hans Asperger (1904–1980) himself explicitly raised this issue, for he viewed the condition he described more as a personality than as a developmental disorder, and he noted similar problems in the fathers of these children. As a practical matter, today this consideration arises in the issue of whether a person with social vulnerabilities, but who otherwise functions well, might be seen as having a disorder or a lifestyle/personality type. Some such persons might prefer to avoid feeling stigmatized by a categorical label, while others might wish to share their awareness of differences with others, including those with similar problems (Rutter, 2011).

A range of approaches to classification can be employed, including categorical and dimensional. In medicine, the dominant approach has been the categorical (presence/absence) model. Dimensional and categorical systems are not at all incompatible, however; it only becomes necessary to decide at what arbitrary point along a dimension a disorder is diagnosed. These

distinctions apply both to common medical problems such as hypertension or hypocholesterolemia and to developmental disorders such as intellectual disability. Of course, the selection of such cutoffs can be complex.

Dimensional approaches provide more information than simple categorical ones. The use of screening, rating scales, and diagnostic instruments for autism are all good examples of these approaches. A specific categorical cutoff is somewhat arbitrary. For autism, screening instruments, rating scales, and diagnostic instruments have been developed (see Ibañez, Stone, & Coonrod, 2014; Lord, Corsello, & Grzadzinski, 2014). While several instruments are now essentially of mere historical interest, it is important to realize the challenges that developers face when they undertake to develop new such approaches. Issues involving development of these instruments are profoundly complex. For example: Who will endorse the information? Is direct observation used? How much training is needed? Does the instrument take various forms (e.g., depending on the child's ability level)? What is the intended age range and developmental level? Psychometric issues such as reliability, validity, and administration fidelity must be addressed. Practical issues of ease of use, performance, and administration are all important. Also, the question of whether the instrument can yield some categorical measure (e.g., of presence/absence or severity of autism) may also need to be addressed. Increasingly, diagnostic instruments have been used as "change" measures. This reflects both the paucity of such instruments for measuring treatment effects and the complexity of developing an instrument that is, on the one hand, supposed to be stable but, on the other hand, is measuring treatment efficacy.

A different approach has been adopted, for example, by the Childhood Autism Rating Scale, Second Edition (CARS-2; Schopler, Van Bourgondien, Wellman, & Love, 2010) which rates behaviors in 15 categories with a 4-point scale—from normal to severely autistic. The summary score can be translated into an estimate of the severity of autism—absent, mild, moderate, or severe. This test, frequently used in schools, strikes a balance between research and clinical tensions. Other approaches have used results from normative assessment instruments like the Vineland (Volkmar et al., 1987). Still others occur more on screening or more complex procedures for researched autism (see Volkmar, Booth, McPartland, & Wiesner, 2014a).

Developmental considerations in diagnostic classification schemes began in the late 1960s as it became more complex; for example, it was thought that multiaxial methods might be needed (Rutter et al., 1969). With some modifications, this approach continues to be used, although in the recent DSM-5 (American Psychiatric Association, 2013) the broader childhood-onset group has been largely eliminated and replaced with a shorter neurodevelopmental disorders section (including autism). This reflects the long-standing goal of minimizing the age-related "sections" of

the manual. It remains to be seen how well this approach worked for other conditions.

The role of theory in guiding classifications has long been a thorny issue for classification schemes. Probably the primary lesson of the success of DSM-III (American Psychiatric Association, 1980) has been the importance of adopting a theoretical, but reliable, approach. Earlier versions of DSM were heavily theoretical, which posed problems at many levels. It is clear that for research purposes the research diagnostic criteria approach (American Psychiatric Association, 1980) has significantly advanced the study of many disorders, including autism.

For disorders like autism that are of very early onset, the fundamental nature of the particular condition and its impact on so many areas of development can markedly impact development in many ways. Understanding developmental factors, both typical and atypical, thus remains important. This importance is exemplified in the early impression that echolalia in autism was a maladaptive symptom and only later were its many potential adaptive functions recognized (Prizant & Rydell, 1984). Early-onset problems also increase the risk for comorbid conditions. In the study of individuals with intellectual deficiency, it became apparent that "diagnostic overshadowing" often led clinicians and investigators to fail to appreciate the presence of markedly increased rates of other disorders (Reiss, Levitan, & Szyszko, 1982). Conversely, longitudinal data also show the potential positive effects of environmental influence (e.g., lower rates of intellectual deficiency over time if the real-life acquisition of adaptive skills is considered; Rutter, 1991).

The role of etiology in the development of classification systems is an interesting one. Even when an etiology is known, for example, for a single-gene disorder, there usually is phenotypic heterogeneity. In some ways, tensions surrounding the issue of etiology are reflected in how Rett's disorder is approached in the DSMs. In DSM-IV (American Psychiatric Association, 1994), this syndrome was included in the overarching pervasive developmental disorder category but not because it was thought to be a form of autism (although this had been Rett's [1986] first impression). Instead, it was felt that this was the closest category and that, given its distinct course and clinical pattern, some etiology would likely be found. This was the case with the discovery of the *MECP-2* gene (Moretti & Zoghbi, 2006). In DSM-5, the disorder was eliminated, although cases could still, in theory, meet the criteria for the ASD category (in reality, this is not likely). The consideration of how to best approach classification of disorders with behavioral and developmental features but also strong genetic etiologies remains a matter of some controversy.

Like any human construction, classification systems can be misused (e.g., Hobbs, 1975; Gould, 1996). One of the worst mistakes in this regard is to equate people with diagnostic labels (e.g., "the autistic" or

"the schizophrenic" or "the diagnostic") rather than with person-centered language, which is always indicated. It is important to realize that categorical terms only capture a small fraction of the information relevant to the condition as it is expressed in that person, and much less the complexity of the individual in all of his or her facets. Scientifically, of course, it also presents a danger in that having a label can be assumed to be an explanation. Moreover, of course, having a categorical label should not be an obstacle to helping any person achieve his or her maximum potential in the home, school, and community settings.

Diagnostic Issues Specific to Classification of ASD

The history of autism research illustrates the importance of a robust and generally agreed-upon definition for research purposes. Before 1980, when DSM-III (American Psychiatric Association, 1980) was published, it was challenging to interpret research on what then was considered "childhood schizophrenia." After its official recognition in DSM-III, research on autism substantially increased; for example, in 2000 about 350 papers were published, while in 2010 this number had increased to 2,000 and during 2017, roughly 4,000 papers appeared! These numbers reflect the importance of diagnostic awareness and useful and generally agreed-upon definitions.

It is also important to reflect on the importance of such recognition for clinical purposes. Better awareness and classification can lead to better data to guide health policy and, of course, facilitate research on treatment. As we will discuss shortly, changes in diagnostic systems present opportunities as well as challenges. The opportunities arise given the possibility that data-based "tweaks" improved reliability. Conversely, if changes are significant, they may disrupt organizing research and may also have unintended consequences for service relative to eligibility.

From Kanner's Report to DSM-5

Kanner's Report

It is likely that reports of so-called "feral" children may represent the first reports of children with autism (Wolff, 2004; Candland, 1993). Although intellectual disability had been recognized since antiquity (Harris, 2006), interest in it and in child development, in general, began to increase with the Enlightenment and the debate of the role of nature versus nurture in child development (Hunt, 1961). By the mid-1800s, interest in psychiatric problems in children began to increase, and continuities with adult forms of mental illness were suggested (e.g., Maudsley, 1867), although little

provision was made for recognizing the relevance of developmental factors in syndrome expression. The late 1800s were, in particular, a time of great activity in psychiatric taxonomy with the recognition of dementia praecox (now termed schizophrenia) and manic–depressive illness (bipolar disorder). These concepts were rapidly extended to children (e.g., "dementia praecossisima"; de Sanctis, 1906). The early tendency to equate severe psychiatric disturbance in childhood with adult schizophrenia later posed a difficulty in recognizing the validity of autism as a different diagnostic category.

Leo Kanner emigrated to the United States from Nazi Germany first to work in a state mental hospital, but he was then recruited to Johns Hopkins Hospital to bridge the gap between psychiatry and pediatrics. He had completed the first textbook in the field in the 1930s before publishing his seminal paper "Autistic Disturbances of Affective Contact" (Kanner, 1943).

In this original paper recognizing autism, Kanner presented 11 cases of children he believed to lack orientation to people and social interaction that characterizes typical development. He was careful to acknowledge the early development of social engagement as noted by Arnold Gesell at Yale in his studies of typical infants. He provided thoughtful, careful, and insightful descriptions of these first cases. He then synthesized his observations into an overall summary emphasizing two features he felt were essential for diagnosis: (1) autism—or the early lack of social interaction and engagement and (2) insistence on sameness, or, put another way, difficulties dealing with change in the nonsocial world. With the wisdom of hindsight (and the tremendous body of work on normal social development), we now recognize that these may be the flip sides of the same coin—that is, if you are ready to play the social game, you are also ready for constant change. Kanner's use of the term *resistance to change* or *insistence on sameness* was intended to note the significant problems these children had with a change in nonsocial work. Although not recognizing communication problems as a central diagnostic factor, Kanner did mention many of the unusual features of language/communicative development that we now consider hallmarks of autism. For the individuals who engaged in communicative speech, unusual prosody and difficulties with nonliteral language and pronoun use were noteworthy.

Kanner's report was prescient in many respects. It focused on the apparent paradox of overengagement with change in the nonsocial world and lack of interest in the social world (Klin, Jones, Schultz, & Volkmar, 2005; McPartland & Pelphrey, 2012). In his first paper, Kanner suggested that autism was congenital, and he particularly noted the attractive appearance of the children in his study group (i.e., unlike the appearance of those diagnosed with other syndromes associated with mental retardation or what we now term *intellectual disability*.

Unfortunately, a few aspects of this groundbreaking work ultimately caused confusion. First, Kanner's use of the word *autism* was meant to convey the socially isolated and isolationist quality of the child's existence, but also it harkened back to its early meaning as self-centered *thinking* in schizophrenia. Similarly, his observation of the fact that some of his subjects did well on some parts of IQ tests suggested that children with infantile autism, as he termed it, were not also intellectually disabled. Early reports of individuals with autism and savant skills also fueled this impression. It was assumed that good performance on some parts of the IQ tests (puzzles and other nonverbal activities) was typical of overall abilities and that poor performance on verbal tasks reflected lack of engagement or desire to cooperate. Similarly, his observation of a lack of unusual physical features (as in trisomy 21) and the ability of the children to do well on some parts of IQ tests suggested that children with infantile autism were not also mentally retarded. It took many decades to realize that this assumption was not correct and that many individuals also developed a co-occurring intellectual disability (see Goldstein, Naglieri, & Ozonoff, 2009; Klin, Saulnier, Tsatsanis, & Volkmar, 2005; Volkmar & Nelson, 1990). This led to the impression that parents *had* to be very successful to have a child with autism, and this impression, in turn, led to speculation in the 1950s that parents might actually *cause* autism. This thinking led to an entire generation of parents who were traumatized by being blamed for their child's condition and by following the recommendation of ineffective intensive therapy, which in the end did nothing at all for their child's problem. This issue was one of several that became clarified in the 1970s as longitudinal data became available and it became clear that children with autism were very much at risk for development of seizure disorder (epilepsy).

As mentioned earlier, Kanner noted the attractive appearance of his cases and emphasized that they were without obvious physical signs of conditions like Down syndrome. Only as the children were followed did it become clear that they were at increased risk for developing seizure disorder and that autism was associated with a small number of highly genetic conditions (see Rutter & Thapar, 2014). Finally, in his initial case series, parents tended to be highly successful and accomplished people, but there was little awareness of the potential for selection bias (i.e., that only well-informed and successful parents would be able to find the one child psychiatrist in the country). Indeed, Kanner's early report likely reflected the fact that it was educated parents who were most likely to be able to reach the likes of Leo Kanner in the decades long before the internet! As a result, the impression in the 1950s and 1960s was that autism was a disorder of higher status (educationally and occupationally) families, and this notion, in turn, led to an impression that potentially experienced or deviant parenting might play a role in pathogenesis (e.g., Bettelheim, 1974). Subsequently,

autism was seen to be unrelated to parental education or socioeconomic status (SES; Wing, 1980).

From Kanner to DSM-III

Following Kanner's original description (1943), interest in autism gradually began to increase. Much of the work done relative to patients is, unfortunately, hard to interpret given the confusion with childhood schizophrenia. An entire line of work focused on parents as the "cause" of autism. This work (e.g., Bettelheim, 1974) suggested that separation from parents and psychodynamic therapy represented the only hope for remediation. This unfortunate thinking rested, in part, on Kanner's description of the parent's high levels of success and the very strong interest at the time in the effect of experience within a psychoanalytic theoretical framework. Several lines of work questioned the view of autism as a psychogenic disorder, and in addition, it was lumped in with the broad childhood schizophrenia/psychosis category.

In the United States, Rimland (1964), a psychologist and parent, provided a neurological model of autism; proposed some guidelines for diagnosis, along with the first screening/diagnostic instrument focused on new approaches to objective diagnosis; and provided a hypothetical neurobiological mechanism for autism (Rimland, 1964). Several lines of evidence also questioned the lumping of autism into the broader childhood psychosis category. In a series of studies, Kolvin and colleagues examined the clinical phenomenology of a large group of "psychotic children" (Kolvin, 1971). This work revealed a bimodal age of onset of "childhood psychosis," with a large group of cases identified as having difficulty in the first year of life, and then another group emerged with an onset in early and midadolescence. The first group of cases clinically resembled the children with autism described by Kanner, while the later-onset group had symptoms suggestive of schizophrenia (hallucinations, delusions, etc.). Family history data also suggested higher rates of schizophrenia in the later-onset than in the early-onset cases. Kolvin's study was convincing evidence that autism was a distinctive condition. Rutter (1972) summarized this work and suggested a reconsideration of autism as a distinctive condition.

Several other lines of evidence that emerged in the 1970s also provided valuable insights into autism. As children were followed over time, it appeared that the course of autism was also unusual (Kanner, 1971). As children were followed, a much higher than expected rate of epilepsy (seizure disorder) was seen (Volkmar & Nelson, 1990). While the early impression was that autism was not a strongly genetic condition, the first twin study (Folstein & Rutter, 1978) revealed substantial genetic contributions, with a very high rate of concordance in monozygotic twins (as compared

to same-sex maternal twins) who also had a higher than expected rate of recurrence, but not nearly as high as in the identical twin pairs.

Finally, the first studies of treatment efficacy (e.g., Rutter & Bartak, 1973) observed that structured teaching was much more frequently associated with improvement than unstructured psychotherapy. It was around this time that the first behavioral intervention studies were conducted, leading to a vast body of work on applied behavioral analysis (e.g., Lovass & Smith, 1988; Ferster, 1972). In the United States and the United Kingdom, parents began to organize intervention programs based on these principles.

By the late 1970s, a consensus emerged that autism (then referred to as infantile autism, autism, or Kanner's autism) was a distinctive condition. Several attempts were made to provide better diagnostic guidelines. Rutter (1978) proposed a straightforward definition based on Kanner's work. Rutter's definition included social difficulties (not just due to associated developmental delay/intellectual disability [ID]), communication problems (again not just due to ID), and unusual behaviors of the type that Kanner had noted, for example, resistance to change, stereotyped mannerisms, and so forth. Rutter's definition also included a requirement for early onset (by age 30 months). In contrast, Ritvo and colleagues (National Society for Autistic Children [NSAC], 1978), working in conjunction with the newly organized National Autism Society, proposed a slightly different definition that included features such as unusual rates/patterns of development and hypo/hypersensitivity. Both definitions have had a substantial impact on subsequent official approaches to defining autism. At the same time, there was consideration of the best multiaxial diagnostic approaches to use. Importantly, the Washington University in St. Louis group pioneered the use of research diagnostic criteria in the definition of psychiatric disorder (Spitzer, Endicott, & Robbins, 1978). This approach avoided the conundrums of past, more theoretically based definitions by adhering strictly to the descriptive phenomenology of the condition.

DSM-III

The growing body of work led to the decision to include a new category for "infantile autism" as an officially recognized diagnosis in the groundbreaking DSM-III (American Psychiatric Association, 1980). A new term, *pervasive developmental disorder* (PDD), was coined as the class to which infantile autism and similar conditions were assumed to belong. Unfortunately, this term led to some confusion, and with the wisdom of hindsight, better terminology could have been employed. However, the critical accomplishment was the official recognition of autism as a distinctive condition.

At that time, autism was indeed regarded as one of the best examples of a "disorder" in psychiatry since it did not seem to shade off into normalcy

(Rutter & Garmezy, 1983). This view, of course, has now substantially changed (Ingersoll & Wainer, 2014). The definition provided for infantile autism was monothetic (i.e., all features have to be present), including social and communication features consistent with Rutter's (1978) definition as well as early onset. The social criterion of pervasive lack of response to others was not applicable to older individuals since children did develop some social skills over time, so a new category of "residual" infantile autism was included. Three other conditions were included in the new PDD class: (1) childhood-onset PDD (for children who developed autism after a period of normal development but then otherwise met the criteria for infantile autism); (2) residual childhood-onset PDD (a parallel to the residual infantile autism category); and (3) as was true throughout DSM-III, a new atypical pervasive developmental disorder for situations that did not meet all the features of a specific PDD, but the individual had problems suggestive of autism or a related condition. (This last-named category would morph into pervasive developmental disorder not otherwise specified [PDD-NOS] in DSM-III-R and DSM-IV.) Importantly, in many respects the foundation of the now recognized spectrum of difficulties associated with autism in some fundamental sense arose from this attempt, in DSM-III, to acknowledge that individuals had social and related difficulties that did not quite correspond to the official definitions provided.

DSM-III was a tremendous advance over its predecessors. For autism and related conditions, the official recognition of autism stimulated an already growing body of research. Given its rigorous approach, DSM-III was quickly adopted around the world, and, for autism, this further advanced international work on the condition. For autism and related conditions, several problems quickly became apparent the first time autism had been so recognized, and it is not surprising that some problems were quickly identified (Volkmar, Cohen, & Paul, 1986).

As noted earlier, the definition of "infantile" autism indeed focused on the most classic, presumably earliest, forms of the condition marked by little responsiveness to others. Clearly, any developmental approach was lacking, and the term *residual* infantile autism seemed highly inappropriate given the potentially very severe manifestations of the disorder in older individuals who no longer were "pervasively" unresponsive. As Wing and others have noted, social skills do develop, although often in unusual ways as children with autism age (Wing & Gould, 1979). The language (not communication) criteria were similarly somewhat narrow. The homothetic approach to the diagnosis of infantile autism was inflexible, and, somewhat paradoxically, the polythetic (various features could lead to a diagnosis) definition for childhood-onset PDD was much more flexible. The rationale for including this latter condition was also somewhat questionable: it appeared to be aimed specifically at the rare children who developed normally for

a considerable period of time (usually 3 or 4 years) and then seemed to develop a particularly severe clinical presentation of autism. These children were first described by Heller (1908), and a few had been seen in Kolvin's case studies (Kolvin, 1971). Finally, use of the term *atypical* PDD has a somewhat unique history and complications relative to autism. Rank and colleagues had used an earlier term—atypical development—to describe children with unusual developmental patterns, some of which suggested features of autism (Rank, 1949; Rank & MacNaughton, 1949). Finally, given the long history of confusing autism with schizophrenia, it is perhaps not surprising that this was made an exclusionary criterion for autism. Of course, given the frequency of schizophrenia, there would be no reason to assume that adolescents or adults with autism were somehow protected from developing schizophrenia. Moreover, studies suggest that indeed this does occur at about the rate expected in the general population (Volkmar & Tsatsanis, 2002). Although DSM-III was a significant and clear advance problem in this and other categories, it prompted a relatively rapid revision in DSM-III-R (American Psychiatric Association, 1987).

DSM-III-R

Given the significant difficulties identified with the DSM-III definition, major revisions were undertaken with DSM-III-R (American Psychiatric Association, 1987). In some sense, these are summarized in the change of name of the main category—from *infantile autism* to *autistic disorder*. The emphasis was on providing a more flexible and developmentally oriented definition (see Siegel, Vukicevic, Elliott, & Kraemer, 1989; Waterhouse, Wing, Spitzer, & Siegel, 1993).

A polythetic definition was adopted, with *criteria/items* grouped into three domains: social development, communication and play, and restricted interests and repetitive behaviors (sometimes giving an example within the criteria). To achieve a diagnosis of autistic disorder, at least eight criteria had to be endorsed, with at least two social and one each from the other two categories. A field trial was used to help refine the DSM-III-R definition. Unfortunately, it suffered from several problems (cases were rated based on records, and the comparison group was highly inappropriate).

The pros and cons of the new definition quickly became evident. The positives included much better attention being given to issues of developmental change and developmental level (Volkmar, Cicchetti, Cohen, & Bregman, 1992b). At the same time, it also was noted that the new system appeared to have increased false-positive rates (Volkmar et al., 1992b; Factor, Freeman, & Kardash, 1989; Hertzig, Snow, New, & Shapiro, 1990).

The nature of the criteria was problematic in inclusion of examples within criteria. Clearly, the new approach focused more on present

assessment than on history. Concern increased as it appeared that the new DSM-III-R was diverging substantially from the draft ICD-10 approach (Volkmar, Cicchetti, Bregman, & Cohen, 1992a).

The World Health Organization's *International Classification of Diseases, 10th edition* (ICD-10; World Health Organization, 1994) differed from DSM in several ways. At first a "two-books" approach was adopted, with clinical guidelines published separately from diagnostic criteria for research. Other issues were noted as well, for example, in the approach to dealing with comorbidity. Adopting the two-volume approach did mean that the research definition could be substantially more detailed. Clearly, significant differences between these two official systems had the potential for complicating research (Volkmar et al., 1992a). These issues were given serious consideration in the major revision of the diagnostic approach undertaken for DSM-IV (American Psychiatric Association, 1994).

DSM-IV and ICD-10

The revision process for DSM-IV was extensive. It included work groups for various diagnostic categories; these reviewed existing research and identified areas of consensus and controversy. Changes from DSM-III-R were made only when they could be justified and also in consideration of the pending changes with ICD-10 (Volkmar & Tsatsanis, 2002). For the autism/PDD categories, this included a series of commissioned literature reviews (see *Journal of Autism and Developmental Disorders,* December 1992 issue) that addressed a range of issues. One major problem was whether and how best to include other specified disorders (i.e., other than the atypical/not otherwise specified) category. The draft ICD-10 included Asperger syndrome (Sharma, Woolfson, & Hunter, 2012; Szatmari, 1991), Rett syndrome (Rutter, 1994; Gillberg, 1994; Tsai, 1992), and the apparently rare condition variously termed *disintegrative psychosis, Heller's syndrome,* or *childhood disintegrative disorder* (Volkmar, 1992). It was clear that, if possible, comparability of DSM-IV and ICD-10 was desired (Rutter & Schopler, 1992).

For the diagnosis of autistic disorder, a series of papers suggested that DSM-III-R was indeed more developmentally oriented but also overly broad (Volkmar et al., 1992b). Thus, a major issue was the balance of sensitivity and specificity of diagnostic approach while simultaneously maintaining a flexible and developmentally oriented definition of the condition. As a significant part of this process, an international field trial was conducted (Volkmar et al., 1994). This field trial included over 20 sites and over 100 raters, with nearly 1,000 cases rated. To avoid the problem of the DSM-III-R, field trial cases were included from clinics/clinicians only when the clinician felt that autism was a reasonable part of the differential diagnosis.

Information on raters, for example, level of experience, was also obtained. The criteria rates included previous DSM criteria as well as draft ICD-10 and new potential criteria. For most of the cases the raters in the field trial had multiple sources of information available to them (history, past examinations, current assessment). Clinicians were also asked to give their best estimate of the clinical diagnosis regardless of the criteria used.

The results of this field trial can be briefly summarized. Most importantly, the data suggested that DSM-III-R emerged as a diagnostic outlier (compared to DSM-III in the "lifetime" diagnostic sense or ICD-10 or clinician judgment). DSM-III-R had a high rate of false-positive cases, particularly in cases with greater intellectual impairment. The detailed draft ICD-10 research definition worked well but was more detailed and extensive than desired for DSM-IV. In concordance with the ICD-10 revision process and after extensive analysis, a new set of criteria were proposed that were conceptually identical in ICD-10 and DSM-IV.

Although reliability among clinicians has been questioned relative to clinical diagnosis (Lord et al., 2012), for the field trial data agreement among experienced raters on clinical diagnosis was excellent. For less experienced raters, reliability was increased by the use of the new diagnostic approach. Factor analysis produced several potential solutions, including the traditional three categories of criteria approach (social, communication, and restricted interests). A two-factor solution (social communication and restricted interests) and a five-factor solution (in which restricted interests criteria sorted into three groups) also were identified based on the various constraints imposed.

The field trial did not focus solely on the autistic disorder. As part of the field trial, data were collected on a potential "new" disorder tentatively included in ICD-10. These conditions, now seen as part of the broader autism phenotype, included Asperger syndrome, Rett syndrome, and childhood disintegrative disorder. Of these conditions, Asperger syndrome was probably the most widely recognized. The condition had been initially described by Hans Asperger (1944) a year after Kanner's paper was published. Asperger emphasized not only the autism and social vulnerability of the disorder but also its heritability. He noted that he had reviewed the condition more as a personality issue than as a developmental disorder—all issues that had been raised in recent years relative to the broader autism phenotype (Ingersoll & Wainer, 2014) and the growing body of work on the diverse genetic contributions to autism and related conditions (Rutter et al., 2014).

The DSM-IV field trial provided some data supporting the inclusion of Asperger's disorder in DSM-IV. Nearly 50 cases with this clinical diagnosis had been included, and these cases differed in important ways from both similarly cognitively able cases of autism and PDD-NOS (Volkmar et al., 1994). Interest in the condition increased dramatically from fewer

than 100 scientific papers from 1944 to 1993 to about 1,700 after DSM-IV appeared. The decision to include Asperger syndrome was somewhat controversial, and the criteria finally proposed were, unfortunately, a compromise that, in retrospect, could have been better addressed with a more detailed and explicit definition, given that autistic disorder took precedence (see Rutter, 2011; Volkmar, Klin, and McPartland, 2014b). Based on the results of the field trial, support was also given to including Rett syndrome within the overarching PDD class—not so much because it was thought to be a form of autism (Rett's [1966] initial presumption) but because it was felt to be essential to include it somewhere. Cases with unusually late onset of autism were also explicitly sought for the field trial and provided some support for including a "new" category of childhood disintegrative disorder as well (Volkmar & Rutter, 1995).

As was true in DSM-III and DSM-III-R, a "subthreshold" condition was included in DSM-IV for cases with problems suggestive of autism, but it failed application of formal diagnostic criteria. In DSM-IV, this was termed *pervasive developmental disorder not otherwise specified* (PDD-NOS) and in ICD-10, *atypical autism*. As noted, this category previously had its own compelling history antedating DSM-III, and as the study of the broader phenotype has increased, this group of cases has assumed increasing importance (Ingersoll & Wainer, 2014). The DSM-IV and ICD-10 convergence lasted for almost two decades, when both systems began to be revised again.

DSM-5

The process was different for DSM-5 as compared to its immediate predecessors. The project was based at the headquarters of the American Psychiatric Association rather than at a university (as had been the case since DSM-III). The American Psychiatric Association also played a greater role in the organization and structure of the process. Some notable early decision was made, for example, to revamp the multiaxial categories, eliminate (as much as possible) subthreshold categories, and use large datasets collected in structured diagnostic instruments (i.e., rather than contemporaneous clinician ratings). Another important goal was, as much as possible, to include childhood-onset disorders within regular diagnostic groupings, rather than have a special child's section. For some conditions (including autism, intellectual deficiency, and related developmental problems), this was not possible, and so these conditions were grouped into a neurodevelopmental category to emphasize their special status. Goals included improving DSM-5 as much as possible over its predecessor. Of course, for autism there were some unique challenges: the DSM-IV/ICD-10 criteria had been used successfully around the world, and research and clinical interest had exploded since DSM-IV appeared. These criteria had themselves been

translated into diagnostic assessment instruments, and the results of these assessments were now being used to inform the DSM-5 criteria.

In creating the DSM-5 criteria, the Neurodevelopmental Disorders Work Group sought to preserve the strengths of the DSM-IV approach while improving upon its limitations. The criteria provided in DSM-IV were highly effective in supporting the development of standardized assessment methods and in facilitating research, with scientific publications on autism increasing dramatically. Several criticisms of the DSM-IV approach could readily be made. The DSM-IV field trial was large and international in scope and included many individuals, but it was not based on an epidemiological sample. While young children had been included in that field trial, there had been an increased interest in making a diagnosis as early as possible (Chawarska, Klin, & Volkmar, 2008), and work using DSM-IV criteria suggests reduced diagnostic stability before age 3 (Lord, 1996).

More serious concerns were raised about the validity of the various additional subtypes of autism included in DSM-IV (e.g., Mayes, Calhoun, & Crites, 2001; Ozonoff & Griffith, 2000). Probably the greatest disagreement concerned the inclusion and definition of Asperger syndrome. The criteria included in DSM-IV were unsatisfactory in some respects, and given the very significant difference in the diagnostic approach, it is not surprising that this compromise definition proved controversial (see Volkmar et al., 2014b; Bennett et al., 2008). In a partial response to this dissatisfaction, the entire text of the Asperger syndrome category in DSM-IV was replaced in DSM-IV-TR (American Psychiatric Association, 2000). Lord et al. (2012) reported in a large multisite study that assessment location was more predictive of a diagnosis of Asperger syndrome than were characteristics of the individual child, suggesting the persistence of various specific approaches to diagnosis. For childhood disintegrative disorder, concerns were raised about the apparent rarity of the condition and its differences from autism (Hansen et al., 2008; Jones & Campbell, 2010; Kurita, Koyama, Setoya, Shimizu, & Osada, 2004; Luyster et al., 2005; Rogers, 2004).

For the broader PDD-NOS group, the growing body of genetic work suggests a reconceptualization of PDD-NOS as a milder end of the autism spectrum, that is, the broader autism phenotype (Piven, 2001; Wainer, Block, Donnellan, & Ingersoll, 2013).

In DSM-5 (American Psychiatric Association, 2013), a new term, *autism spectrum disorder* (ASD), was proposed for what had been the pervasive developmental disorder category. Within this category, the prior diagnosis was merged into a single ASD, and a new communication disorder was proposed as well: social communication disorder. Rett syndrome was eliminated unless a child with the syndrome also met the criteria for the new ASD category. Based on a factor analysis of a large set of data from standardized instruments, the traditional three sets of criteria (social,

communication, and restricted interests/repetitive behavior) were reduced to two (social and communication features having been combined). The new social communication category became nomothetic; that is, it required that an individual demonstrate symptoms across all three clusters to meet the criteria for ASD. In contrast, the restricted and repetitive behaviors domain remained polythetic, requiring evidence of symptoms in two of four symptom groupings. A new criterion related to sensory difficulties was included in the latter category. The new diagnosis of social communication disorder was defined by pragmatic difficulties and problems in the use of verbal and nonverbal communication in social contexts. This condition, a communication disorder, was seen as distinct from ASD, although it had many similarities to the older PDD-NOS concept. The DSM-5 revision process was complicated and data-oriented (Guthrie, Swineford, Wetherby, & Lord, 2013; Huerta, Bishop, Duncan, Hus, & Lord, 2013; King, Veenstra-VanderWeele, & Lord, 2013; Lord & Gotham, 2014).

DSM-5 also introduced a series of specific conditions for ASD, reflecting a general effort to include themes and descriptors that apply transdiagnostically. For example, a first specifying condition marks the presence of any associated etiological condition; a second specifying condition, common across DSM-5 diagnostic categories, describes the required level of support and impact on a person's functioning in the two symptom domains; a third specifying condition notes the level of any associated intellectual disability; and, similarly, a fourth indicates whether language impairment is present. The final specifying condition indicates whether catatonia is present. As noted previously, the use of these specifying conditions is meant, in some ways, to replace the previous multiaxial system.

Questions about DSM-5 were raised even before DSM-5 appeared, and two studies had seriously questioned whether the new diagnostic label significantly narrowed the diagnosis concept and substantially reduced eligibility for service in children who were previously provided therapeutic interventions. The first of these studies, Mattila et al. (2011) used an earlier version of the draft DSM-5 criteria and found significant difficulties with the system not "capturing" the problems of higher functioning individuals on the spectrum (including both autism and Asperger syndrome). Since this study had used an early version of the criteria, the results were questioned. However, McPartland, Reichow, and Volkmar (2012) reanalyzed data from the DSM-IV field (essentially creating algorithms to "cross walk" between the old and new system). Their study used the most recent criterion set, but several problems were again raised about DSM-5. A notably large number of higher functioning (IQ > 70) individuals failed to meet the new criteria. The authors raised the issue of whether DSM-5's increased stringency was consistent with awareness of the problems faced by more cognitively able but socially disabled individuals. In their study, a large proportion of individuals with autism (as diagnosed in DSM-IV) who were high functioning lost their

label, while a substantial majority of those with Asperger syndrome and PDD-NOS also lost their label and thus their eligibility for services. Given these concerns, a final criterion was added to DSM-5, allowing individuals with "well-established" diagnoses of autism, Asperger syndrome, and PDD-NOS to keep their diagnosis. While addressing the immediate problem, this solution created other issues since it effectively established the continuity of the old system while simultaneously creating a new and more stringent one.

Some 5 years after DSM-5, a reasonably large body of work appeared, generally supporting the results earlier obtained, for example, by Mattila et al. (2011) and McPartland et al. (2012) but with some notable additions. In one study of cognitively able adults compared on DSM-IV, ICD-10, and DSM-5 criteria (Wilson et al., 2013), over half of the cases with an ICD-10 PDD diagnosis also met DSM-5 criteria for ASD, with nearly 20% of those not meeting criteria for DSM-5 ASD meeting criteria for social communication. Worley and Matson (2012) demonstrated that individuals meeting proposed DSM-5 criteria tended to have more severe impairments than individuals meeting DSM-IV-TR criteria, a pattern replicated by Matson and colleagues (Matson, Beighley, & Turygin, 2012a; Matson, Hattier, & Williams, 2012b) who also reported that 47.8%—nearly half of toddlers meeting DSM-IV-TR ASD criteria—did not meet DSM-5 criteria. Another study noted some potential difficulties with DSM-5 criteria among females (Fraser et al., 2012, and see Matson et al., 2012a). A study of an existing dataset of adults indicated higher sensitivity to parent reports than observational assessment, highlighting the influence of the assessment method on ascertainment of cases (Mazefsky, McPartland, Gastgeb, & Minshew, 2013).

The utility of DSM-5 in toddlers has attracted considerable attention, given the increased emphasis on the presence of repetitive, restricted behaviors; these behaviors are generally believed to manifest robustly only somewhat later in development (Chawarska, Marcari, Volkmar, Kim, & Shic, 2014). Worley and Matson (2012) confirmed this concern, and it has been reported by other investigators as well (e.g., Barton, Dumont-Mathieu, & Fein, 2012). Other studies have raised concern that cases with a previous diagnosis of PDD-NOS often failed to exhibit the breadth of symptoms required by DSM-5 (Gibbs, Aldridge, Chandler, Witzlsperger, & Smith, 2012; Taheri & Perry, 2012).

In their recent meta-analysis of 25 papers, Smith, Reichow, and Volkmar (2015) found that most studies showed that between 50 and 75% of individuals would maintain their diagnosis. The significant difficulties related to higher functioning cases, and those—paradoxically, given the name change in DSM-5—are part of the broader autism spectrum. Many potential limitations were found in the studies reviewed, notably, use of historical data and reliance on specific assessment methods (i.e., clinician observation versus parent report). Methodological variations may have a significant impact on the results obtained (Mazefsky et al., 2013). In

addition, most of the studies to date have been conducted within research settings so the question of how findings generalize to more traditional clinical settings remains unclear (Tsai, 2012). Somewhat paradoxically, highly relevant data relative to the validity of Asperger syndrome appeared after the decision to eliminate it from DSM-IV. For example, Chiang, Cheung, Brown, and Li's (2014) meta-analysis examining reported IQ profiles in 52 studies comparing cases with autism spectrum disorder and Asperger syndrome showed robust differences in patterns obtained across all studies. This suggested that they indeed represent distinctive subtypes on the autism spectrum. As a result, many of these cases will no longer qualify for a label and potentially for relevant services.

Several vital decisions likely had a deleterious impact on DSM-5. At a surface level, if one compares the over 2,200 ways a person could achieve a diagnosis of autistic disorder in DSM-IV to the 12 ways in DSM-5, one would reasonably assume that the latter is a stricter, less flexible diagnostic construct. Some aspects of the process were severely constrained by the American Psychiatric Association, presumably in the interest of streamlining the process but also to cut costs (Greenberg, 2013). For example, the use of data from excellently structured research interests may not capture the reality of real-world settings, and in contrast to DSM-IV, the field trials for DSM-5 focused primarily on aspects of reliability. Including sensory issues raises other problems in the slightly different approach of the DSM-IV field trial. This item had *not* worked well in differentiating autism from intellectual disability (Volkmar et al., 1994). The inclusion of a new communication disorder was not well justified in research, and it does not precisely correspond to the needs of individuals with Asperger syndrome or PDD-NOS in DSM-IV terms. Other aspects of the system are somewhat arbitrary. For example, use of catatonia as a specific modifier for ASD seems odd given the rarity of that association. The usefulness of the specifiers also remains to be clarified (Gardner, Campbell, Keisling, & Murphy, 2018).

The move from the traditional three-symptom clusters to two has some practical disadvantages. It rests on the results of a factor analytic study of a large set of data (Huerta et al., 2012). However, other studies (e.g., Sipes & Matson, 2014) note that several solutions are possible, and in DSM-IV, field trial factor analysis yielded reasonable two-, three-, or five-factor solutions (Volkmar et al., 1994). Any of these approaches might have been justified but the three-factor, polythetic solution yielded more flexible applications consistent with the notion of an autism spectrum.

Thus, paradoxically, despite the welcome name change of the overall category to autism spectrum disorder, the concept itself is now the narrower "Kanner's autism" that we had moved away from in the past. Perhaps the swing of the pendulum is welcome, but if it prevents early detection of cases or provision of services to individuals, it is quite problematic. Of course,

the "grandfathering" in of cases from DSM-IV also creates issues for longitudinal and epidemiological research.

Dimensional Approaches to Diagnosis

Although dimensional instruments and dimensional assessments are not the primary focus of this chapter, they are highly relevant and, particularly with DSM-5, have had a significant role in the development of categorical criteria. Since Rimland's first development of a diagnostic checklist (Rimland, 1971), many such instruments have been developed—some for screening and others for diagnosis (see Lord & Gotham, 2014, and Ibañez et al., 2014, for comprehensive reviews). Some of these instruments focus on infants and younger children, other older individuals, or the more cognitively able; other instruments are based on parent or teacher reports and still others on direct observation; and most focus on autism but a few on Asperger syndrome (Campbell, 2005).

In some cases, instruments have been developed specifically to assess the range of issues relevant to the broader autism phenotype (e.g., Constantino & Todd, 2000). It is important to note that, particularly for the most psychometrically robust instruments, a high degree of training is needed (a topic relevant to their use in DSM-5). Other dimensional approaches, such as use of tests of intelligence, executive functioning, and adaptive skills, are, of course, also highly relevant to the assessment of persons with ASD. It is important to note that, unlike these normative approaches, those designed for use in ASD face unique challenges.

Challenges for dimensional assessment in autism/ASD include the broad range of syndrome expression, age, comorbidity, and IQ-related issues in syndrome expression; the relevance of historical information versus current examination; and the degree to which sometimes highly infrequent (but essential) behaviors are sampled. There are all the usual problems of reliability and so forth (see Lord and Gotham, 2014). Aspects of item administration or scoring can present challenges; we have seen examples in which motor tics were mistakenly coded as stereotyped mannerisms. The degree to which clinical judgment is essential also varies across instruments, as do flexibility in administration and the intended range of age or developmental level.

In theory, the potential for quantifying symptoms has essential research implications, providing measures of severity that can be assessed during treatment or providing potential new approaches to subtypes or consistency in genetic studies. These approaches have essential uses and limitations. An understandable tension exists between research and clinical use. Screening instruments present other complexities and sources of controversy (see Barton et al., 2012; Øien, et al., 2018a, 2018b) as compared

to diagnostic instruments. The latter may focus either on parent report or direct observation (or both). Available diagnostic instruments also probably work best in school-age children with ASD who have some language and mild to moderate cognitive disability. Their use becomes more complex at other parts of the age and IQ range. The problems raised by comorbidity are major and substantial (see Miot et al., 2019; Hawks & Constantino, 2020) and are approached differently in official categorization schemes. For autism and related disorders, there is also a growing awareness that having such conditions increases the risk for other problems. In more cognitively able individuals with autism and Asperger syndrome, for example, higher than expected rates of mood and anxiety problems are noted (White, Bray, & Ollendick, 2012; Spiker, Lin, Van Dyke, & Wood, 2012; Stewart, Barnard, Pearson, Hasan, & O'Brien, 2006). These issues may have important implications for assessment and treatment, but sadly, basic data are lacking in many areas (e.g., there is more or less a total absence of work on rates of suicidal ideation/behavior in adolescents and adults with ASD). These issues become very relevant to DSM-5, given the decision to rely on data from tests rather than the results of field trials as in DSM-IV. Although the desire to make use of a considerable body of research on these instruments is commendable (Regier et al., 2012), there may be many challenges in translating them into "real-world" clinical practice.

Current Areas of Debate and Controversy

Several different areas are the source of some controversy or concern. As previously noted, DSM-5 apparently takes a much more stringent approach to a diagnosis of autism (at least for new cases as old cases are "grandfathered" in). This may present some challenges for newly diagnosed young children who in the past under DSM-IV would have had a diagnosis of autism, as well as for higher cognitively functioning individuals who often come to diagnosis somewhat later (and of course for higher functioning toddlers).

A second area of concern centers on the complex issues of comorbidity (Rutter, 1997). This issue has been dealt with in different ways by different systems. ICD-10, for example, had a preference for avoiding additional diagnosis and explicitly included several comorbid categories. DSM has historically been more welcoming of multiple diagnoses. These issues become more complex as children become older and are at risk for other problems—notably anxiety and depression—increases. Of course, even in young children comorbid diagnosis can be an issue, for example, within seizure disorder or intellectual disability. DSM-5 provides some, although limited, coding for certain possibilities, such as specific genetic diseases. It

remains unclear as to what approach, over time, will prove the most helpful.

Finally, relatively little research has been conducted to assess the potential biases in instruments used for purposes such as screening and assessment; such biases can be toward cultures, ethnicities, genders, and SES. The potential biases of such instruments are of great importance, as they often affect clinicians' judgments; thus, selecting the most appropriate one will be critical (Cicchetti, 1994).

Even though few studies are standardized across demographic variables, such as age, gender, education, culture, ethnicity, and a range of other variables that could potentially affect the performance of such tools, it is important that we acknowledge such limitations to concurrent instruments, the so-called "gold-standard" instruments. Standardizing instruments according to such variables allows for norms relevant to a given nation, gender, or a specific culture. However, multiple factors affect the possibility of standardizing a given test for all potential biases.

Concurrent gold-standard instruments perform well in identifying ASD in most studies, especially when using ADOS (Autism Diagnostic Observation Schedule) and ADI-R (Autism Diagnostic Interview—Revised) (see Lord et al., 2014) to complement each other (Øien & Nordahl-Hansen, 2018). However, there is sparse knowledge of how screening and diagnostic instruments detect ASD in, for example, eastern cultures, different ethnicities, or between sexes (Øien & Nordahl-Hansen, 2018). Many instruments specific to the identification and assessment of ASD are developed and standardized in the United States and Europe and are often translated into different languages for use in other populations. In many such cases, no validation study is conducted for each translation. On these grounds, it is increasingly important to be aware of and to develop new research on how culture, ethnicity, SES, and gender affect the performance of such instruments. A potential factor, such as sex, could affect how well screening instruments perform across various cultures because humans, especially parents, could rate behaviors differently based on their cultural views on normality.

A study by Vanegas and colleagues (Vanegas, Magaña, Morales, & McNamara, 2016) revealed that the sensitivity and specificity of the ADI-R were moderate in a U.S. Latino sample, but lower than previously reported. These authors argued that the tool needs to be standardized for different languages and cultures. Cross-cultural differences in ASD have been reported in earlier studies (Elsabbagh et al., 2012) and are thought to affect how ASD is perceived, diagnosed, and treated in different cultures (Freeth et al., 2014). An example comes from the ethnic Norwegian minority, the Sami population, among whom disorders such as ASD and other mental health issues have been less prevalent than in the majority community

(Nergård, 2006). It is also important to think about the concurrent validity of an instrument in the context of culture; for example, societal and cultural changes happen over time and might cause a gap between what was considered normative at the time of development and what is so considered currently. Furthermore, a range of other factors such as behaviors and temperament in males and females could affect how well instruments perform in detecting and diagnosing ASD (Dworzynski, Ronald, Bolton, & Happé, 2012; Øien et al., 2018a, 2018b). Some of the more complex issues in identifying and diagnosing autism at an early age are related to the heterogeneous presentation of the disorder. Heterogeneity in etiology, behaviors, core symptoms, cognitive skills, adaptive skills, language and communication, the patterns and time onset of diagnosis, and core symptom patterns elicits immense complexities in the clinical detection of the disorder and ultimately affects treatment and treatment planning (Ozonoff et al., 2010; Zwaigenbaum et al., 2015). Previous DSMs had a strict age-of-onset criterion, but it was removed from DSM-5 (American Psychiatric Association, 2013) because symptoms might not become evident until social demands exceed the child's capabilities (Ozonoff et al., 2015). Furthermore, symptom expression might also vary depending on verbal and nonverbal functioning (Chawarska et al., 2014). These challenges do not apply only to clinical detection, but potentially affects when parental concern emerges, consequently leading to diagnosis later rather than early.

Both categorical and diagnostic instruments face additional challenges relative to diagnosis of autism in infants and young children. These complexities include the marked potential for developmental change, the sometimes later development of autism (e.g., following regression), and challenges posed by the complex clinical presentation of other disorders such as language/communication problems and intellectual disability. In a study by Ozonoff et al. (2015), nearly half of the children with ASD outcomes were not so identified at age 2 and didn't receive a diagnosis until age 3. In her original longitudinal study, Lord (1996) noted that stereotyped mannerisms frequently developed in significant ways after the age of 2. Before 36 months of age, there is much more potential for diagnostic instability. After that time, however, this instability becomes much less common, and children who clearly have autism tend to retain this diagnosis (Ozonoff et al., 2015). However, truly major changes can be made in very young children, with some 3- and 4-year-olds dramatically responding to treatment.

These problems will, of course, be less problematic should good biomarkers for autism be identified. These might be genetic (Rutter et al., 2014), biochemical (Anderson, 2014), neurophysiological (McPartland, Dawson, Webb, Panagiotides, & Carver, 2004), neuroanatomical (Chawarska, Chang, & Campbell, 2015), or even behavioral (Chawarska, Ye, Shic, & Chen, 2016). But to date, no biomarker has been identified. Accordingly,

in both clinical and research settings, it will be wise to be careful relative to the surety of early diagnostic assignment and to include an explicit follow-up aspect to both clinical diagnosis and research. For the latter, it is also critical that control and comparison groups include infants and young children with developmental delays *not* associated with vulnerability. Attention to the standardization of methods and stimuli is also critical, for major differences can be noted depending on the methods used (e.g., see Ozonoff et al., 2010; Rowberry et al., 2015; and Chawarska, Macari, & Shic, 2013). Work attempting to identify specific subtypes in early-age groups may be of great interest in this regard (Kim, Macari, Koller, & Chawarska, 2016).

Summary

In the more than 75 years that have passed since Kanner's classic description of infantile autism, noteworthy changes have taken place in our conceptualization of this disorder. The first decades of work on the condition were plagued with confusion about its relationship to other conditions (notably, schizophrenia), along with some noteworthy misconceptions about etiology, social class factors, cognitive abilities, and treatment approaches. During the 1970s, evidence emerged showing autism to be a unique condition that was distinctive in many ways. It had a strong brain and genetic basis, and it responded best to structured treatment designed to help the child compensate for the obstacles the syndrome poses for learning and development. The official recognition of the condition as "infantile autism" in 1980 by DSM-III was particularly important, and since that time the literature on autism has vastly increased.

As the same time, several tensions continue to the present. The first definition of autism in DSM-III focused on "infantile" autism—that is, in its most classic form, as it presumably exhibited itself in infancy. The issue of development was dealt with by including a "residual" category for those who had once met the criteria for the infantile form of the disorder. This approach was unsatisfactory, and so the next revision adopted an explicit orientation. The DSM-IV and ICD-10 convergence remained the gold standard for several decades. As noted above, with DSM-5 the pendulum has (somewhat paradoxically given the name change to autism spectrum disorder) swung backward to focus more on the prototypical cases of "Kanner's autism." As we note above, higher cognitive function cases are now more likely be excluded from the diagnosis, although, again paradoxically, those who had a diagnosis before DSM-5 are allowed to keep it!

The move in DSM-5 to autism spectrum disorder reflects an interesting and growing body of research on the autism spectrum. This had its origins in the recognition of atypical or "not otherwise specified" forms

of the disorder. With time, a growing body of work suggests that autism does indeed shade off into normalcy. This work is consistent, in many ways, with what have become the rather complex genetic origins of the condition.

As we note in this chapter, for infants and young children, a growing awareness of the disorder and provision of new evidence-based treatments have presented important opposites for optimizing learning and eventual outcomes. As the same time, our lack of biological markers for the conditions and the rather variable performance of screening tests present important obstacles. As noted elsewhere in this volume, a range of new methodologies are being presented to focus on autism as it is first expressed. These studies will move to even earlier development, for example, looking for differences in utero. Clearly, while much work remains to be done, many advances have been made.

REFERENCES

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: Author.

Anderson, G. M. (2014). Biochemical biomarkers for autism spectrum disorder. In F. R. Volkmar, R. Paul, S. J. Rogers, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (4th ed., pp. 457–481). Hoboken, NJ: Wiley.

Asperger, H. (1944). Die "autistichen Psychopathen" im Kindersalter. *Archive für Psychiatrie und Nervenkrankheiten, 117,* 76–136.

Barton, M. L., Dumont-Mathieu, T., & Fein, D. (2012). Screening young children for autism spectrum disorders in primary practice. *Journal of Autism and Developmental Disorders, 42*(6), 1165–1174.

Bennett, T., Szatmari, P., Bryson, S., Volden, J., Zwaigenbaum, L., Vaccarella, L., . . . Boyle M. (2008). Differentiating autism and Asperger syndrome on the basis of language delay or impairment. *Journal of Autism and Developmental Disorders, 38*(4), 616–625.

Bettelheim, B. (1974). *A home for the heart*. New York: Knopf.

Campbell, J. M. (2005). Diagnostic assessment of Asperger's disorder: A review of five third-party rating scales. *Journal of Autism and Developmental Disorders, 35*(1), 25–35.

Candland, D. K. (1993). *Feral children and clever animals: Reflections on human nature*. New York: Oxford University Press.

Chawarska, K., Chang, J., & Campbell, D. (2015). Clinical correlates of early general-

ized overgrowth in autism spectrum disorder: In reply. *Journal of the American Academy of Child and Adolescent Psychiatry, 54*(11), 958–959.

Chawarska, K., Klin, A., & Volkmar, F. (Eds.). (2008). *Autism spectrum disorders in infants and toddlers: Diagnosis, assessment, and treatment*. New York: Guilford Press.

Chawarska, K., Macari, S., & Shic, F. (2013). Decreased spontaneous attention to social scenes in 6-month-old infants later diagnosed with autism spectrum disorders. *Biological Psychiatry, 74*(3), 195–203.

Chawarska, K., Macari, S., Volkmar, F. R., Kim, S., & Shic, F. (2014). ASD in infants and toddlers. In F. R. Volkmar, R. Paul, S. J. Rogers, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (4th ed., Vol. 1, pp. 121–147). Hoboken, NJ: Wiley.

Chawarska, K., Ye, S., Shic, F., & Chen, L. (2016). Multilevel differences in spontaneous social attention in toddlers with autism spectrum disorder. *Child Development, 87*(2), 543–557.

Chiang, H.-M., Cheung, Y. K., Brown, A., & Li, H. (2014). A meta-analysis of differences in IQ profiles between individuals with Asperger's disorder and high-functioning autism. *Journal of Autism and Developmental Disorders, 44*(7), 1577–1596.

Cicchetti, D. V. (1994). Guidelines, criteria, and rules of thumb for evaluating normed and standardized assessment instruments in psychology. *Psychological Assessment, 6*(4), 284–290.

Constantino, J. N., & Todd, R. D. (2000). Genetic structure of reciprocal social behavior. *American Journal of Psychiatry, 157*(12), 2043–2045.

de Sanctis, S. (1906). On some variations of dementia praecox. *Revista Sperimentali di Frenciatria, 32,* 141–165.

Doehring, P., & Becker-Conttrill, B. (2013). *Autism services across America*. Baltimore, MD: Brookes.

Dworzynski, K., Ronald, A., Bolton, P., & Happé, F. (2012). How different are girls and boys above and below the diagnostic threshold for autism spectrum disorders? *Journal of the American Academy of Child and Adolescent Psychiatry, 51*(8), 788–797.

Elsabbagh, M., Divan, G., Koh, Y.-J., Kim, Y. S., Kauchali, S., Marcín, C., . . . Fombonne, E. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research, 5*(3), 160–179.

Factor, D. C., Freeman, N. L., & Kardash, A. (1989). A comparison of DSM-III and DSM-III-R criteria for autism. *Journal of Autism and Developmental Disorders, 19*(4), 637–640.

Ferster, C. B. (1972). Clinical reinforcement. *Seminars in Psychiatry, 4*(2), 101–111.

Folstein, S., & Rutter, M. (1978). Genetic influences and infantile autism. *Annual progress in child psychiatry and child development* (pp. 437–441). New York: Brunner/Mazel.

Fraser, R., Cotton, S., Gentle, E., Angus, B., Allott, K., & Thompson, A. (2012). Non-expert clinicians' detection of autistic traits among attenders of a youth mental health service. *Early Intervention in Psychiatry, 6*(1), 83–86.

Freeth, M., Milne, E., Sheppard, E., & Ramachandran, R. (2014). Autism across cultures: Perspectives from non-western cultures and implications for research. In F. R. Volkmar, R. Paul, S. J. Rogers, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders, Vol. 2* (4th ed., pp. 997–1013). Hoboken, NJ: Wiley.

Gardner, L. M., Campbell, J. M., Keisling, B., & Murphy, L. (2018). Correlates of DSM-5 autism spectrum disorder levels of support ratings in a clinical sample. *Journal of Autism and Developmental Disorders, 48*(10), 3513–3523.

Gibbs, V., Aldridge, F., Chandler, F., Witzlsperger, E., & Smith, K. (2012). An exploratory study comparing diagnostic outcomes for autism spectrum disorders under DSM-IV-TR with the proposed DSM-5 revision. *Journal of Autism and Developmental Disorders, 42*(8), 1750–1756.

Gillberg, C. (1994). Debate and argument: Having Rett syndrome in the ICD-10 PDD category does not make sense [Comment]. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 35*(2), 377–378.

Goldstein, S., Naglieri, J. A., & Ozonoff, S. (Eds.). (2009). *Assessment of autism spectrum disorders*. New York: Guilford Press.

Gould, S. J. (1996). *The mismeasure of man*. New York: Norton.

Greenberg, G. (2013). *The book of woe: The DSM and the unmaking of psychiatry*. New York, Penguin.

Guthrie, W., Swineford, L. B., Wetherby, A. M., & Lord, C. (2013). Comparison of DSM-IV and DSM-5 factor structure models for toddlers with autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 52*(8), 797–805.

Hansen, R. L., Ozonoff, S., Krakowiak, P., Angkustsiri, K., Jones, C., Deprey, L. J., . . . Hertz-Picciotto, I. (2008). Regression in autism: Prevalence and associated factors in the CHARGE Study. *Ambulatory Pediatrics, 8*(1), 25–31.

Harris, J. C. (2006). *Intellectual disability: Understanding its development, causes, classification, evaluation, and treatment*. New York: Oxford University Press.

Hawks, Z. W., & Constantino, J. N. (2020). Neuropsychiatric "comorbidity" as causal influence in autism. *Journal of the American Academy of Child and Adolescent Psychiatry, 59*(2), 229–235.

Heller, T. (1908). Dementia infantilis. *Zeitschrift fur die erforschung und behandlung des jugenlichen schwachsinns, 2,* 141–165.

Hertzig, M. E., Snow, M. E., New, E., & Shapiro, T. (1990). DSM-III and DSM-III-R diagnosis of autism and pervasive developmental disorder in nursery school children. *Journal of the American Academy of Child and Adolescent Psychiatry, 29*(1), 123–126.

Hobbs, N. (1975). *Issues in the classification of children*. San Francisco: Jossey-Bass.

Huerta, M., Bishop, S. L., Duncan, A., Hus, V., & Lord, C. (2013). Response to Ritvo and Ritvo letter: Commentary on the application of DSM-5 criteria for autism spectrum disorder. *American Journal of Psychiatry, 170*(4), 445–446.

Hunt, J. M. (1961). *Intelligence and experience*. New York: Ronald Press.

Ibañez, L. V., Stone, W. L., & Coonrod, E. E. (2014). Screening for autism in young children. In F. R. Vollkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (4th ed., Vol. 2, pp. 585–608). Hoboken, NJ: Wiley.

Ingersoll, B., & Wainer, A. (2014). The broader autism phenotype. In F. R. Vollkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (4th ed., Vol. 1, pp. 28–56). Hoboken, NJ: Wiley.

Jackson, S. L., & Volkmar, F. R. (2019). Diagnosis and definition of autism and other pervasive developmental disorders. In F. Volkmar (Ed.), *Autism and pervasive developmental disorders* (3rd ed., pp. 1–24). Cambridge, UK: Cambridge University Press.

Jones, L. A., & Campbell, J. M. (2010). Clinical characteristics associated with language regression for children with autism spectrum disorders. *Journal of Autism and Developmental Disorders, 40*(1), 54–62.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child, 2*, 217–250.

Kanner, L. (1971). Follow-up study of eleven autistic children originally reported in 1943. *Journal of Autism and Childhood Schizophrenia, 1*(2), 119–145.

Kim, S. H., Macari, S., Koller, J., & Chawarska, K. (2016). Examining the phenotypic heterogeneity of early autism spectrum disorder: Subtypes and short-term outcomes. *Journal of Child Psychology and Psychiatry, 57*(1), 93–102.

King, B. H., Veenstra-VanderWeele, J., & Lord, C. (2013). DSM-5 and autism: Kicking the tires and making the grade. *Journal of the American Academy of Child and Adolescent Psychiatry, 52*(5), 454–457.

Klin, A., Jones, W., Schultz, R., & Volkmar, F. R. (2005). The enactive mind—from actions to cognition: Lessons from autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders: Diagnosis, development, neurobiology, and behavior* (3rd ed., Vol. 1, pp. 682–703). Hoboken, NJ: Wiley.

Klin, A., Saulnier, C., Tsatsanis, K., & Volkmar, F. R. (2005). Clinical evaluation in autism spectrum disorders: Psychological assessment within a transdisciplinary framework. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., Vol. 2, pp. 772–798). Hoboken, NJ: Wiley.

Kolvin, I. (1971). Studies in childhood psychoses: I. Diagnostic criteria and classification. *British Journal of Psychiatry, 118*, 381–384.

Kurita, H., Koyama, T., Setoya, Y., Shimizu, K., & Osada H. (2004). Validity of childhood disintegrative disorder apart from autistic disorder with speech loss. *European Child and Adolescent Psychiatry, 13*(4), 221–226.

Lord, C. (1996). Follow-up of two-year-olds referred for possible autism. *Journal of Child Psychology and Psychiatry, 36*(8), 1065–1076.

Lord, C., Corsello, C., & Grzadzinski, R. (2014). Diagnostic instruments in autistic spectrum disorders. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and developmental disorders* (pp. 610–650). Hoboken, NJ: Wiley.

Lord, C., & Gotham, K. (2014). DSM-5 and ASD: Reflections and commentary. In T. E. Davis, III, S. W. White, & T. H. Ollendick (Eds.), *Handbook of autism and anxiety* (pp. 247–261). Cham, Switzerland: Springer International.

Lord, C., Petkova, E., Hus, V., Gan, W., Lu, F., Martin, D. M., . . . Risi, R. (2012). A multisite study of the clinical diagnosis of different autism spectrum disorders. *Archives of General Psychiatry, 69*(3), 306–313.

Lovass, O. I., & Smith, T. (1988). Intensive behavioral treatment for young autistic children. In B. B. Lahey & A. E. Kazdin (Eds.), *Advances in clinical child psychology* (Vol. 11, pp. 285–324). New York: Plenum Press.

Luyster, R., Richler, J., Risi, S., Hsu, W.-L., Dawson, G., Bernier, R, . . . Lord, C. (2005). Early regression in social communication in autism spectrum disorders: A CPEA study. *Developmental Neuropsychology, 27*(3), 311–336.

Matson, J. L., Beighley, J., & Turygin, N. (2012a). Autism diagnosis and screening: Factors to consider in differential diagnosis. *Research in Autism Spectrum Disorders, 6*(1), 19–24.

Matson, J. L., Hattier, M. A., & Williams, L. W. (2012b). How does relaxing the algorithm for autism affect DSM-V prevalence rates? *Journal of Autism and Developmental Disorders, 42*(8), 1549–1556.

Mattila, M. L., Kielinen, M., Linna, S. L., Jussila, K., Ebeling, H., Bloigu, R., . . . Moilanen, I. (2011). Autism spectrum disorders according to DSM-IV-TR and comparison with DSM-5 draft criteria: An epidemiological study. *Journal of the American Academy of Child and Adolescent Psychiatry, 50*(6), 583–592.

Maudsley, H. (1867). *The physiology and pathology of mind*. New York: Appleton & Co.

Mayes, S. D., Calhoun, S. L., & Crites, D. L. (2001). Does DSM-IV Asperger's disorder exist? *Journal of Abnormal Child Psychology, 29*(3), 263–271.

Mazefsky, C., McPartland, J., Gastgeb, H., & Minshew, N. (2013). Brief report: Comparability of DSM-IV and DSM-5 ASD research samples. *Journal of Autism and Developmental Disorders, 43*(5), 1236–1242.

McPartland, J., Dawson, G., Webb, S. J., Panagiotides, H., & Carver, L. J. (2004). Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. *Journal of Child Psychology and Psychiatry, 45*(7), 1235–1245.

McPartland, J. C., & Pelphrey, K. A. (2012). The implications of social neuroscience for social disability. *Journal of Autism and Developmental Disorders, 42*(6), 1256–1262.

McPartland, J. C., Reichow, B., & Volkmar, F. R. (2012). Sensitivity and specificity of proposed DSM-5 diagnostic criteria for autism spectrum disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 51*(4), 368–383.

Miot, S., Akbaraly, T., Michelon, C., Couderc, S., Crepiat, S., Loubersac, J., . . . Baghdadli, A. (2019). Comorbidity burden in adults with autism spectrum disorders and intellectual disabilities—a report from the EFAAR (Frailty Assessment in Aging Adults with Autism Spectrum and Intellectual Disabilities) study. *Frontiers in Psychiatry Frontiers Research Foundation, 10,* 617.

Moretti, P., & Zoghbi, H. Y. (2006). MeCP2 dysfunction in Rett syndrome and related disorders. *Current Opinion in Genetics and Development, 16*(3), 276–281.

National Society for Autistic Children. (1978). Definition of the syndrome of autism. *Journal of Autism and Childhood Schizophrenia, 8*(2), 162–169.

Nergård, J. I. (2006). *Den levende erfaring: En studie i samisk kunnskapstradisjon*. Oslo: Cappelen Akademisk.

Øien, R. A., & Nordahl-Hansen, A. (2018). Bias in assessment instruments for autism. In *Encyclopedia of autism spectrum disorders* (Vol. 51, pp. 1–2). New York: Springer.

Øien, R. A., Schjolberg, S., Volkmar, F. R., Shic, F., Cicchetti, D. V., Nordahl-Hansen, A., . . . Chawarska, K. (2018a). Clinical features of children with autism who passed 18-month screening. *Pediatrics, 141*(6), 6.

Øien, R. A., Vambheim, S. M., Hart, L., Nordahl-Hansen, A., Erickson, C., Wink, L., . . . Grodberg, D. (2018b). Sex-differences in children referred for assessment: An exploratory analysis of the Autism Mental Status Exam (AMSE). *Journal of Autism and Developmental Disorders, 48*(7), 2286–2292.

Ozonoff, S., & Griffith, E. M. (2000). Neuropsychological function and the external validity of Asperger syndrome. In A. Klin, F. R. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome* (pp. 72–96). New York: Guilford Press.

Ozonoff, S., Iosif, A.-M., Baguio, F., Cook, I. C., Hill, M. M., Hutman, T., . . . Young, G. S. (2010). A prospective study of the emergence of early behavioral signs of

autism. *Journal of the American Academy of Child and Adolescent Psychiatry, 49*(3), 256–266.

Ozonoff, S., Young, G. S., Landa, R. J., Brian, J., Bryson, S., Charman, T., . . . Iosif, A.-M. (2015). Diagnostic stability in young children at risk for autism spectrum disorder: A Baby Siblings Research Consortium study. *Journal of Child Psychology and Psychiatry, 56*(9), 988–998.

Piven, J. (2001). The broad autism phenotype: A complementary strategy for molecular genetic studies of autism. *American Journal of Medical Genetics, 105*(1), 34–35.

Prizant, B. M., & Rydell, P. J. (1984). Analysis of functions of delayed echolalia in autistic children. *Journal of Speech and Hearing Research, 27*(2), 183–192.

Rank, B. (1949). Adaptation of the psychoanalytic technique for the treatment of young children with atypical development. *American Journal of Orthopsychiatry 19,* 130–139.

Rank, B., & MacNaughton, D. (1949). A clinical contribution to early ego development. In A. Freud & H. Hartmann (Eds.), *The psychoanalytic study of the child* (Vol. 3, pp. 53–65). Oxford, UK: International Universities Press.

Regier, D. A., Narrow, W. E., Clarke, D. E., Kraemer, H. C., Kuramoto, S. J., Kuhl, E. A., & Kupfer, D. J. (2012). DSM-5 field trials in the United States and Canada: Part II. Test-retest reliability of selected categorical diagnoses. *American Journal of Psychiatry, 170*(1), 59–70.

Reiss, S., Levitan, G., & Szyszko, J. (1982). Emotional disturbance and mental retardation: Diagnostic overshadowing. *America Journal of Mental Deficiency, 86,* 567–574.

Rett, A. (1966). Uber ein eigenartiges hirntophisces Syndroem bei hyperammonie im Kindersalter. *Wein Medizinische Wochenschrift, 118,* 723–726.

Rett, A. (1986). Rett syndrome: History and general overview. *American Journal of Medical Genetics, 1986*(Suppl 1), 21–25.

Rimland, B. (1964). *Infantile autism: The syndrome and its implications for a neural theory of behavior.* New York: Appleton-Century-Crofts.

Rimland, B. (1971). The differentiation of childhood psychoses: An analysis of checklists for 2,218 psychotic children. *Journal of Autism and Childhood Schizophrenia, 1*(2), 161–174.

Rogers, S. J. (2004). Developmental regression in autism spectrum disorders. *Mental Retardation and Developmental Disabilities Research Reviews, 10*(2), 139–143.

Rowberry, J., Macari, S., Chen, G., Campbell, D., Leventhal, J. M., Weitzman, C., & Chawarska, K. (2015). Screening for autism spectrum disorders in 12-month-old high-risk siblings by parental report. *Journal of Autism and Developmental Disorders, 45*(1), 221–229.

Rutter, M. (1972). Childhood schizophrenia reconsidered. *Journal of Autism and Childhood Schizophrenia, 2*(4), 315–337.

Rutter, M. (1978). Diagnosis and definitions of childhood autism. *Journal of Autism and Developmental Disorders, 8*(2), 139–161.

Rutter, M. (1991). Isle of Wight revisited: Twenty-five years of child psychiatric epidemiology. In S. Chess & M. E. Hertzig (Eds.), *Annual progress in child psychiatry and child development, 1990* (pp. 131–179). Philadelphia: Brunner/Mazel.

Rutter, M. (1994). Debate and argument: There are connections between brain and mind and it is important that Rett syndrome be classified somewhere [comment]. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 35*(2), 379–381.

Rutter, M. (1997). Comorbidity: Concepts, claims and choices. *Criminal Behaviour and Mental Health, 7*(4), 265–285.

Rutter, M. (2011). Research review: Child psychiatric diagnosis and classification: Concepts, findings, challenges and potential. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 52*(6), 647–660.

Rutter, M., & Bartak, L. (1973). Special educational treatment of autistic children: A comparative study: II. Follow-up findings and implications for services. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 14*(4), 241–270.

Rutter, M., & Garmezy, N. M. (1983). Developmental psychopathology. In E. M. Hetherington (Ed.), *Mussen's handbook of child psychology: Socialization, personality and child development* (Vol. 4, pp. 755–911). New York: Wiley.

Rutter, M., Lebovici, S., Eisenberg, L., Sneznevskij, A. V., Sadoun, R., Brooke, E., & Lin, T. Y. (1969). A tri-axial classification of mental disorders in childhood: An international study. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 10*(1), 41–61.

Rutter, M., & Schopler, E. (1992). Classification of pervasive developmental disorders: Some concepts and practical considerations [comments]. *Journal of Autism and Developmental Disorders, 22*(4), 459–482.

Rutter, M., & Thapar, A. (2014). Genetics of autism spectrum disorders. In F. R. Volkmar, R. Paul, S. J. Rogers, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (4th ed., pp. 411–423). Hoboken, NJ: Wiley.

Schopler, E., Van Bourgondien, M. E., Wellman, G. J., & Love, S. R. (2010). *Childhood Autism Rating Scale, Second edition (CARS2).* Torrance, CA: Western Psychological Services.

Sharma, S., Woolfson, L. M., & Hunter, S. C. (2012). Confusion and inconsistency in diagnosis of Asperger syndrome: A review of studies from 1981 to 2010. *Autism, 16*(5), 465–486.

Siegel, B., Vukicevic, J., Elliott, G. R., & Kraemer, H. C. (1989). The use of signal detection theory to assess DSM-III-R criteria for autistic disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 28*(4), 542–548.

Sipes, M., & Matson, J. L. (2014). Factor structure for autism spectrum disorders with toddlers using DSM-IV and DSM-5 criteria. *Journal of Autism and Developmental Disorders, 44*(3), 636–647.

Smith, I. C., Reichow, B., & Volkmar, F. R. (2015). The effects of DSM-5 criteria on number of individuals diagnosed with autism spectrum disorder: A systematic review. *Journal of Autism and Developmental Disorders, 45*(8), 2541–2552.

Spiker, M. A., Lin, C., Van Dyke, M., & Wood, J. J. (2012). Restricted interests and anxiety in children with autism. *Autism, 16*(3), 306–320.

Spitzer, R. L., Endicott, J. E., & Robbins, E. (1978). Resarch diagnostic criteria. *Archives of General Psychiatry, 35,* 773–782.

Stewart, M. E., Barnard, L., Pearson, J., Hasan, R., & O'Brien, G. (2006). Presentation of depression in autism and Asperger syndrome: A review. *Autism, 10*(1), 103–116.

Szatmari, P. (1991). Asperger's syndrome: Diagnosis, treatment, and outcome. *Psychiatric Clinics of North America, 14*(1), 81–93.

Taheri, A., & Perry, A. (2012). Exploring the proposed DSM-5 criteria in a clinical sample. *Journal of Autism and Developmental Disorders, 42*(9), 1810–1817.

Tsai, L. (1992). Is Rett syndrome a subtype of pervasive developmental disorder? *Journal of Autism and Developmental Disorders, 22,* 551–561.

Tsai, L. Y. (2012). Sensitivity and specificity: DSM-IV versus DSM-5 criteria for autism spectrum disorder. *American Journal of Psychiatry, 169*(10), 1009–1011.

Vanegas, S. B., Magaña, S., Morales, M., & McNamara, E. (2016). Clinical validity of the ADI-R in a U.S.-based Latino population. *Journal of Autism and Developmental Disorders, 46*(5), 1623–1635.

Volkmar, F. R. (1992). Childhood disintegrative disorder: Issues for DSM-IV. *Journal of Autism and Developmental Disorders, 22*(4), 625–642.

Volkmar, F. R., Booth, L. L., McPartland, J. C., & Wiesner, L. A. (2014a). Clinical evaluation in multidisciplinary settings. In F. R. Volkmar, S. J. Rogers, R. Paul, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders: Assessment, interventions, and policy* (4th ed., Vol. 2, pp. 661–672). Hoboken, NJ: Wiley.

Volkmar, F. R., Cicchetti, D. V., Bregman, J., & Cohen, D. J. (1992a). Three diagnostic systems for autism: DSM-III, DSM-III-R, and ICD-10. Special Issue: Classification and diagnosis. *Journal of Autism and Developmental Disorders, 22*(4), 483–492.

Volkmar, F. R., Cicchetti, D. V., Cohen, D. J., & Bregman, J. (1992b). Brief report: Developmental aspects of DSM-III-R criteria for autism. *Journal of Autism and Developmental Disorders, 22*(4), 657–662.

Volkmar, F. R., Cohen, D. J., & Paul, R. (1986). An evaluation of DSM-III criteria for infantile autism. *Journal of the American Academy of Child Psychiatry, 25*(2), 190–197.

Volkmar, F. R., Klin, A., & McPartland, J. C. (2014b). Asperger syndrome: An overview. In J. C. McPartland, A. Klin, & F. R. Volkmar (Eds.), *Asperger syndrome: Assessing and treating high-functioning autism spectrum disorders* (2nd ed., pp. 1–42). New York: Guilford Press.

Volkmar, F. R., Klin, A., Siegel, B., Szatmari, P., Lord, C., Campbell, M., . . . Towbin, K. (1994). Field trial for autistic disorder in DSM-IV. *American Journal of Psychiatry, 151*(9), 1361–1367.

Volkmar, F. R., & Nelson, D. S. (1990). Seizure disorders in autism. *Journal of the American Academy of Child and Adolescent Psychiatry, 29*(1), 127–129.

Volkmar, F. R., & Rutter, M. (1995). Childhood disintegrative disorder: Results of the DSM-IV autism field trial. *Journal of the American Academy of Child and Adolescent Psychiatry, 34*(8), 1092–1095.

Volkmar, F. R., Sparrow, S. S., Goudreau, D., Cicchetti, D. V., Paul, R., & Cohen, D. J. (1987). Social deficits in autism: An operational approach using the Vineland Adaptive Behavior Scales. *Journal of the American Academy of Child and Adolescent Psychiatry, 26*(2), 156–161.

Volkmar, F. R., Sukhodolsky, D., Schwab-Stone, M., & First, M. B. (2017). Diagnostic classification. In A. Martin, M. Bloch, & F. R. Volkmar (Eds.), *Lewis's child and adolescent psychiatry: A comprehensive textbook* (pp. 354–363). Philadelphia: Wolters-Kluwer.

Volkmar, F. R., & Tsatsanis, K. (2002). Psychosis and psychotic conditions in childhood and adolescence. In D. T. Marsh & M. A. Fristad (Eds.), *Handbook of serious emotional disturbance in children and adolescents* (pp. 266–283). New York: Wiley.

Wainer, A. L., Block, N., Donnellan, M. B., & Ingersoll, B. (2013). The broader autism phenotype and friendships in non-clinical dyads. *Journal of Autism and Developmental Disorders, 43*(10), 2418–2425.

Waterhouse, L., Wing, L., Spitzer, R. L., & Siegel, B. (1993). Diagnosis by DSM-III-R versus ICD-10 criteria. *Journal of Autism and Developmental Disorders, 23*(3), 572–573.

White, S. W., Bray, B. C., & Ollendick, T. H. (2012). Examining shared and unique aspects of social anxiety disorder and autism spectrum disorder using factor analysis. *Journal of Autism and Developmental Disorders, 42*(5), 874–884.

Wilson, C., Gillan, N., Spain, D., Robertson, D., Roberts, G., Murphy, C. M., . . . Murphy, D. G. (2013). Comparison of ICD-10R, DSM-IV-TR and DSM-5 in an adult autism spectrum disorder diagnostic clinic. *Journal of Autism and Developmental Disorders, 43*(11), 2515–2525.

Wing, L. (1980). Childhood autism and social class: A question of selection? *British Journal of Psychiatry 137*, 410–417.

Wing, L., & Gould, J. (1979). Severe impairments of social interaction and associated abnormalities in children: Epidemiology and classification. *Journal of Autism and Developmental Disorders, 9*(1), 11–29.

Wolff, S. (2004). The history of autism. *European Child and Adolescent Psychiatry, 13*(4), 201–208.

World Health Organization. (1994). *International classification of diseases* (10th ed.). Geneva, Switzerland: Author.

Worley, J. A., & Matson, J. L. (2012). Comparing symptoms of autism spectrum disorders using the current DSM-IV-TR diagnostic criteria and the proposed DSM-V diagnostic criteria. *Research in Autism Spectrum Disorders, 6*(2), 965–970.

Zwaigenbaum, L., Bauman, M. L., Stone, W. L., Yirmiya, N., Estes, A., Hansen, R. L., . . . Wetherby, A. (2015). Early identification of autism spectrum disorder: Recommendations for practice and research. *Pediatrics, 136*(4, Suppl. 1), S10–S40.