Autism and Child Psychopathology Series
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Johnny L. Matson
Peter Sturmey
Editors

International Handbook of

Autism and Pervasive Developmental Disorders

Springer

Autism and Child Psychopathology Series

Series Editor

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Editors

International Handbook of Autism and Pervasive Developmental Disorders

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Part I
Overview

History and Evolution of the Autism Spectrum Disorders

Julie K. Irwin, Jennifer MacSween, and Kimberly A. Kerns

Autistic spectrum disorders (ASDs) are characterized by impaired social interactions and communication, as well as restricted and repetitive behaviors and interests. Impairment in each of these dimensions can vary in severity, and symptomatologies among individuals with ASDs are often quite diverse. Over the past 20 years, there has been a marked increase in the diagnosis of individuals with ASDs. In 1966, Lotter undertook the first epidemiological study of autism, estimating the prevalence of autism disorder to be 4.5/10,000. Two decades later, the estimate rose to 10.1/10,000 (Bryson, Clark, & Smith, 1988). Currently, the community prevalence of ASDs is estimated to be *at least* 36.4/10,000 (Fombonne, 2005b), with some estimates as high as 67/10,000 (Bertrand et al., 2001). It has been suggested that this increase in the number of cases of ASDs is due, at least in part, to more inclusionary definitions of disorders within the autism spectrum (Fombonne, 2005a). While more in-depth discussion of this increase in ASD prevalence can be found in Chapter 3 of this volume, there is at least some suggestion that changes in prevalence may be due to changes in, and broadening of, the diagnostic conceptualization of ASD. Indeed over the 60 years that autism has been recognized, there have been many changes in our understanding of this disorder. The historical changes in the conception of autism and other pervasive developmental disorders are outlined below.

In 1943, Dr Leo Kanner, an Austrian child psychiatrist at John Hopkins University, published the paper "Autistic Disturbances of Affective Contact," where he described the behavior of 11 children. Observing one of the children, Kanner remarked "He does not observe the fact that anyone comes or goes, and never seems glad to see father or mother or any playmate… he seems almost to draw into his shell and live within himself" (1943, p. 218). Prior to Kanner's case studies, children with similar behaviors were likely seen as being either mentally retarded or insane. Indeed, since Kanner (1943) first published case studies of these children whom he labeled "autistic," there have been several re-interpretations of historical accounts of "odd," emotionally disturbed, or mentally retarded children, who may, in fact, have been autistic. For example, Uta Frith (1989) has suggested that Victor, the "wild boy of Aveyron," showed signs of autism, including abnormal nonverbal expression and impaired attachment to his caregivers (based on the writings of Bonnaterre, 1800). Similarly, it may be that the children Henry Maudsley (1867) labeled as having "instinctive insanity" had autism. However, it was not until Kanner that an attempt to classify autistic children was made, with explicit criteria with which to identify them.

Kanner's detailed description of the 11 children in his landmark paper asserted that these children had a condition that differed "markedly and uniquely from anything reported so far" (p. 217). He borrowed the term "autism" from Bleuler (1911), a Swiss psychiatrist, who also coined the word "schizophrenia." From the Greek *autos* (self) and *ismos* (a suffix of state or action), "autistic" was used to describe the autocentric thinking and apparent withdrawal from the social world by children with schizophrenia. It was also a defining trait of the children Kanner studied and gradually the adjective, "autistic," led to the noun, "autism."

Kanner (1943) postulated that the fundamental disorder in children with autism was an "inability to relate themselves in the ordinary way to people and situations from the beginning of life" (p. 242). They were characterized by the following: (i) an "autistic aloneness"; (ii) a failure to use language to effectively communicate (e.g. mutism, echolalia, and overly literal language); (iii) repetitive, constricted activity and an "anxiously obsessive desire for the maintenance of sameness" (p. 245); and (iv) "good cognitive potential" shown in "excellent rote memory" (p. 243) and on performance tests. Indeed many of these distinctive and unusual behaviors described by Kanner remain cardinal to our definition and understanding of the disorder.

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After publishing this first account, Kanner continued to work with autistic children, observing up to 120 further cases at Johns Hopkins Hospital. In 1956, he and Leon Eisenberg co-authored a paper revising what had come to be called early infantile autism. The authors proposed that the two defining features of autism were "extreme self-isolation" and an "obsessive insistence on sameness," believing autism could be reliably identified by these two characteristics alone. The non-communicative use of language, or its delay or failure to develop in autistic children, was seen as a direct result of a lack of relatedness with other people. As such, language impairment was originally seen as neither a core characteristic nor a reliable indicator of autism. Michael Rutter (1978) would later argue that language and prelanguage impairments are essential deficits of autism, asserting that this should be included as a diagnostic criterion along with the impairments in social relationships and insistence on sameness suggested by Eisenberg and Kanner.

In his earlier paper, Kanner (1943) also noted that the parents of the children he studied were highly intelligent, cold, and tended to be uninterested in people. However, since autistic aloneness was present from early infancy, he concluded that children with autism were born with no innate ability to form emotional connections with people. Later, Kanner (1949) assumed a much greater role of what was to be called the non-emotional "refrigerator mother" in the withdrawal of autistic children, a notion that fit well within the psychoanalytic zeitgeist current at the time. Indeed, while Kanner and Eisenberg (1956) acknowledged that outright rejection, neglect, or abuse of autistic children by their parents was rare, they viewed children with autism as experiencing "emotional refrigeration" by cold, unaffectionate, and mechanical parents and wrote that they could not dismiss the home environment as playing a role in the "genesis" of the disorder. However, since both still believed autism to be present from birth or very near to it, they also recognized that emotionally distant parenting alone was not sufficient to produce the disorder. Instead, they suggested that autism was the result of biological, psychological, and social factors (1956). Nonetheless, in an interview, Kanner (as cited in Thomas, 1960) still suggested that "refrigerator mothers" were a factor in the etiology of autism and proclaimed that parents of children with autism were so cold that they "just happen[ed] to defrost long enough to produce a child." Ironically, Kanner (1941) had earlier written a book called "In Defense of Mothers," subtitled, "How to Bring Up Children in Spite of the More Zealous Psychologists."

It was, however, Bettelheim's work that served to widely disseminate the "refrigerator mother" theory of the etiology of autism in the medical field, and even popularized it in the public sphere. Bettelheim, an Austrian-born American child psychologist, was the most well-known proponent of the view that autism was due to unemotional or unavailable mothers. Bettelheim's "Feral Children and Autistic Children" (1959) outlines the cases of Kamala and Amala, the "wolf girls" of Midnapore, India. The girls, aged eight and one and a half years old when found, were thought to have been raised by a she-wolf, demonstrating many "wolf-like behaviors." As has been done retrospectively with other feral children, Bettelheim speculated that these girls had actually been severely autistic. He argued that all parents of autistic children viewed these children as "unacceptable" and "wished to be rid of them." He reasoned that the girls had probably been emotionally, if not physically, abandoned. Bettelheim believed that being in the company of a wolf had little to do with Kamala and Amala's "animal" behavior and outlined cases of autistic children whose behavior paralleled that of these feral children in the absence of physical abandonment. Bettelheim asserted that both types of children displayed the same behaviors because they had both been subjected to extreme emotional isolation in combination with experiences interpreted as threatening to the self, stating "feral children seem to be produced not when wolves behave like mothers but when mothers behave like non-humans" (1959, p. 467).

Bettelheim's own life experiences deeply influenced his theories about autism. He had been detained in Nazi concentration camps, afterward writing about the loss of humanity he had witnessed in response to extreme situations (1943). In *The Empty Fortress: Infantile Autism and the Birth of the Self* (1967), Bettelheim likened the plight of autistic children to that of prisoners in German concentration camps. Autism, he contended, was a state of mind developed in reaction to "extreme situations," created by maternal rejection and hostility. Such situations left children feeling hopeless, anxious, apathetic, and increasingly unable to communicate with the outside world. In response, they withdrew from reality into themselves.

A serious challenge to this psychoanalytic view of autism, as purported by Bettelheim, came with Rimland's book, *Infantile Autism: The Syndrome and Its Implications for a Neural Theory of Behavior* (1964). The parent of an autistic son himself, Rimland argued convincingly that there was no evidence to support a psychogenic theory of autism. Instead, he believed there was a neurological basis for the disorder. In contrast to the unsubstantiated psychoanalytic theories, Rimland's arguments were based on empirical research and strongly influenced the field of autism research.

In Kanner's original description of autistic children (1943), he differentiated autism from schizophrenia, especially since some children with autism had been previously diagnosed with the latter disorder. He recognized that children with schizophrenia experienced some normal development before their behavior changed. Importantly, they withdrew from *previously existing* social relationships, while autistic children, he thought, failed from birth to ever

form these social relationships. He viewed children with schizophrenia as trying to solve their problems by withdrawing from a world with which they had been engaged. Conversely, children with autism would cautiously begin to engage *more* with that world as they grew older.

Early researchers such as Bender (1947) and Kanner (1949; influenced by Despert) argued that autism was likely an early form of adult schizophrenia and some used "childhood schizophrenia" instead of "early infantile autism" to reflect the belief that these children were displaying the earliest expression of the adult disorder. Indeed, Creak (1961) described the results of a "working party" whose aims were to "discuss the present confused terminology and perhaps reach some clearer definition" (p. 501) for the number of children presenting with symptomatology variously classified as "autistic," "schizophrenic," and "atypical." Due to these overlapping descriptions, variations of the terms "autism," "childhood psychosis," and "schizophrenia" were used interchangeably by many clinicians and researchers at the time. Given the initial use of "autistic" as a descriptor of social withdrawal in individuals with schizophrenia, it is perhaps unsurprising that autism and schizophrenia remained linked in the literature until the 1970s. Indeed, Creak (1961) described nine criteria for "schizophrenic syndrome in childhood," which was the working party's agreed upon term for labeling "childhood psychosis," and represented his belief that these disorders were a childhood form of the later adult disorder. Interestingly, there is a high degree of overlap in these nine criteria and current diagnostic criteria for autistic disorder.

Kolvin and colleagues (Kolvin, 1971; Kolvin, Ounsted, Humphrey, & McNay, 1971) provided clear evidence that age of onset could be used to distinguish between autism and childhood schizophrenia. To reinforce this point, Rutter (1972) used the term *schizophrenic psychosis in childhood* to denote the child-onset type of schizophrenia as it is known today. He contrasted this with early infantile autism with an onset prior to age 3. Rutter (1968) also argued that autism could, in fact, be differentiated from schizophrenia, citing a number of factors such as the higher male-to-female incidence, the absence of delusions or hallucinations, and the overall stable course of autism in comparison to schizophrenia.

During the 1970s, those who researched autism's etiology focused increasingly on biological factors, including genetic ones. Examinations of gross chromosomal abnormalities (Böök, Nichtern, & Gruenberg, 1963), twin concordance rates (Rutter, 1967), and family studies (Lowe, 1966) were conducted during the 1960s. These failed to uncover a clear genetic basis of autism, though Folstein and Rutter (1977), in a study of 21 twins with autism, suggested that the inheritance of a cognitive abnormality, with or without perinatal brain damage, may lead to autism. This was arguably the strongest evidence at the time of a genetic basis for the disorder, though Spence (1976) had already concluded that inheritance of autism was likely to be polygenic or multifactorial, and thus would be difficult to ascertain.

As evidence for biological explanations of autism mounted, psychogenic theories of the disorder lost credibility. Many alternative mechanisms and causes of autistic features were also proposed including, but not limited to, seizures (Schain & Yannet, 1960; Rutter & Lockyer, 1967), congenital defects of the reticular formation leading to hyperoxia (Rimland, 1964), very early developmental insults in utero (Walker, 1977), prenatal and perinatal complications (Lobascher, Kingerlee, & Gubbay, 1970; Taft & Goldfarb, 1964), and organic brain abnormality (Rutter, 1967). Most researchers agreed that they were searching for multiple and interacting causal factors of the disorder (Rutter, 1968), consistent with current biopsychosocial theories for the majority of mental disorders.

While Kanner is credited with first documenting children with autistic features, around the same time that he published his first account of autistic children, a German psychiatrist, Asperger, published a paper describing children he had been observing with similar characteristics (1944). Coincidentally, Asperger also used the label "autistic" for these children, calling their disorder "autistic psychopathy." The use of "psychopathy" was meant in the sense of an abnormality of personality. In choosing Bleuler's term "autism," Asperger, like Kanner, drew parallels between the behavior of the children he observed and that of adult patients with schizophrenia. Again he noted that those with schizophrenia and those with autistic psychopathy shared common features; they had all shut off relations between the self and the outside world to some degree. However, he did not think children with autistic psychopathy were psychotic or showed the "disintegration of personality" apparent in schizophrenia; their symptoms had not developed but had been present from birth (Asperger, 1944, as translated by Frith, 1991).

In addition to this broad characterization, Asperger (1944) described the children not only as (1) poor in social and emotional relationships and (2) showing a narrow, limited, and intense focus on special interests but also as (3) often extraordinary in some cognitive domain (e.g. mathematics); (4) having no obvious delay in language acquisition; (5) sometimes possessing excellent linguistic skills but with some abnormalities of speech and nonverbal communication; and (6) clumsy and uncoordinated (as noted in Frith, 1991). Asperger noticed that parents of many of the children he observed had similar personality characteristics and so thought it likely the traits were constitutional.

Asperger's work, originally published in German, did not come to be widely recognized in English-speaking countries until the 1980s; nearly four decades after Kanner first published, Asperger's original paper was translated into English

by Frith (1991). Wing (1981), however, wrote about his work earlier than this, though she chose the more neutral term "Asperger's syndrome" (AS) as "psychopathy" was popularly associated with sociopathy. After presenting a summary of Asperger's original findings, Wing noted certain additional features she had observed in children with AS, namely that young infants with the disorder tended not to enjoy human interaction and made fewer social overtures (babbling, smiling, showing, and engaging in joint attention). These infants also showed a lack of imaginative pretend play; if it was present, it tended to be limited and repetitive.

From Wing's observations of these children, she disagreed with Asperger on two major points. First, Asperger (1944) thought that speech consistently developed before walking in his sample and wrote that many of the children he observed were very strong linguistically. In contrast, Wing (1981) found inconsistencies in whether these milestones were met on time. Upon careful analysis of the speech of children with AS, she found several peculiarities within the content, such as the inappropriate use of words, which often appeared to have been copied verbatim from a source. Second, though Asperger contended that many of those with AS were of high intelligence, he did not test this formally. Wing reasoned it was probable that the appearance of high intelligence was based mostly on excellent rote memory with little comprehension underlying this "knowledge" and noted that those with Asperger's syndrome showed a marked lack of common sense. Wing (1981) concluded by suggesting that those with AS could be identified by the presence of autistic features in combination with grammatical speech and less impaired social functioning.

In 1979, Wing and Gould published their findings from a large epidemiological study of children with any known impairments living in an area outside London, UK. They identified children under age 15 who had any features which were frequently associated with autism. Analysis from this study suggested a "triad of impairments" with deficits in social communication, verbal and nonverbal communication, and imagination (a lack of symbolic play) often found together. These impairments tended to be combined with narrow and repetitive routines and/or interests. In clustering children together by symptoms and the degree to which they displayed them, Wing and Gould (1979) suggested that these sub-groups did not represent distinct categories of disorder but rather a "continuum" of similar symptoms varying in severity. This, along with Wing's (1981) paper describing "Asperger's syndrome," was the first formal suggestion that autism was not a discrete, categorical disorder but represented a continuum of impairments and competencies. A review of the literature revealed that the first published mention of the "autism spectrum disorders" (ASDs) followed shortly (Tsai, Stewart, & August, 1981). Thereafter, the concept of an autistic spectrum took on vigor in the scientific literature. Rutter (2005) notes that even in the 1960s, clinicians were aware of "children who showed disorders that were closely comparable to autism but did not quite meet the prevailing diagnostic criteria" (p. 4). Indeed, he contends that the broadening of the diagnostic concept of autism was brought about by four different causes: (1) epidemiological evidence has highlighted that there is a high frequency of autistic-like problems in children with severe or profound intellectual disability and that intellectual disability does not preclude autism; (2) there is a sizeable proportion of children with autism who were shown to have a diagnosable somatic disease or disorder that previously would have excluded the diagnosis of autism; (3) twin and family studies showing the high frequency of autistic-like features in relatives of individuals with autism leading to a "broader phenotype" of autism; and (4) the recognition of features of Asperger syndrome.

While Kanner's original description of autism was made in 1943, autism was not listed as a disorder in the first Diagnostic and Statistical Manual of Mental Disorders (DSM-I) published in 1952 (American Psychological Association). Children with autistic symptoms instead would have been diagnosed as exhibiting "schizophrenic reaction, childhood type," reflecting the mentality and belief that behaviors of children with autism were most similar to, and were likely an early manifestation of, adult schizophrenia. By the release of the DSM-II in 1968, despite Rimland's work, the increasing understanding of the biological nature of the disorder, and evidence suggesting children with autism had a "distinct" disorder, they were still diagnosed as "schizophrenic, childhood type." Features of the disorder were listed as "autistic, atypical, and withdrawn behavior," "general unevenness, gross immaturity, and inadequacy in development," and "failure to develop identity separate from the mother's" (APA, 1968). The latter criterion may be a reflection of psychodynamic theories at the time, wherein mental disorders were seen as caused by broad underlying conflicts or maladaptive reactions to life problems.

It was not until 1980, in the DSM-III (American Psychiatric Institute), that "infantile autism" was listed as a separate diagnostic category. The publishing of the DSM-III marked a radical reorganization of psychiatry and mental illness, with the role of diagnosis changing from marginal to being the basis of the specialty (Mayes & Horowitz, 2005). The DSM-III, which heralded the "medicalization" of mental illness, also introduced a system of five axes or dimensions for assessing all aspects of an individual's mental and emotional health. Autism was originally coded on Axis II, which included disorders of development and personality disorders. Infantile autism was listed as one of two "pervasive developmental disorders" (PDDs). Coined for use in the DSM-III, the term PDD was chosen for two main reasons. First, it was meant to subsume both autism and a number of conditions that shared some features with it. Second, it was meant to

convey the broad extent (i.e. pervasiveness) of impairment across different domains in the disorders it encompassed (Volkmar & Cohen, 1991). One fundamental new aspect in the DSM-III was its use of categorical, symptom-based diagnosis to define mental illnesses. The criteria for a diagnosis of infantile autism included the following: (a) an onset before 30 months of age; (b) pervasive lack of responsiveness to other people; (c) gross deficits in language development; (d) if speech is present, peculiar speech patterns such as immediate and delayed echolalia, metaphorical language, and pronominal reversal; (e) bizarre responses to various aspects of the environment (e.g. resistance to change, peculiar interest in, or attachments to, animate or inanimate objects); and (f) absence of delusions, hallucinations, loosening of associations, and incoherence as in schizophrenia. The other diagnosis in the PDD category was pervasive developmental disorder – not otherwise specified (PDD-NOS) whose diagnostic criteria included the following: (a) gross and sustained impairment in social relationships; (b) three features from a list including excessive anxiety, constricted or inappropriate affect, resistance to change, oddities of motor movement, abnormalities of speech, hypo- or hypersensitivities, or self-mutilation; and (c) an onset of the full syndrome after 30 months of age and before 12 years of age with the absence of delusions, hallucinations, incoherence, or marked loosening of associations.

A major revision of the DSM was published in 1988 (DSM-III-R) wherein autistic disorder (299.00) was listed as the primary diagnosis still within the category of pervasive developmental disorders on Axis II. Diagnostic criteria were greatly expanded such that individuals had to exhibit at least two specific behaviors (listed within the criteria) from each of three broad categories: (a) qualitative impairments in reciprocal social interaction; (b) qualitative impairments in verbal and nonverbal communication and in imaginative activity; (c) markedly restricted repertoire of activities and interests, and an onset during infancy or early childhood. The second diagnosis within the category remained "pervasive developmental disorder – not otherwise specified" (PDD-NOS; 299.80), which included children with qualitative impairments in reciprocal social interactions and verbal and nonverbal communication skills but who did not meet the full criteria for autistic disorder.

The introduction of the DSM-IV in 1994, our current diagnostic criteria, represented another major shift in the conceptualization of the diagnosis of autism. First, autistic disorder was re-classified onto Axis I, as a clinical disorder, with Axis II diagnoses limited to coding intellectual disabilities (recognizing the independent nature of autism and intellectual capacity) and personality disorders. Second, PDD was reconceptualized as including a greater number of disorders, now frequently referred to as autistic spectrum disorders (ASDs) in the literature, which are defined as disorders primarily impacting social interaction. Interestingly, while commonly used, the term ASD does not actually appear in any version of the DSM (which instead uses the PDD designation) but is nonetheless widely used in the scientific literature, and even in clinical settings. Indeed, the social deficits in ASD are now viewed as forming a continuum from severe to relatively mild and are not necessarily correlated with the individual's language skills or intellectual capacity (Constantino, Przybeck, & Friesen, 2000; Posserud, Lundervold, & Gillberg, 2006). The category of PDD expanded to include autistic disorder, Asperger's disorder, Rett's disorder, childhood disintegrative disorder, and pervasive developmental disorder – not otherwise specified. While this grouping includes several disorders with many similar characteristics, it does not include all disorders which lead to a pervasive, lifelong developmental disorder (e.g. disorders such as fragile X and Landau–Kleffner syndromes were not included despite their impact on social interaction). Likewise, some have argued that Rett's disorder and childhood disintegrative disorder, while sharing some features with autistic disorders, are likely "separate" disorders and are perhaps best conceptualized of as including "autistic-like features" (Ozonoff, Goodlin-Jones, & Solomon, 2007; Rutter, 2005).

Rett's syndrome (RS) was first described in 1966 by Andreas Rett, a pediatrician working in Vienna. He wrote, in German, about the cases of 31 girls who had showed a regressive decline in mental ability early in life. In addition to mental retardation, they all showed behavioral abnormalities, stereotypic hand wringing, and also evidenced hand and gait apraxia. Rett also used the term "autism" as a descriptor of the disorder (Rett, 1966, as described by Hagberg, 1993). It was almost two decades later, when a publication of the disorder was made in English (Hagberg, Aicardi, Dias, & Ramos, 1983), that the disorder became a focus of study with the first set of diagnostic criteria for Rett's syndrome established in 1984 (Hagberg, Goutières, Hanefeld, Rett, & Wilson, 1985). The infant with RS characteristically develops typically for the first 5 months of life, followed by a noted deceleration in head growth between 6 months and 4 years of age, and a regression of already developed skills, including use of hands, motor coordination and gait, as well as communication skills. Mutations in the *MECP2* gene have been identified as a cause of RS (Amir et al., 1999) and are detected in most individuals with RS (Percy, 2008), though importantly these mutations do not appear to play a role in autism (Beyer et al., 2002). Indeed, some researchers have noted qualitative differences in the social behavior of individuals with RS and those with autism (Dahlgren Sandberg, Ehlers, Hagberg, & Gillberg, 2000; Olsson & Rett, 1987). Others have even argued that criteria for autistic disorder are frequently not met in those with RS and that it should be seen as a possible "co-morbid" diagnosis for some individuals

(Wulffaert, VanBerckelaer-Onnes, & Scholte, 2009) as, given current DSM-IV (TR) criteria (APA, 2000), individuals with RS cannot be separately diagnosed with autistic disorder.

Childhood disintegrative disorder (CDD), otherwise known as Heller syndrome or disintegrative psychosis, was first described by Heller in 1908. Heller described six cases of children who, after a normal developmental period of 3–4 years, showed a severe developmental regression. He proposed the term *dementia infantilis* for the condition. Diagnostic guidelines were subsequently set (Heller, 1930, as described by Volkmar, 1992) and included the following: (1) age of onset between 3 and 4 years; (2) loss or impairment of speech at onset with progressive intellectual and behavioral deterioration; (3) affective symptoms (fear, overactivity) and possible hallucinations; and (4) an absence of obvious markers of neurologic dysfunction (e.g. facial abnormalities). In comparison to autism, the most typical onset for CDD is between 2 and 4 years of age and is characterized by a substantial period of normal development followed by a profound developmental regression. Children affected by CDD can lose all of their language, toileting abilities, self-care skills, and interest in their environment, at which point the behaviors seen in affected children mimic those seen in autistic disorder. CDD does differ from autism in the pattern of onset, course, and aspects of outcome. In fact, estimates based on four surveys of individual ASD found fewer than 2 children per 100,000 could be classified as having CDD (Frombonne, 2002). Additional studies comparing children with autism and CDD have found that children with CDD have a significantly higher rate of epilepsy (Hendry, 2000; Kurita, Osada, & Miyake, 2004; Mouridsen, Rich, & Isager, 1998), and in one study, children with AD had significantly more even levels of intellectual functioning, and a tendency for less abnormality in auditory responsiveness than did children with CDD (Kurita et al., 2004). As noted by Rutter (2005), it remains unknown whether CDD "constitutes an unusual variant of autism or is something quite different" (p. 5).

Since Kanner's original characterization of autism, a fairly broad range of disorders with social interaction impairment at their core have been seen as being part of a broader phenotype, now labeled "autistic spectrum disorders." More recently, advances in genetic analysis, neuroimaging, and more refined descriptions of neurobehavioral symptomatology have allowed a better characterization of disorders such as Rett's disorder and CDD which, while sharing features of autism, certainly appear to have a very different etiology and course. While this may arguably preclude them from categorization as being autistic spectrum disorders, there is also a question of the utility and validity of identifying "separate disorders" in children who often present with such significant overlaps in behavioral symptomatology.

Indeed, during a discussion of the inclusion of Asperger's syndrome as a diagnosis, Schopler (1985) stated that AS was actually a "higher level" (i.e. higher functioning) autism. Wing (1981) originally agreed with this and formally suggested that either "AS or high-functioning autism" be placed at the mild end of the autistic spectrum (1991). Stemming from considerable overlap between Asperger's syndrome and autism, controversy still exists regarding whether AS is a distinct diagnostic category or whether it is indistinguishable from high-functioning autism (HFA). Furthermore, if they are meaningfully "separate" disorders, there still remains a question of the utility of treating them as such. Modern diagnostic systems have been categorical, though recently there has been a shift toward a dimensional classification system, given the significant occurrence of both co-morbid mental disorders and the overlap of symptomatology across diagnostic categories. It is argued that a dimensional approach is more suited to capture the entire range of severity and symptomatology of a disorder and to allow for greater flexibility in diagnosis (Kraemer, Noda, & O'Hara, 2004; MacCallum, Zhang, Preacher, & Rucker, 2002).

According to DSM-IV (TR) criteria (APA, 2000), a primary feature that distinguishes autism from AS is a language delay. Thus, a child is given a diagnosis of autism only if typical language milestones are not met, such as the use of single words by age 2 and meaningful phrases by age 3. However, research has shown that in some cases, children who have received diagnoses of autism demonstrate symptoms that are more consistent with AS, with the sole exception of the language criterion (Prior et al., 1998). Also, not all individuals with diagnoses of HFA demonstrate a severe language delay (Miller & Ozonoff, 2000). Furthermore, when children with autism or AS are grouped according to early language history, no other significant differences exist between the groups (Manjiviona & Prior, 1999). An additional study found that language ability measured in 6–8-year olds accounted for greater variability in later adaptive behavior and autism severity than the presence of an early language delay (Bennett et al., 2008). Overall, this research suggests that the presence of a language delay may not truly differentiate between HFA and AS and calls into question the diagnostic utility of differentiating AS as a separate disorder rather than as a variable expression of the symptomatology of autism. Indeed, questions about the importance and utility of the early language deficits were noted early on by Kanner and colleagues.

Contrary to these findings, other researchers have found support for the notion that groups of children with HFA and AS are indeed distinguishable on the basis of their early language developmental histories. In one study, a group of children with HFA displayed more severe behavioral symptoms in general and within the language domain had longer