Sowing the Seeds of the Autism Field: Leo Kanner (1943)

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Abstract

More than 65 years after Leo Kanner published his seminal article, research on autism continues to be an area of increasing interest. Although much progress has been made, this field is still in its infancy, and many avenues of research are just beginning to be pursued. Despite the time that has passed, the syndrome Kanner identified and his comments about the children he observed continue to have meaning today, and although some of his suggestions about the etiology and presentation of autism were grounded in the thinking of his day, many of his observations were quite prescient. In this paper we explore Kanner's contributions to the field of autism, discuss how the field has changed, and suggest ways that research on autism spectrum disorders can continue to move forward.

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These characteristics form a unique ''syndrome,'' not heretofore reported, which seems to be rare enough, yet is probably more frequent than is indicated by the paucity of observed cases''—Kanner (1943, p. 242)

In 1943, Leo Kanner published a paper entitled "Autistic Disturbances of Affective Contact," in which he presented, in vivid detail, the family background of 11 young children with seriously disordered behavior. This clinical account, in which Kanner understood and conveyed autism as distinct from other conditions, has been considered a seminal article on autism. By now, two thirds of a century later, there have been over 1,000 citations to it (psycINFO psychological abstract database). Our purpose here is to consider Kanner's early contributions from the perspective of advances in our definition and understanding of autism over the ensuing years and to consider how research on autism might move forward. We conclude that many of Kanner's prescient observations still find expression in the current avenues of research on autism.

Kanner's Cases: The Original 1943 Context

Kanner (1894–1981) was an Austrian psychiatrist who immigrated to the United States and occupied a leadership position at Johns Hopkins University Hospital. He was considered to be the father of child and adolescent psychiatry, and his textbook defined this new field. His most abiding influence, though, has been his 1943 paper, in which he introduced the term *early infantile autism*, thus identifying a unique group of children. Today, autism is no longer rare (Grether, Rosen, Smith, & Croen, 2009), with the incidence of autism estimated as high as 1 in 110 births by the Center for Disease Control (Rice, 2006). Although some question whether there is a true increase in special education classrooms in the United States (Shattuck, 2006), it is no longer a question of whether public school teachers will encounter children with autism, but when.

Kanner was an acute observer of children. In Table 1, we present the behavioral symptoms that he noted for each of the 11 children, ages 2 to 10 years, described in his seminal paper. We also note relevant information he presented about the child's family, medical history, and developmental history. Synthesizing the information from the different cases, Kanner inferred that the observed symptoms represented a syndrome that was probably less unusual than the number of cases would indicate. Here are the key symptoms that Kanner reported:

Table 1 Kanner (1943) Case Symptoms and *DSM*-IV (2000) Diagnostic Criteria

Kanner's symptoms	Other: Predictive	Social interaction	Language	Repetitive behavior	Onset before Age 3	At least 6 total criteria
	Donald T. (age 5-1)					
Does not look at the person while speaking	"Obsessive detail" from father's typewritten history	X	X	X	X	X
Does not use communicative gestures	Eating always a problem for Donald; sweets not a temptation					
Not interested in playing with other children; ignores other children	Walked at 13 months					
Prefers objects to people	Excellent rote memory for song lyrics, alphabet and numbers					
Father reports that he appears to "draw into his shell and live within himself." He does not seem to notice the coming or going of other people nor react to their coming with pleasure.	Had temper tantrums in which he was destructive					
Does not come when called	Had difficulty associating his punishment with his behavior					
Regards other people as an interference or plays with their hands/feet as an object	Showed learning – better contact with the environment, became more imaginative over time					
Delay in use of questions and ability to answer questions	Later learned to read and play the piano					
Conversation was obsessive in nature – inexhaustible questioning	No initiation of activities other than the limited interests in which he was absorbed; mother had to direct him in all other activities					
Ejaculating words and sentences such as "Chrysanthemum"						
Inflexible use of language						
Pronoun reversal						
A "mania" for spinning round objects						
Arranging beads, blocks, etc. in a certain manner						
Verbal rituals requiring others to repeat certain phrases during particular activities (e.g., bedtime)						
Stereotyped finger movements, shaking his head and whispering or singing the same three-note tune						
Difficulty in social communication (learning to ask and respond to questions) present at age 2						
	Frederick W. (age 6-0)					
Does not respond to questions	Aggressive and withdrawing behavior	(X)	X	(X)	X	?
Does not anticipate being picked up	Afraid of mechanical things (e.g., vacuum, elevators)					
Not interested in playing with other children; ignores other people	Walked at 18 months					
Regards other people as an interference or plays with their hands/feet as an object	Head circumference as 21 in					

Table 1 continued

Table 1 Continued

Kanner's symptoms	Other: Predictive	Social interaction	Language	Repetitive behavior	Onset before Age 3	At least 6 total criteria
Refuses affection from mother Very interested in objects (in contrast to people) Failed to speak more than two words prior to age 2; later occasional word use Pronoun reversal Ejaculating unintelligible sounds and echolalic responses to questions or commands Failed to develop cooperative play Frequent kissing or sucking sounds Maintained his furniture in the same arrangement Spoke 2 words prior to age 2 and used multiple words between age 2 and 3; never used words to communicate	Skull X-ray (normal) Sang at age 2½ Lack of cooperation in testing Father and paternal uncle talked late in development					
	Richard M. (age 3–3)					
Did not anticipate being picked up nor did he adjust his body once picked up Not interested in other people; regards other people as an interference Did not speak before age 3 Responds to some commands and makes occasional unintelligible sounds, but no recognizable words Upon entering a room, turned lights on and off repeatedly Regression in abilities such as sound imitation over the last 2 years (prior to his 3rd birthday). Did not speak prior to age 3	Appeared deaf early on Walked at approximately 1 year of age Mother noted that he stopped imitating word sounds and moved "backward mentally gradually for the last 2 years" Went into a rage when he didn't get what he wanted Head circumference 54½ cm EEG normal Note: Before 5 years old, placed into foster home Mother's note: "Indicated obsessive preoccupation with details" Father immersed in work, "almost entirely to the exclusion of social contacts"	?	X	?	X	?

Table 1 continued

Table 1 Continued

Kanner's symptoms	Other: Predictive	Social interaction	Language	Repetitive behavior	Onset before Age 3	At least 6 total criteria
	Paul G. (age 5-0)					
Does not look at people's faces Not interested in other people or other children his age, but very interested in objects Treats other people, or parts of them, as objects Rarely responds when called Meaningless verbal statements repeated in response to objects or activities without communicative value Pronoun reversal Ejaculates sounds and utterances that could not be linked to the immediate situation; often echolalic repetitions of phrases said to him Frequent masturbation Running in circles; shaking a blanket while yelling—all repeated each day in the same manner No mention of deficits prior to age 3	Thought to be feebleminded Frequent temper outbursts Memorized songs and poems at age 3 Vomited frequently in his first year of life, but stopped when he began eating solid food Walked and was toilet trained within typical limits	(X)	X	(X)	?	?
	Barbara K. (age 8-3)					
No affective contact with interviewer Ordinary vocabulary at 2 years of age, but had difficulty putting sentences together Pronoun reversal Repeated phrases Perseverative interest in "motor transports" and "piggy-back"... also smoke stacks, pendulums, and toilets Holds things and takes them to bed with her Stereotyped drawings of a house, man, pumpkin, etc. Protruding tongue Played with hand as others might play with a toy No mention of deficits prior to age 3	Eating difficulties until 18 months Phenomenal ability to spell and read (though no age given for this), but unable to relay what was read Could do arithmetic, but only as a "memory feat" Appeared deaf (but wasn't) Scribbled words spontaneously, with words all running together Fears of changing things like the wind and large animals	?	(X)	X	?	?

Table 1 Continued

Table 1 Continued

Kanner's symptoms	Other: Predictive	Social interaction	Language	Repetitive behavior	Onset before Age 3	At least 6 total criteria
	Virginia S. (age 10-6)					
Responds to being called (and other commands) but does not look at the person who called her Not interested in other people No display of affection Does not speak A few words at age 9—*mama* and *baby* (sometimes in response to a question)—but not used to communicate Delay in use of any language prior to age 9 and no attempts at communicating through other means	Appears deaf (but wasn't); also diagnosed as feebleminded and placed in a state training facility for the feebleminded Strong concentration and focus on what she is doing: able to finish complicated puzzles and determined to do so in one sitting From Dr. Esther L. Richards: "All findings seem to be in the nature of a congenital abnormality which looks as if it were more of a personality abnormality than an organic defect." Seems to be in a world of her own Hums to herself, makes some sounds Nonverbal IQ of 94; believed to be higher According to father, mother treats children like "dolls" Father only involved if there is a disciplinary problem Brother describes home as "a frosty atmosphere"	?	X	?	X	?
	Herbert B. (age 3-2)					
Becomes agitated at interferences but does not look at the person interfering Not interested in other people Does not respond to being called nor to other words directed at him	Initially thought to be deaf; profoundly mentally retarded Vomited all food until 3 months of age	?	X	X	X	?

Table 1 continued

Table 1 Continued

Kanner's symptoms	Other: Predictive	Social interaction	Language	Repetitive behavior	Onset before Age 3	At least 6 total criteria
Total lack of language and no clear attempts to communicate through other means	Walked at age 2, but without usual precursors, such as crawling					
Inarticulate sounds repeated in a sing-song manner	Fear of running water, gas burners, and many other things					
Pulls blinds up and down, tears cardboard, and opens and closes the wings of doors	EEG normal					
Changes in accustomed patterns met with fussiness and crying	Father a psychiatrist; described as "not interested in people, mostly living within himself"					
Delay in language through at least 5 years of age	Sister, 3 years older than Herbert, regressed at 2 years old; she wanted to be left alone, made "queer" noises, ignored people completely except mother; speech was meager and she had difficulties with pronoun use; later "recovered." By the time she attended school, became more social and had an IQ of 108.					
	Alfred L. (age 3-6)					
Plays alone	Minor, early problems with formula (for 2 months)	?	(X)	X	?	?
Avoids other children when playing	Swallowed odd objects during infancy/young childhood					
Pronoun reversal	Difficulty taking intelligence test, but had IQ of 140					
Does not ask questions or uses appropriate inflection when doing so	Fears, mainly of mechanical noises … sometimes turn into perseverative interests					
Later, confused by the meaning of words such as *about*	Excellent rote memory					
Later, obsessive questions about topics such as windows	Mother obsessive and excitable					
Repeats a particular phrase over and over						
Parents describe him as having one special interest that preoccupies him; frets when not allowed to indulge his interest						
Upset by changes in routines or the environment, such as the sun setting						
No mention of deficits prior to age 3						

Table 1 continued

Table 1 Continued

Kanner's symptoms	Other: Predictive	Social interaction	Language	Repetitive behavior	Onset before Age 3	At least 6 total criteria
	Father did not get along with others, suspicious, preferred to be alone (e.g., gardening, reading) Maternal grandfather had tics, was obsessive; repeated hand washing, protracted thinking along one line	?	(X)	X	?	?
	Charles N. (age 4-5)					
Does not appear to recognize or attend to mother's presence Fails to look at or make eye contact with other people Does not come when called Interacts with body parts (e.g., mother's hand) without recognition that it belongs to a person Detached from other children at school Fails to initiate a conversation Pronoun reversal Repeats or quotes other people Spins lids and jars for hours at a time Interested in reflected light Obsession with feces No mention of delays in these areas prior to age 3	Suspected of having hypothyroidism as a baby and given "thyroid extract" without any ensuing change At 18 months he recognized 18 symphonies Walked at less than 15 months Strong memory for words and able to repeat whole conversations Destruction of objects and toys Musical talent in the family (maternal grandmother, maternal aunt, maternal uncle) Maternal grandmother described as "dynamic, forceful, hyperactive, almost hypomanic"	(X)	(X)	X	?	?
	John F. (age 2-4)					
Limited affective contact only with certain people Pronoun reversal Repeats or quotes other people; not used to conversing	Substantial feeding problems starting in early infancy; hospitalized	?	(X)	X	?	?

Table 1 continued

Table 1 Continued

Kanner's symptoms	Other: Predictive	Social interaction	Language	Repetitive behavior	Onset before Age 3	At least 6 total criteria
Daily rituals must be adhered to rigidly or else appears to panic Maintenance of environment and extreme upset at changes to environment Although seen prior to 3 years of age, no discussion of his language use or social interaction	Anterior fontanel closed late (2.6 years) Walked at 20 months Excellent rote memory: endless repetition of sentences, prayers, rhymes, songs in different languages Later improvements in pronoun use At 2 years, 4 months failed to associate objects Difficulty making comparisons (e.g., which is bigger?) Convulsions at age 5 (right-sided), with transient paresis of the right arm EEG indicated "focal disturbances in the left occipital region" Mother hypomanic Paternal grandmother "obsessive about religion and washes her hands every few minutes"	?	(X)	X	?	?
	Elaine C. (age 7-2)					
No communicative gestures No facial expressions when speaking Does not look at the person she appears to be addressing Knows facts about other children but unable to enter into a relationship with them Four words spoken at the end of her first year of life, but a delay in linguistic development until 5 years of age	Plateau in language development: four words from 12 months until 5 years Walked at less than 12 months of age and spoke 4 words by the end of first year of life	X	X	X	X	X

Table 1 continued

Table 1 Continued

Kanner's symptoms	Other: Predictive	Social interaction	Language	Repetitive behavior	Onset before Age 3	At least 6 total criteria
Rarely able to answer questions; echolalic repetitions of the question	Deafness later suspected, but ruled out					
Pronoun reversal and inflexible use of phrases	Also previously diagnosed as feebleminded, postencephalitic behavior as a result of fever at 13 months					
Difficulty with intonation						
Mechanical phrases unrelated to situation or related in bizarre way	For brief period given "thyroid preparations"					
Ejaculating bizarre phrases	Frightened by noises (e.g., vacuum)					
Upset at interruptions of routines	EEG normal					
Rhythmical movements—masturbation	Maternal grandmother "emotionally unstable"					
Delay in language until 5 years of age						

Note: X = meets criteria, (X) = some symptoms or impairment and likely meets criteria, ? = insufficient information.

Autistic Aloneness

Kanner indicated that the fundamental core issue of this disorder is the children's inability to relate to people and objects in an ordinary way. He described their lack of social relatedness as "extreme aloneness." He spoke of this preference for being alone by noting the child's avoidance of noisy objects as well. "Yet it is not the noise or motion itself that is dreaded. The disturbance comes from the noise or motion that intrudes itself, or threatens to intrude itself, upon the child's aloneness" (Kanner, 1943, p. 245). Regarding the child's lack of interest in others, Kanner stated that "it would be best to get these interferences over with, the sooner to be able to return to the still much desired aloneness" (pp. 249–250).

Kanner (1943) noted that many of the children had been diagnosed with schizophrenia at one point. He differentiated the two disorders, however, stating that a person with schizophrenia steps outside his or her world and departs from already existing relationships, whereas the children he described had never established such relationships, experiencing an extreme aloneness from very early on.

Speech Disturbances

Kanner (1943) further noted the centrality of speech disturbances to this disorder, observing that many of the children were delayed in their speech and that those who were verbal often used speech in peculiar ways (e.g., echolalic repetition of phrases and/or inflexible use of language as seen in the exact repetition of pronouns).

Preservation of Sameness and Rote Memory

Kanner also observed that the children's behavior was governed by an anxious and obsessive desire for sameness and that this resulted in their repetitions of actions and limited spontaneous activity. A related cognitive attribute noted by Kanner was that many of the children had an excellent rote memory, which led their parents to "stuff" them with verse, lists of animal and botanical names, favorite songs, and random facts.

Developmental History

Kanner indicated that 4 of the children had been considered deaf or hard of hearing early on. He also reported early difficulties with eating and suggested that eating may have represented the first intrusion into the children's extreme aloneness. He noted that the children had no particular health difficulties and that their EEG results were normal. He did, however, observe that 5 of the 11 children had relatively large heads and a few were somewhat clumsy in their gait.

Parents

Kanner also recounted his observations of the children's families, a topic that we will expand upon shortly. He noted the high level of intelligence characterizing parents and relatives, while at the same time asserting that there were few warm-hearted parents among the families he observed. He suggested that parenting might contribute to the development of autism, but he also balanced this suggestion with the observation that the aloneness of these children was present very early on, making it unlikely that the whole picture of the disorder was the result of parenting.

Kanner's 1943 paper eventually had a profound impact on the fields of psychology, education, and developmental disability. In his "discovery" of autism, Kanner focused on the clinical presentation of a group of individuals and then inferred the common features of the disorder. Although he gave some insight into possible pathways through which the disorder might emerge, Kanner's description of autism was largely atheoretical, without a clear hypothesis about the etiology of the disorder. His methodology was, in some ways, a return to earlier psychological research, grounded in clinical presentation and case studies as opposed to the predominant psychoanalytic theory of the times. His work preceded a productive; enlightening; and, in the case of families, a corrective body of autism research.

Autism From 1943 to Today

Thirty years after Kanner's seminal publication, renowned psychiatrist Sir Michael Rutter (1973) paid this tribute to Kanner's observations.

> The field of childhood psychoses is strewn with descriptions of this or that syndrome which purport to represent some distinct entity. Most of these have passed into the sands of time but one—a careful clinical description of 11 children—remains as important today as when it first appeared. … Nearly all of the basic points made in the original paper have been amply confirmed by other workers. (pp. VII, VIII)

In addition, Schopler, Chess, and Eisenberg (1981) published a memorial to Kanner in which they recognized his work in autism as well as in a number of unrelated fields (e.g., teeth). Here, in order to evaluate further the relevance of Kanner's original description of an autism syndrome to the current field of autism research, we consider whether and how our understanding of autism has changed over time, especially in recent years.

History of Diagnosing Autism

Behaviors similar to those in Kanner's conception of autism had been described prior to 1943, with accounts of the Wild Boy of Aveyron (Land, 1977) and stories of Brother Juniper of St. Francis of Assisi (Frith, 1991) comprising some of the earliest descriptions. Few researchers, however, discussed the similarities between individual cases and/or grouped children into categories based on similar clinical presentations. Only in the late 19th and early 20th centuries did researchers begin to consider similarities in the behavioral phenotypes of these children. Rather, earlier investigators considered these groups of children to represent subgroups of childhood-onset schizophrenia or childhood psychosis (for a review, see Wing, 1997). Like his colleagues, Kanner described and named a cluster of behavioral characteristics. He was the first, however, to give this phenotype an identity separate from childhood psychosis. Moreover, he distinguished himself by presenting his 11 cases in substantial detail, something that some of his colleagues failed to do (Wing, 1997). For this, Kanner's 1943 paper is often accredited as the foundational paper in autism research.

Just one year after Kanner, in 1944, Hans Asperger, an Austrian pediatrician, published an account of four children with typical intellectual functioning and speech, but with significant impairment in social interactions and restricted stereotyped patterns of interests and behaviors (Asperger, 1944). Published during the war years and in German, Asperger's observations went largely unnoticed and were not translated into English until 1991 by Frith. Asperger's writings were quite similar to Kanner's; he coined the term *autism* or *autistic psychopathy* to describe the children he observed and similarly indicated that the syndrome he described was distinct from childhood schizophrenia. Unlike Kanner, however, Asperger believed that the syndrome he described was rarely, if ever, recognized in infancy (Frith, 1991; Wing, 1981). He noted the similarities between his syndrome and Kanner's early infantile autism but considered the two to be distinctly different.

Although in his original article Kanner clearly delineated a number of behavioral characteristics

common to the 11 individuals he studied, he did not establish specific diagnostic criteria for autism. In a later article, co-written with Harvard psychiatrist Leon Eisenberg, diagnostic criteria were spelled out more clearly (Eisenberg & Kanner, 1956). They emphasized the children's extreme aloneness and a preoccupation with the "preservation of sameness (p. 57)." They also discussed the children's "failure to use language for the purpose of communication" (p. 56) but suggested that this feature was "derivative of the basic disturbance in human relatedness" (p. 57). Moreover, they noted that "symptoms" (using the nomenclature of the time) must be present in the first 2 years of life. Despite Kanner's (1943) description of the syndrome and the diagnostic criteria presented by Eisenberg and Kanner (1956), however, autism was not included as a diagnosis in the first two versions of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*-I published in 1952, *DSM*-II published in 1968).

After benign neglect in the 1940s and 1950s, the 1960s witnessed the beginning of a fascination with autism that rapidly grew. There was increasing confusion as well between traditional psychodynamic views and the newer behavioral ones, resulting in further focus on diagnostic criteria. Rutter (1978) argued that by reducing the detailed case studies to a few broadly defined core symptoms, Eisenberg and Kanner (1956) inadvertently encouraged other researchers to redefine or reinterpret the criteria for autism, using very different behaviors as indicators of the underlying clinical phenomenon.

As a result, autism research in the 1960s and 1970s involved a multitude of different diagnostic criteria that placed individuals with very different clinical presentations under the umbrella of autism. In addition, many researchers and clinicians continued to use the terms *autism*, *childhood schizophrenia*, and *childhood psychosis* interchangeably. In his criticism of the various diagnostic systems, Rutter (1978) pointed out that by describing a cluster of symptoms and labeling this cluster autism, Kanner had offered the hypothesis that a distinct disease entity was responsible for the noted symptoms. Rutter expanded upon the *disease entity*, defining it as the presence of impaired social relationships marked by a lack of attachment behavior and lack of eye-to-eye gaze prior to age 5, a lack of cooperative group play, a failure to make personal friendships, and a lack of empathy/social perception after age 5. Rutter also noted deficits in language as seen in the delayed development of language skills and/or prelanguage skills, such as social imitation and imaginative play. For those children who did develop language, Rutter reported a number of characteristic abnormalities, such as *echolalia* (i.e., echoing another's speech). Finally, Rutter also kept an *insistence on sameness* as a criterion for autism, elaborating that it referred to rigid play patterns, attachments to bizarre objects, preoccupations with particular activities, and rigid routines. In older children, this insistence on sameness could appear as obsessive or compulsive behavior and resistance to transitions or changes in the environment.

Rutter's (1978) description of autism, then, represented a return to the original clinical phenomenon as presented by Kanner. However, Rutter improved upon the original diagnostic criteria by presenting clear behavioral manifestations of the core symptoms. Finally, Rutter's criticisms underscored the need for diagnostic criteria that represented autism as a syndrome as well as for further research examining the validity of this syndrome as a singular entity. [We note that the professional insistence on use of the term *symptoms* implied that autism was a disease or disorder of biomedical origin. Today's acknowledgement that autism is a neurodevelopmental disorder is not strikingly different, in that subsequent editions of the *DSM* refer to autism symptoms rather than traits.]

Two years after Rutter's (1978) article, the *DSM*-III was published (American Psychiatric Association, 1980). Now autism and related disorders were grouped together as pervasive developmental disorders and listed on Axis I. The term *pervasive developmental disorders* was used to refer to the pervasive developmental delays seen in these disorders, and this category included infantile autism, atypical pervasive developmental disorders, and childhood-onset pervasive developmental disorders. Criteria for infantile autism did not depart much from those put forward by Kanner (1943) and elaborated on by Eisenberg and Kanner (1956) and Rutter (1978). To meet criteria for infantile autism, an individual had to display a pervasive lack of responsiveness to other people, gross deficits in language development or peculiar speech patterns (e.g., echolalia), and bizarre responses to various aspects of the environment. The *DSM*-III also specified that symptoms had to occur in the

absence of delusions, hallucinations, etc., and that symptom onset must have occurred prior to 30 months of age. Childhood onset pervasive developmental disorders was described in the *DSM*-III as being characterized by extremely disturbed emotional relationships and at least three of the following symptoms: inappropriate affect, resistance to change, oddities of motor movement, prosodic abnormalities, abnormal sensitivity to sensory stimuli, and self-mutilation. Here, too, all symptoms must occur in the absence of delusions or hallucinations, with symptom onset between the ages of 3 and 12 as opposed to prior to age 3 in autism. Finally, atypical autism was used as a "catch-all" diagnosis for individuals not fully meeting criteria for infantile autism or childhood pervasive developmental disorders.

Perhaps one of the biggest contributions of the *DSM*-III was the clear distinction between autism and childhood schizophrenia. In addition, the *DSM*-III represented the initial designation of multiple similar disorders within a larger category. The distinction between these different pervasive developmental disorders was based to some degree on the age of onset. It is interesting that the *DSM*-III criteria for infantile autism followed the triad of deficits identified by Kanner, whereas the criteria for childhood-onset pervasive developmental disorders included some similar symptoms but did not follow the triad of deficits. Finally, the inclusion of atypical autism represented the recognition that individuals may experience symptoms of the disorder without meeting the full criteria.

The idea that autism may have spectrum qualities was introduced by Wing (1997; Wing & Gould, 1979) who argued that disruption in reciprocal social interaction was the key component in a continuum of disorders and could be accompanied by a variety of other impairments. She indicated that some combinations of these impairments had been identified as specific syndromes, perhaps because of the frequency of their occurrence. However, she emphasized that the autism spectrum was complex and went considerably beyond "a straight line from severe to mild" **(p.)**. In particular, she argued that any pathological process might lead to deficits in reciprocal social interaction and that although disruption of social interaction might occur in isolation, it is more likely to occur as part of more widespread neurological dysfunction. In defining the spectrum in this manner, Wing allowed for the inclusion of multiple other disorders, such as fragile X and child disintegrative disorder, under the broad label of *autism spectrum disorders*. Although this term was quickly adopted by researchers in the field, the concept of the autism spectrum was not incorporated into the following version of the *DSM*.

With the publication of the *DSM*-III-R (American Psychiatric Association, 1987), the name of the disorder was changed from *infantile autism* to *autistic disorder* and 16 specific criteria were added under the three major areas of deficit. To receive a diagnosis of autistic disorder, one had to meet criteria on at least 8 of the 16 items, with at least 2 from the reciprocal social interaction domain and one each from the verbal/nonverbal communication and restricted repertoire of activities domains. In addition, the onset of symptoms must have occurred during infancy or childhood. The *DSM*-III-R also introduced the label of pervasive developmental disorder–not otherwise specified (NOS) and included in this category all individuals not meeting full criteria for autistic disorder. It is interesting that the pervasive developmental disorders-NOS criteria required qualitative impairments in social interaction and verbal/nonverbal communication, but individuals with pervasive developmental disorders-NOS may or may not show a markedly restricted repertoire of interests and activities. Thus, pervasive developmental disorders-NOS represented an extension of the concept of pervasive developmental delays to a broader group of potentially less impaired individuals. Moreover, by relaxing the age of onset to include both infant and childhood onset, the *DSM*-III-R extended the concept of autistic disorder to incorporate individuals who would have been diagnosed with either infantile autism or childhood onset pervasive developmental disorders based on the *DSM*-III. Likewise, the *DSM*-III-R autistic disorder included individuals with potentially milder symptom presentations and/or individuals experiencing a regression in abilities possibly as a result of organic etiologies, such as brain disease (Waterhouse, Wing, Spitzer, & Siegel, 1992).

The *DSM*-IV (American Psychiatric Association, 1994) and its offspring with the same diagnostic classifications but revisions in the text (*DSM*-IV-TR, American Psychiatric Association, 2000) carried on the tradition of diagnostic tinkering by reducing the 16 criteria for autistic disorder delineated in *DSM*-III-R to 12 criteria within the same three domains. These current

versions further refine the pervasive developmental disorders concept by not only including autism and pervasive developmental disorders-NOS, but also three other disorders: Rett syndrome, childhood disintegrative disorder, and Asperger syndrome. Although these additions represent an effort to clarify and specify the umbrella category of pervasive developmental disorders, it is unclear how successful these changes have been in capturing unique variance within the autism spectrum or pervasive developmental disorders category. Waterhouse et al. (1992) disputed that delineating different subgroups requires understanding the metadiagnostic domain from which the subgroups are drawn while at the same time distinguishing them from one another based on, for example, etiology or specific neurological deficits. These authors argued that the new pervasive developmental disorders subgroups (including Asperger and Rett syndromes) were defined by multiple characteristics and did not necessarily overlap with autism or pervasive developmental disorders symptoms.

Examining these changes in the name and diagnostic criteria for autism hint at the difficulties that have plagued research on and treatment of this now wide-ranging disorder. Over the long haul, the diagnostic criteria for autism have become more and more specific and the definition of the disorder narrowed (e.g., from *DSM*-III to *DSM*-IV). At the same time, the field has tried to capture the substantial heterogeneity of this disorder through the concept of an autism spectrum. It is interesting that the perception of autism as comprised of three core areas of deficit has remained relatively stable across time. In this light, the observations made by Kanner in 1943 continue to accurately direct the study of autism.

Kanner's Cases in Today's Diagnostic Scheme

In Table 1, we have considered the summary of the symptoms Kanner observed that in some combination constituted the syndrome of autism. Table 1 also indicates which of the three current major criteria for autism as designated in the *DSM*-IV-TR (American Psychiatric Association, 2000) that each child would meet. Examining Kanner's observations from this perspective, it becomes clear that few would fully meet today's criteria.

Donald T. (and perhaps Elaine C.) is the only case, based upon Kanner's description and assuming nothing else, that clearly would meet current diagnostic criteria. In some cases, Kanner's descriptions are not sufficiently detailed to make a diagnosis. Richard M., for example, "immediately proceeded to turn the lights on and off" when he entered the examination room; although this observation relates to the criterion of perseverative interests, self-stimulatory, and stereotyped behaviors, no others are mentioned. It is unclear in such cases if the child engaged in other behaviors that were not observed or noted by Kanner or if he or she did not experience difficulties in this area.

Other children would not fully meet *DSM*-IV-TR age of onset criterion, as Kanner did not state whether deficits in social interaction, language as used in social communication, and/or symbolic play were present before 3 years of age. Given that all but one of the children were observed after their third birthday, Kanner needed to rely on parent reports of the age at which children began to experience problems. Thus, we have the dual problem of limitations in parents' awareness and memory of problem onsets and Kanner's inattention to age of onset in his reporting. One might assume, though, that for many the onset was early, as Kanner soon referred to early infantile autism.

Despite the incompleteness of evidence by today's standards, it is important to note that all 11 children experienced symptoms in at least one or two of today's three core areas of deficit. It is also important that many parents reported deficits in associated areas, such as delays in expressive language. The combination of these associated deficits prior to age 3 (e.g., no expressive language) and current functioning (e.g., failure to make eye contact; ignores other people) suggests that the children may have experienced deficits in social interaction, social communication, and symbolic play prior to age 3 and would be likely to meet today's diagnostic criteria were more information available. In any event, it is noteworthy that two thirds of a century later, Kanner's description of these children does sound like today's autism.

At the same time, because these 11 cases overlap enough to suggest a syndrome, we also find substantial heterogeneity in the group. Actually, great heterogeneity among children diagnosed with autism spectrum disorders persists today. Despite the fact that some subgroups on the autism spectrum have been defined (e.g., Asperger syndrome, pervasive developmental disorder–NOS), many more children remain unclassified and lumped together under the umbrella of autism.

Moreover, the distinction between subgroups is not sufficiently clear, and, thus, the utility of subgroup-focused research is compromised.

The confusion from heterogeneous symptom presentations and not clearly differentiated diagnostic subtypes continues to be an impediment to research and intervention alike. Consider the increasing emphasis on genetic research into etiology of autism. Although heritability estimates indicate that autism and autism spectrum disorders are largely the result of genetic influences, genetic researchers have failed to find consistent evidence for more than a few chromosomal regions and specific candidate genes (Baader, Sanlioglu, Berrebi, Parker-Thornburg, & Oberdick, 1998; Badner & Gershon, 2002; Sutcliffe & Nurmi, 2003; for a review see Losh, Sullivan, Trembath, & Piven, 2008). Furthermore, the heterogeneity among cases challenges the idea of autism as a singular entity and the autism spectrum as a metadiagnostic category. Ultimately, research on the genetics, endophenotypes, and Gene × Environment interactions in autism will help to answer these questions by elucidating the pathways of autism spectrum disorders and whether different subgroups are genetically distinct from one another (Losh et al., 2008; Rutter, 2005).

Finally, Kanner developed his clinical descriptions using a range of assessment tools, including the observations of interns, parent reports, and his own careful observations. Today, researchers and psychologists continue to use a range of assessments, though with more attempts to quantify or to standardize data. This is accomplished, ideally, by using standard instruments in the diagnosis of autism that are still based on live observation (Autism Diagnostic Observation Schedule: Lord et al., 2000) and parent report (Autism Diagnostic Interview-Revised: Lord, Rutter, & LeCouteur, 1994).

Family Characteristics

The most controversial aspect of autism research, in the past, has been the portrayal of families. Kanner's initial observations of the children's family members led the field in two subsequent directions: One was the destructive and eventually unsupported notion of parental responsibility for autism (Bettelheim, 1967); the other was the more recent recognition of a broader autism phenotype (Piven & Palmer, 1997; Piven, Palmer, Jacobi, Childress, & Arndt, 1997).

Revisit Table 1 and consider Kanner's notes about families: These highlight delays, obsessive thinking, compulsive behaviors, and social isolation. Frederick W.'s father talked late. Donald T.'s father's written history of his child's behavior contained "obsessive detail." Richard M.'s mother's notes contained "obsessive preoccupation with details," and his father was immersed in his work "almost to the exclusion of social contacts." Similarly, Alfred L.'s mother was obsessive, and his father did not get along with others and preferred to be alone. Herbert B.'s father was "not interested in people, mostly living within himself." Grandparents, too, were singled out as having related characteristics: Alfred L.'s maternal grandfather had tics, was obsessive, demonstrated "repeated hand washing, protracted thinking along one line." Elaine C.'s maternal grandmother was described as "emotionally unstable," and John F.'s maternal grandmother was "obsessive about religion and washes her hands every few minutes." Kanner also noted that Charles N.'s maternal grandmother (as well as a maternal aunt and maternal uncle) had extreme musical talent. Only one clear-cut sibling case was mentioned. Herbert B.'s older sister regressed at the age of 2, when she desired to be alone, made "queer noises," demonstrated some pronominal confusion, and—with the exception of her mother—ignored people completely. Kanner reported that by the time she entered school, her IQ was measured at 108, and she became more social, although he did not cite clear changes in her social behavior.

Kanner (1943) was not sure how these family characteristics contributed to autism: "It is not easy to evaluate the fact that all of our patients have come of highly intelligent parents. This much is certain, that there is a great deal of obsessiveness in the family background." Kanner did suggest parenting as a contributing factor to the extreme aloneness of these individuals. He further said of their parents that there were few warm-hearted individuals among them and stated: "The question arises whether or to what extent this fact has contributed to the condition of the children."

Kanner also mentioned parenting behaviors that could negatively influence the course of autism. He spoke of one child, Vivian S., as having "been dumped in a school for the feebleminded," clearly with a tone suggesting a value judgment. Indeed, there is a tendency to imply that things were done to, or enacted upon, these children; for example, as noted earlier, parents "stuffed" them

full of facts, songs, or poems. Kanner asserted, "It is difficult to know for certain whether the stuffing as such contributed essentially to the course of the psychopathologic condition" (p. 243). Kanner's psychodynamic references and interpretation of the children's motives rather than simple descriptions of their behavior further reflect the historical context. For example, spinning—either of the child himself or of objects–is referred to as indicating "the presence of masturbatory orgastic gratification" (p. 246). In another case, Kanner stated that the child has a good "relation to objects."

The subsequent views of parents of children with autism as being cold and rejecting and the derogatory term *refrigerator parents* had their origins in these early observations, especially as viewed in a psychodynamic zeitgeist. This notion took its roots in Kanner's observations and was further propagated in some of his later writings (Kanner, 1949). In particular, he stated in his 1949 paper that his patients "were exposed from the beginning to parental coldness, obsessiveness, and a mechanical type of attention to material needs only." He went on to say that the patients were "kept neatly in refrigerators which did not defrost," conjecture rooted in the psychodynamic tradition of thinking about relationships with primary caregivers as the foundation of psychopathology. The psychoanalyst Bruno Bettelheim, unfortunately, promulgated the idea that rejection of the child by parents—especially the mother—somehow played a causal role in the development of their child's autism. This now unsubstantiated idea appeared in many of Bettelheim's writings, but it was popularized in his 1967 book. (Although Kanner introduced the possibility of a relationship between parenting and early infantile autism in his earlier writings, he clarified his position in many of his later writings. In particular, as he stated in his 1968 article, "at no time have I pointed to the parents as the primary, postnatal sources of pathogenicity."

Moreover, Kanner himself recognized that parenting was not the sole factor, as he pointed out that these children had experienced their self-induced isolation beginning at a very young age. Researchers subsequently determined that Kanner, with his widespread prominence in child psychiatry, had attracted parents concerned about their child's odd behavior from all over the country. Thus, the sample he based his initial observations on were parents who had the intelligence (and perhaps obsessiveness) to have read and learned about his work as well as the financial means to go to Baltimore for consultation. The idea that parents of children with autism were highly intelligent and from upper socioeconomic status was dispelled decades later, when researchers examined more representative samples of families and found that autism was no respecter of privilege; it was present in families ranging widely in intelligence and socioeconomic status. The *DSM*'s description of autism first included high family socioeconomic status in *DSM* III (American Psychiatric Association, 1980) but eliminated this correlate 7 years later in *DSM*-III-R (American Psychiatric Association, 1987).

Rather than ascribing blame to Kanner for planting this psychodynamic seed that led the field astray for several decades, however, we can appreciate his observations as providing the catalyst for later work on what we now know as the *broader autism phenotype* (Piven et al., 1997). This phenotype refers to a family genetic liability for autism found in nonautistic relatives of the individual with autism (Piven & Palmer, 1997; Piven et al., 1997.) The relatives described by Kanner (e.g., parents as well as grandparents, uncles, aunts, and even more distant relatives) often possessed some social communicative deficits and/or unusual linguistic or obsessive ritualistic characteristics, though not to the degree that would warrant a diagnosis of autism (Daniels et al., 2008; Macali, Chakrabarti, & Fombonne, 2004; Piven et al., 1997). The concept of a broader autism phenotype has contributed substantially to our understanding of the etiology of autism spectrum disorders, providing evidence for a genetic model of many genes, each with small effects. Moreover, research on relatives of individuals with autism aids our understanding of the etiology of this disorder by providing the means for examining the processes or endophenotypes that contribute to the development of autism. In light of the research on the broader autism phenotype, Kanner's aforementioned observations illustrated his foresight in recognizing the role of genetics in autism. In his concluding paragraph, Kanner (1943) stated: "We must, then, assume that these children have come into the world with innate inability to form the usual, biologically provided affective contact with people, just as other children come into the world with innate physical or intellectual handicaps" (p. 163).

Discussion

Kanner was an outspoken advocate for children, especially those with what we now call

intellectual disability. His sensitivity to what he perceived as parental rejection (i.e., his negative evaluation of parents for "dumping" their children in schools for the feeble-minded) reflected a value judgment to be sure, but also a concern for children and the abysmal state of institutional treatment at that time. Kanner both illustrated and criticized the treatment in the 1940s of children with autism, intellectual disability, and mental health disorders as objects or lesser individuals.

Although Kanner (1943) suggested that autism was highly stigmatizing, his labeling of the disorder was the beginning of autism awareness, so important today as the first step in proper diagnosis and treatment. Almost a half-century later, members of the Autism Rights Movement (founded in 1992 with the inception of the Autism Network International (Ward & Meyer, 1999) took a different perspective towards autism awareness by fighting the notion that autism and Asperger syndrome were mental disorders at all. Instead, groups such as Autism Rights Movement and the Autistic Self-Advocacy Network emphasized the idea of neurodiversity, in which autism is only a variation in typical functioning. These groups are concerned with disparaging societal views towards people with autism. Their members seek understanding and acceptance and ask that individuals with autism not be pressured by the neurotypical majority to imitate the norm. In this light, current emphasis on treating and/or curing autism is viewed by some advocates as attempts to eliminate a minority population. These groups allow individuals with autism to advocate for their right to be themselves and represent a valuable viewpoint that questions traditional conceptions of autism. Although Kanner's article was the foundation for our understanding of autism as a disorder (and in this manner, counter to the neurodiversity movement), we believe that Kanner would be pleased by the growing perspective that (a) mental health disorders in general lie on a spectrum and, thus, can reflect typical variations in behavior; (b) we should recognize people as people and not define them by the disorder they have; and (c) we should allow individuals appropriate independence and provide humane treatment. Likewise, we hope for an increasingly nuanced understanding of autism and appreciate the challenges the self-advocacy movement provides to traditional viewpoints.

Although Kanner (1943) focused on individual clinical case descriptions using the tools available to him, today our researchers are more likely to use group designs and seek statistical significance. There are trends toward more fine-grained analysis in the quest to clarify the heterogeneity in autism spectrum disorders, define meaningful subgroups, and find the genetic correlates of endophenotypes. Nevertheless, Kanner's keen observations of the lack of play and peer interaction as well as the presence of unusual sensory deficits in children with autism spawned current research on topics such as joint attention (Chiang, Soong, Lin, & Rogers, 2008; Colombi et al., 2009; for a review see Bruinsma, Koegel, & Koegel, 2004; Charman, 1998) and neurological deficits (Di Martino et al., 2009; Stanfield et al., 2008). Likewise, his observations of family members initiated research on relatives of individuals with autism and the broader autism phenotype (Bailey, Palferman, Heavey, & LeCouteur, 1998; Bolton et al., 1994; Fombonne, Bolton, Prior, Jordan, & Rutter, 1997; Landa, Piven, Wzorek, & Gayle, 1992; Murphy et al., 2000; Piven & Palmer, 1997). Although Kanner's early observations and diagnoses were not conceptualized within a neurodevelopmental or genetic framework, he did look for biological signs or symptoms of autism, most notably through the use of x-ray, EEG, and measurements of brain size and head circumference. Today, of course, researchers have amassed a wealth of knowledge about the brain structure and function of children with autism through the use of MRI technology (Hardan, Muddasani, Vemulapalli, Keshavan, & Minshew, 2006; Stanfield et al., 2008). Moreover, genetic researchers have established the prominent role of genetics in the etiology of autism (Baader et al, 1998; Badner & Gershon, 2002; for a review see Losh et al., 2008), although specific chromosomal regions and genes continue to elude us.

Today, we pay more methodological attention to our observations, and we recognize that research has grown much more sophisticated. This is due, in part, to some of the same factors that characterized the growth of the field of developmental disabilities in general: (a) the increased national concern for individuals with autism spectrum disorders and their families; (b) a concomitant and exponential increase in funding for research, for university-based centers, and individual investigators; and (c) the growth of technology, allowing for the use of sophisticated analytic techniques, genetic mapping, and neurological imaging.

Conclusion

A glance at the proposed revisions for the upcoming 5th edition of the *DSM* provides a clear

picture of the strides this field has made and continues to make in understanding the complexity of what is now considered a continuum of disorders. Perhaps the most striking development has been the growing view that autism is not a single disorder with specific behavioral manifestations, but part of a spectrum that overlaps with other genetic disorders and named syndromes, such as childhood disintegrative disorder and intellectual disability. This spectrum is characterized by core impairments in social communication and restricted, repetitive patterns of behavior and interests. Although there is no assumption that the etiologies of the heterogeneous conditions that comprise this spectrum are identical, researchers no longer consider it necessary to differentiate aspects of autism from Asperger syndrome, Instead, investigators view these labels as reflecting variations in language and cognitive abilities that impact the severity of an individual's clinical presentation. Accordingly, it is seen as more informative to describe individuals as experiencing different levels of severity rather than classifying them as having different disorders.

It is interesting to consider these developments in the context of Kanner's original observations. At first glance, the current view of autism spectrum disorders seems at odds with Kanner's description of a single disorder. Likewise, Kanner's insistence that early infantile autism was distinct from all other disorders appears unjustified in the context of current thought. At the same time, he continued to highlight in his later writings the children's extreme aloneness as the central piece of the disorder. Moreover, following his "discovery" of autism, Kanner immediately became concerned with differentiating this disorder from other forms of childhood psychopathology. In particular, in his 1949 paper, he compared early infantile autism to a host of other disorders, including various aphasias, schizophrenia, and Heller's disease or childhood disintegrative disorder (Kanner, 1949). Although his conclusions regarding the distinct nature of the syndrome may no longer be tenable and his wavering thoughts regarding the relationship between early infantile autism and schizophrenia misguided, his examination of the similarities and dissimilarities between early infantile autism and other conditions is notable. (Kanner continued to address the relationship between early infantile autism and childhood schizophrenia in his later writings. In 1968, he stated, "it matters little whether autism be regarded as a form of schizophrenia or looked upon as a disease sui generis." He went on to state that if there is a "group of schizophrenias" as suggested by Bleuler (1950), then it is merely a semantic issue whether early infantile autism is considered a member of that group. Instead, he emphasized the unique identity of early infantile autism even within that group.)

As the concept of an autism spectrum sees its way into the nosology, it is important to revisit the literature that began this field and carefully consider how it is moving forward. Although Kanner tried to pull together the heterogeneity he saw into a single disorder, researchers are now attempting to expand the current definition to better capture this variability. Our argument here is that perhaps both views provide meaningful directions for research and treatment.

Clinically, the autism spectrum nosology is much more helpful and allows for more thoughtful descriptions of a particular individual's strengths and weaknesses. These descriptions are likely to be more useful in treatment planning than catch-all labels, such as pervasive developmental disorders-NOS. Moreover, by creating a more inclusive category, we are taking steps towards providing services for more individuals. As it stands, some regional centers do not provide services for individuals with Asperger syndrome or pervasive developmental disorders-NOS diagnoses and, instead, require the specific diagnosis of autism for eligibility. The public schools have typically had less stringent standards for diagnosing "autistic-like behavior," but in many states or districts, children with Asperger syndrome are not included. By eliminating distinctions among individuals labeled as having autism, we may provide services to previously excluded children and adults, recognizing (as many already have) that social impairments, even when accompanied by relatively intact language and cognitive functioning, can be devastating in an environment not suited to the individual's needs.

In terms of research, we cannot ignore the fact that we must carefully define a particular phenotype in order to understand its etiology. In this light, the concept of an autism spectrum highlights social communication as the core deficit but provides too heterogeneous a picture to examine the spectrum as a whole (which is likely not the intent of this new nosology). Instead, the concept of the autism spectrum suggests that researchers

consider the various etiological pathways to social communication deficits as conferring evidence regarding the genes and neuroanatomy involved. In order to provide such a phenotype, however, we must consider subgroups of children with similar clinical presentations, whether they have named syndromes or not. In this way, our methodology may adopt elements of Kanner's focus on a narrow definition of a single disorder and his detailed observations of the similarities between individuals. Although we no longer subscribe to the belief that autism is a single disorder, we may find utility in conducting research in this manner, creating homogeneous groups of children, gaining insight about their behavior and the etiology of their symptoms, and then looking for similarities across groups.

Finally, it is important to revisit the discussion of parenting and the role of parents in the lives of children with autism spectrum disorders. Given the history of blame that resulted from Kanner's and especially Bettleheim's writings, as described earlier, it is understandable, if not to be expected, that many researchers have tip-toed around questions of parenting influences on outcomes in autism spectrum disorders. We hope that those researchers who choose to investigate these relationships will take care not to ascribe blame to parents for causing or somehow worsening a disorder that we now believe to be largely genetically determined. At the same time, it is vital that the field not ignore questions about the impact of parenting on children with autism to avoid upsetting and offending parents. Parents are a vital component of their children's environment, and if we are to do right by this field and by the people that we as researchers serve, then we must consider how best to help parents improve the elements of their child's environment that they can control.

Kanner's observations were made through a lens produced in his time, not in ours. As such, he used what was considered acceptable methodology and theoretical guidance. There are indeed earlier views about children with autism and their families that we do not wish to repeat, but there are also some pearls—insightful and forward-looking ideas that should not be lost. As noted by Blacher and Baker (2002) in their history of family research in intellectual disability, one cannot set out to look back without remembering the oft-quoted remark of the philosopher, Santayana, that "those who cannot learn from history are doomed to repeat it." Perhaps the most important lesson in successfully moving forward is to simultaneously ground ourselves in clinical observations of children. We hope that both the prescience and the limitations of Kanner in his 1943 paper will be appreciated by future generations, who increasingly will have the resources, technology, and wealth of accumulated knowledge to refine the field and our understanding of autism.

References

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (1st ed.). Washington, DC: Author.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: Author.

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.

Asperger, H. (1944). Die "autistischen psychopathen" im kindersalter. *Archives fur Psychiatrie und Nervenkrankheiten, 117*, 76–136.

Asperger, H. (1991/1944). "Autistic psychopathology" in childhood. (translated and annotated by U. Frith). In U. Frith (Ed.), *Autism and Asperger syndrome* (pp. 37–92). New York: Cambridge University Press.

Baader, S. L., Sanlioglu, S., Berrebi, A. S., Parker-Thornburg, J., & Oberdick, J. (1998). Ectopic overexpression of engrailed-2 in cerebella Purkinje cells causes restricted cell loss and retarded external germinal layer development at lobule junctions. *Journal of Neuroscience, 18*, 1763–1773.

Badner, J. A., & Gershon, E. S. (2002). Regional meta-analysis of published data supports linkage of autism with markers on chromosome 7. *Molecular Psychiatry, 7*, 56–66.

Bailey, A., Palferman, S., Heavey, L., & LeCouteur, A. (1998). Autism: The phenotype in relatives.

Journal of Autism and Developmental Disorders, 28, 369–392.

Bettelheim, B. (1967). *The empty fortress: Infantile autism and the beauty of the self*. New York: Free Press.

Blacher, J., & Baker, B. (2002). *Families and mental retardation*. Washington, DC: American Association on Mental Retardation.

Bleuler, E. (1950). *Dementia praecox or the schizophrenias*. New York: International University Presses.

Bolton, P., Macdonald, A., Pickles, P., Rios, S., Goode, M., Crowson, A., et al. (1994). A case-control family history study of autism. *Journal of Child Psychology and Psychiatry, 35*, 877–900.

Bruinsma, Y., Koegel, R. L., & Koegel, L. K. (2004). Joint attention and children with autism: A review of the literature. *Mental Retardation and Developmental Disabilities Research Reviews, 10*, 169–175.

Colombi, C., Liebal, K., Tomasello, M., Young, G., Warneken, F., & Rogers, S. J. (2009). Examining correlates of cooperation in autism: Imitation, joint attention and understanding intentions. *Autism, 13*, 143–163.

Charman, T. (1998). Specifying the nature and course of the joint attention impairment in autism in the preschool years: Implications for diagnosis and intervention. *Autism, 2*, 61–79.

Chiang, C., Soong, W., Lin, T., & Rogers, S. J. (2008). Nonverbal communication skills in young children with autism. *Journal of Autism and Developmental Disorders. 38*, 1898–1906.

Daniels, J. L., Forssen, U., Hultman, C. M., Cnattingius, S., Savitz, D. A., Feychting, M., & Sparen, P. (2008). Parental psychiatric disorders associated with autism spectrum disorders in the offspring. *Pediatrics, 121*, 1357–1362.

Di Martino, A., Ross, K., Uddin, L. Q., Skylar, A. B., Castellanos, F. X., & Milham, M. P. (2009). Functional brain correlates of social and nonsocial processes in autism spectrum disorders: An activation likelihood estimation meta-analysis. *Biological Psychiatry, 6*, 63–74.

Eisenberg, L., & Kanner, L. (1956). Early infantile autism: 1943–55. *American Journal of Orthopsychiatry, 26*, 556–566.

Fombonne, E., Bolton, P., Prior, J., Jordan, H., & Rutter, M. (1997). A family study of autism: Cognitive patterns and levels in parents and siblings. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 38*, 667–683.

Frith, U. (1991). *Autism and Asperger syndrome* (pp. 37–92). New York: Cambridge University Press.

Grether, J. K., Rosen, N. J., Smith, K. S., & Croen, L. A. (2009). Investigation of shifts in autism reporting in the California Department of Developmental Services. *Journal of Autism and Developmental Disorders, 39*, 1412–1419.

Hardan, A., Muddasani, S., Vemulapalli, M., Keshavan, M., & Minshew, N. (2006). An MRI study of increased cortical thickness in autism. *American Journal of Psychiatry, 163*, 1290–1292

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

Kanner, L. (1949). Problems of nosology and psychodynamics in early childhood autism. *American Journal of Orthopsychiatry, 19*, 416–426.

Kanner, L. (1968). "Reprint." *Acta Paedopsychiatrica, 35*, 100–136.

Land, H, (1977). *The wild boy of Aveyron*. London: Allen & Unwin.

Landa, R., Piven, J., Wzorek, M. M., & Gayle, J. O. (1992). Social language use in parents of autistic children. *Psychological Medicine, 22*, 245–254.

Lord, C., Risi, S., Lambrecht, L., Cook, E. H., Leventhal, B. L., DiLavore, P., et al. (2000). The Autism Diagnostic Observation Schedule–Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders, 30*, 205–223.

Lord, C., Rutter, M., & LeCouteur, A. (1994). Autism Diagnostic Interview–Revised: A revised version of a diagnostic interview for caregivers of children with possible pervasive developmental disorders. *Journal of Autism and Developmental Disorders, 24*, 659–685.

Losh, M., Sullivan, P., Trembath, D., & Piven, J. (2008). Current developments in the genetics of autism: From phenome to genome. *Journal of Neuropathology and Experimental Neurology, 67*, 829–837.

Macali, N., Chakrabarti, S., & Fombonne, E. (2004). The broad autism phenotype. *Autism, 8*, 21–37.

Murphy, M., Bolton, P. F., Pickles, A., Fombonne, E., Piven, J., & Rutter, M. (2000). Personality traits of the relatives of autistic probands. *Psychological Medicine, 30*, 1411–1424.

Piven, J., & Palmer, P. (1997). Cognitive deficits in parents from multiple-incidence autism families. *Journal of Child Psychology and Psychiatry, 38*, 1011–1022.

Piven, J., Palmer, P., Jacobi, D., Childress, D., & Arndt, S. (1997). The broader autism phenotype: Evidence from a family study of multiple-incidence autism families. *American Journal of Psychiatry, 154*, 185–190.

Rice, C. (2006). Prevalence of autism spectrum disorders: Autism and developmental disabilities monitoring network. Morbidity and mortality weekly report. *Surveillance Summaries*, 58, Number SS-10. Atlanta: Center for Disease Control.

Rutter, M. L. (1973). Foreword. In L. Kanner, *Childhood psychoses: Initial studies and new insights* (pp. vii–ix). Washington, DC: Winston.

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

Rutter, M. (2005). Genetic influences and autism. In F. R. Volkmar, R. Paul, A. Klin, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 425–453). Hoboken, NJ: Wiley.

Schopler, E., Chess, S., & Eisenberg, L. (1981). Our memorial to Leo Kanner. *Journal of Autism and Developmental Disorders, 11*, 257–269.

Stanfield, A. C., McIntosh, A. M., Spencer, M. D., Philip, R., Gaur, S., & Lawrie, S. M. (2008). Towards a neuroanatomy of autism: A systematic review and meta-analysis of structural magnetic resonance imaging studies. *European Psychiatry, 23*, 289–299.

Sutcliffe, J. S., & Nurmi, E. L. (2003). Genetics of childhood disorders: XLVII. autism, part 6: Duplication and inherited susceptibility of chromosome 15q11–q13b genes in autism. *Journal of the American Academy of Child & Adolescent Psychiatry, 42*, 253–256.

Ward, M. J., & Meyer, R. N. (1999). Self-determination for people with developmental disabilities and autism. *Focus on Autism and Other Developmental Disabilities, 14*, 133–139.

Waterhouse, L., Wing, L., Spitzer, R., & Siegel, B. (1992). Pervasive developmental disorders: From DSM-III to DSM-III-R. *Journal of Autism and Developmental Disorders, 22*, 525–549.

Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine, 11*, 115–129.

Wing, L. (1997). The history of ideas on autism: Legends, myths and reality. *Autism. 1*, 13–23.

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