



An Introduction to Autism

What Is It and How Do We Understand It?

WHAT IS AUTISM?

There are many ways to talk about autism. Perhaps the simplest one is to think of autism as a social learning disability. Like other “hidden” learning disabilities/differences (e.g., reading problems/dyslexia) it is not immediately apparent just by looking at the child. The reason autism is so significant is that if you don’t learn from others from the first days of life you really lose out on many important things. Given a lack of interest in people and social interaction, many early processes don’t come into play; for example, you don’t engage in **joint attention** (not looking where and at what your parents are looking at, thus missing out on what is important), you don’t engage in what is called **incidental learning** (learning by watching) or **imitation**. If you don’t “play the social game” you have trouble multitasking and organizing the world (what psychologists call **executive functioning**). Unlike other infants you don’t go rapidly back and forth between different aspects of the world, what the person is saying, doing, how the person is feeling, what the tone of voice or gestures tell you. You do not like change. You easily develop unusual interests in things—particularly if they are stable and unchanging like street signs, alphabet letters, or hood ornaments on cars. This lack of interest in others also means you have less interest in understanding what they say and feel, and as a result you have less interest in communicating and less ability to understand and communicate feelings, wants, and needs. These problems get even worse as you go to school and are expected to sit in a chair and share attention with other children focused on a teacher!

This is a simple (but not incorrect) way to start thinking about autism. Of course, things are much more complex than this, particularly when you discover that there can be a broad range of outcomes in autism—the child who sits and body rocks and rarely talks, all the way to the overly talkative adolescent who wants to talk to you about his toaster collection! The pervasiveness of autism leads to major difficulties in efficient learning. This chapter gives some background on autism and related autism spectrum disorders (ASDs) and sets the stage for more detailed explanations later on in the book

WHEN WAS AUTISM FIRST RECOGNIZED AS A DISORDER?

The recognition of autism as a disorder is a pretty recent one—the disorder was first described in 1943 but not “officially” used as a diagnosis until 1980. Other conditions such as Asperger’s disorder were “officially” recognized even more recently. Given that our understanding of autism and related

conditions has changed, you may hear many different terms used to describe your child's difficulties. Because knowledge has changed over the years there are also some misconceptions about autism that persist and that you may encounter (particularly among people who haven't kept up with the field!). Finally, if you are looking at this book you are probably wondering if a child you know has autism—we think it would be helpful to you to know something about autism and the autism spectrum.

Autism also known as **autistic disorder**, **childhood autism**, or **infantile autism** and now as **autism spectrum disorder** (all the names mean more or less the same thing!) was first described as a medical condition by Dr. Leo Kanner back in 1943. Dr. Kanner, the first child psychiatrist in the country, reported on 11 children who appeared to exhibit what he called “an inborn disturbance of affective contact.” By this he meant that, in contrast to normal babies, these children came into the world without the usual interest in other people. This was in contrast to normally developing babies for whom people are the single most interesting things in the environment. He believed that the difficulty in dealing with the social world was congenital in nature that is the children were born with it. Dr. Kanner gave a careful description of the unusual behaviors these first cases exhibited. He discussed at great length some of the unusual behaviors they exhibited (see Box 1.1).

BOX 1.1 KANNER QUOTE

The outstanding, “pathognomonic,” fundamental disorder is in the children’s *inability to relate themselves* in the ordinary way to people and situations from the beginning of life. Their parents referred to them as having always been “self-sufficient”; “like in a shell”; “happiest when left alone”; “acting as if people weren’t there”; “perfectly oblivious to everything about him”; “giving the impression of silent wisdom”; “failing to develop the usual amount of social awareness”; “acting almost as if hypnotized.” This is not, as in schizophrenic children or adults, a departure from an initially present relationship; it is not a “withdrawal” from formerly existing participation. There is from the start an *extreme autistic aloneness* that, whenever possible, disregards, ignores, shuts out anything that comes into the child from outside. Direct physical contact or such motion or noise as threatens to disrupt the aloneness is either treated “as if it weren’t there,” or, if this is longer and sufficient, resented painfully as a distressing interference.

This insistence on sameness led several children to become greatly disturbed upon the sight of anything broken or incomplete. A great part of the day was spent in demanding not only the sameness of the wording of a request but also the sameness of the sequence of events.

The dread of change and incompleteness seems to be a major factor in the explanation of the monotonous repetitiousness and the resulting *limitation in the variety of spontaneous activity*. A situation, a performance, a sentence is not regarded as complete if it is not made up of exactly the same elements that were present at the time the child was first confronted with it. If the slightest ingredient is altered or removed the total situation is no longer the same and therefore is not accepted as such, or it is resented with impatience or even with a reaction of profound frustration.

From Leo Kanner, Autistic disturbances of affective contact, *Nervous Child*, 2, 217–250, 1943.

In addition to emphasizing the importance of autism (living in your own world) he emphasized that the children exhibited “**resistance to change**.” By this, he meant that they literally had what he termed

“**insistence on sameness.**” For example, a child might require that the parents take the same route to school or church and become very upset if there was any deviation from this routine. They might be very rigid about what kinds of clothes they would wear or foods they would eat. The term “resistance to change” as used by Kanner in 1943 also refers to some of the unusual behaviors frequently seen in dealing with changes in the environment or routine; for example, the apparently purposeless motor behaviors (stereotyped movements) such as body rocking, and hand flapping, often exhibited in autism. Kanner viewed these as purposeful and a way for the child to maintain sameness, a feeling of comfort with an unchanged environment. Dr. Kanner mentioned that when language developed at all it was unusual. For example, the child with autism might fail to give the proper tone to his or her speech (that is, might speak like a robot) or might echo language (**echolalia**) or confuse personal pronouns (**pronoun reversal**). For example, when asked if he or she wanted a cookie, the child might respond “Wanna cookie, wanna cookie, wanna cookie.” Sometimes the language that was echoed was from the distant past (delayed echolalia). Sometimes it happened at once (immediate echolalia). Sometimes part of it was echoed but part had been changed (mitigated echolalia). In his original report Kanner thought there were two things essential for a diagnosis of autism: (1) the autism or social isolation and (2) the unusual behaviors and insistence on sameness.

As time went on, it became clear that language/communication problems were also important in the diagnosis (when you think about it, of course, language is an important aspect of social development!). Including these problems along with the early onset of the condition that Kanner mentioned we have what continue to be the four hallmarks of autism: (1) impaired social development of a type quite different from that in normal children, (2) impaired language and communication skills—again of a distinctive type, (3) resistance to change or insistence on sameness as reflected in inflexible adherence to routines, motor mannerisms and stereotypies, and other behavioral oddities, and (4) an onset in the first years of life.

Of course, autism existed before Kanner described it. Likely children who were described as “wild” or “feral,” presumed to have been living in the wild or raised by animals, may well have been the first children with autism. They may have been abandoned or ran away from their parents (the problem of **bolting** that we’ll talk about when we discuss safety). In their excellent history of autism, Donovan and Zuker (2016) gave examples of individuals with autism in the 1800s, before autism was recognized as a condition as such. There are some other things to know about autism’s history that are very important.

EARLY MISTAKES ABOUT AUTISM

While Kanner’s description remains a “classic” it was not the last word on the subject. Some aspects of his original report inadvertently served to mislead people. Some of these mistaken first impressions took many years to clarify. For example, Kanner originally thought that children with autism probably had normal **intelligence**. He thought this because they did rather well on some *parts* of **intelligence (IQ) tests**. On other parts, however, they did quite poorly or refused to cooperate at all. People assumed that, if they did as well on all parts of the IQ test as they did on the one or two parts on which they seemed to do well, the child would not be intellectual disabled. Unfortunately, it turns out that cognitive or intellectual skills are often difficult to assess, in large part because they are very scattered. Put another way, children with autism often do some things well, such as solving puzzles, but they may have tremendous difficulty with more language-related tasks. It is frequent to find marked discrepancies in

abilities—something not common in the typically developing population but very frequent in autism. For example, we’ve seen children with Asperger’s who have verbal abilities in the genius range on IQ tests but whose nonverbal skills are in the borderline or intellectually disabled range. As time went on it became clear that, in the earliest days of autism work, many children with autism ended up functioning in the intellectually disabled range as based on their entire performance on tests—not just their areas of greater ability. This was a time before effective, evidence-based intervention was available. Many (a majority) of these children were mute and needed considerable care. It took many years to appreciate this but for these early cases the majority ended up functioning in the range of **intellectual disability** when you combine all of their, sometimes quite variable, different scores. Maybe 10% of the time a child with autism will have an unusual ability, for example, to draw, or play music, or memorize things, or sometimes calculate days of the week for events in the past or future (calendar calculation). These abilities are usually isolated (the otherwise very wonderful portrayal of the man with autism in *Rain Man* is misleading in this respect). These individuals, now usually referred to as **autistic savants**, sometimes lose their abilities as they get older. But it was just this kind of remarkable ability that led people to minimize the child’s areas of difficulties (see Box 1.2).

BOX 1.2 EARLY MISTAKES

Early Mistaken Impressions of Autism

- **↑Increased rate in families with higher levels of educations**
Implication: Effects of experience causing autism
Subsequent research: there is no increase in autism among parents with more education
- **No associated medical conditions (children had an attractive appearance)**
Implication: Exclusion of “organic” cases as having autism
Subsequent research: High rates of seizures, higher than expected rates of some disorders—especially some genetic disorders
- **Impression of normal levels of intelligence (children did well on some parts of IQ tests)**
Implication: Bad performance due to lack of motivation of child
Subsequent research: Significant scatter in abilities is often present; marked discrepancies between skills areas (e.g., verbal and nonverbal IQ) are common
- **Autism associated with schizophrenia**
Impression: Confusion with schizophrenia given the use of the word *autism* (earlier used to describe self-centered thinking in schizophrenia)
Implication: Autism might be the earliest manifestation of schizophrenia
Subsequent research: Autism and schizophrenia are not related; occasional (no more than expected by chance) children with who autism develop schizophrenia

Another source of confusion came because Dr. Kanner originally suggested that autism was not associated with other medical conditions. We now know this is not true. Over the years, many hundreds of conditions have been reported to be related to autism, but it now seems that really only a few are especially frequent. For example, we now know that sometimes autism is seen with some genetic conditions like **Fragile X Syndrome** or **Tuberous Sclerosis**—and with advances in genetics that we’ll talk about in a later chapter, we increasingly find genes/genetic abnormalities in children with

autism and have come to appreciate how strong the genetics of autism can be. As children with autism were followed over time, it became apparent that many of them would develop **seizures (epilepsy)**, as we discuss later in the book.

Dr. Kanner originally guessed that autism was a very distinctive condition and we now know that this is true. At the same time, he used the word autism—a word which previously had been used to describe some of the unusual, self-centered, and self-contained thinking seen in a major mental disorder called **schizophrenia**. Thus, his use of the word autism suggested to many that perhaps autism was the earliest form of schizophrenia. It took many years for this to be clarified. We now know that autism and schizophrenia are not related. Very occasionally, but no more than would be expected by chance, individuals with autism may, as adolescents or adults, develop schizophrenia. Autism differs from schizophrenia, however, in many different ways, including its clinical features, course, associated difficulties, and family history.

Finally, Kanner mentioned in his 1943 paper that in 10 of the 11 families, the parent or parents were highly educated and successful. It also appeared that parents and children interacted somewhat unusually at times. This led to the idea, particularly in the 1950s, that highly successful parents somehow ignored or otherwise ill-treated their child to cause autism and that, as a result, autistic children might be well served by isolating them from their families. Over time it became very clear that this is not true. Instead, it is clear that Kanner's original sample was a highly selected one; that is, individuals who were very educated and successful in the 1940s would be just the kinds of people who could find the one person in the country who was doing research on the kinds of problems their children had. It also became clear that unusual aspects of parent-child interaction were just as likely to come from the child, rather than the parent. In contrast to the 1950s, when often the emphasis was on putting the child in an institution, we now believe that children with autism are best served by remaining in their families and communities and schools.

Finally, Dr. Kanner thought children were born with autism—i.e., that it was congenital. He did, in fact, mention a case where there had appeared to be some period of normal development and we know now that in maybe 1 in 5 cases, parents tell us that their child seemed to develop normally before autism developed. We know that most of the time there seem to be very clear warning signs of autism in the first year or so of life—the lack of a specific diagnostic marker or test complicates this issue, of course.

We also know that, very rarely, a child can develop normally to 3 or 4 years of age and then develop autism. The American Psychiatric Association's **Diagnostic and Statistical Manual of Mental Disorders** fourth edition (**DSM-IV**) had a specific term for this rare phenomenon (**childhood disintegrative disorder**). At any rate, it is clear that most children with autism appear to either have it at birth or present symptoms very early in life—with some important exceptions. Box 1.3 provides a short case history of a child with autism.

BOX 1.3 CASE REPORT: AUTISM SPECTRUM DISORDER

John was the second of two children born to middle-class parents after normal pregnancy, labor, and delivery. As an infant, John appeared undemanding and relatively placid; motor development proceeded appropriately, but language development was delayed. Although his parents indicated that they were first concerned about his development when he was 18 months

of age and still not speaking, in retrospect they noted that, in comparison to their previous child, he had seemed relatively uninterested in social interaction and the social games of infancy. Stranger anxiety had never really developed, and John did not exhibit differential attachment behaviors toward his parents. The pediatrician initially reassured John's parents that he was a "late talker," but they continued to be concerned. Although John seemed to respond to some unusual sounds, the pediatrician obtained a hearing test when John was 24 months old. Levels of hearing appeared adequate for development of speech, and John was referred for developmental evaluation. At 24 months, motor skills were age appropriate and John exhibited some nonverbal problem-solving skills close to age level. His language and social development, however, were severely delayed, and he was noted to be resistant to changes in routine and unusually sensitive to aspects of the inanimate environment. His play skills were quite limited and he used play materials in unusual and idiosyncratic ways. His older sister had a history of some learning difficulties, but the family history was otherwise negative. A comprehensive medical evaluation revealed a normal electroencephalogram (or EEG) and CT scan; genetic screening and chromosome analysis were normal as well.

John was enrolled in a special education program, where he gradually began to speak. His speech was characterized by echolalia, extreme literalness, a monotonic voice quality, and pronoun reversal. He rarely used language in interaction and remained quite isolated. By school age, John had developed some evidence of differential attachments to family members; he also had developed a number of self-stimulatory behaviors and engaged in occasional periods of head banging. Extreme sensitivity to change continued. Intelligence testing revealed marked scatter, with a full-scale IQ in the intellectually deficient range. As an adolescent, John's behavioral functioning deteriorated, and he developed a seizure disorder. Now an adult, he lives in a group home and attends a sheltered workshop. He has a rather passive interactional style but exhibits occasional outbursts of aggression and self-abuse.

Comment: With earlier intervention more children with autism are doing better. Unfortunately, in this case although the child developed speech, his overall outcome has not been as good as might have been hoped. Adapted from F. Volkmar, Lord, C., Klin, A., and E. Cook. (2002). Autism and pervasive developmental disorders. In M. Lewis (Ed.), *Child and adolescent psychiatry: A comprehensive textbook* (p. 595). Lippincott.

ASPERGER'S AND THE BROADER SPECTRUM OF AUTISM

The year after Kanner reported on autism, Hans Asperger, a medical student in Vienna, Austria, reported on a group of boys who had trouble joining social groups. In contrast to Kanner's report, these children were very verbal and focused and talked about their special interests (rocks, American gangsters, train schedules, etc.) incessantly. Asperger made the important point that these intense special interests interfered with the child's learning. He also noted that these interests tended to take over the family, for example, they'd have to go to the train station every day to be sure the 6:15 p.m. train was on time. He said these boys were motorically clumsy and noted similar problems in some families. Problems in social interaction, particularly with peers, were very significant and he termed the condition *autistic personality disorder*, using the same word—autism—that Kanner had used. Effectively, this report set the stage for

continuing debate. Asperger's was officially recognized as a diagnostic term from 1994 (in the DSM-IV) until DSM-5 when it was dropped (although individuals with well-established diagnoses of Asperger's could keep the diagnosis!).

We'll talk more about Asperger's shortly but essentially the realization that some individuals have major problems in social interaction, sometimes with special interests that don't fit so neatly with Kanner's first description, raises the question of broad vs. narrow views of autism. Despite the welcome name change in DSM-5 to autism spectrum disorder, in fact, the definition provided (as we'll discuss) is really a narrower view, like that of Kanner. But what about the cases that don't fit this narrow view?

It has been clear for decades that many children (and then adolescents and adults) have problems in social interaction but don't have more classic autism. There has been increasing interest in the broader range of autism (often technically called the broader autism phenotype). In many ways this broader view is more consistent with the growing body of work on the complex genetics of autism. This has been a major focus of discussion in light of the awareness of the complex genetics of autism. Box 1.4 provides a short case history of a child with Asperger's.

BOX 1.4 CASE OF ASPERGER'S

Case Report: Asperger's Disorder

Tom was an only child. Birth, medical, and family histories were unremarkable. His motor development was somewhat delayed, but communicative milestones were within normal limits. His parents became concerned about him at age 4 when he was enrolled in a nursery school and was noted to have marked difficulties in peer interaction that were so pronounced he could not continue in the program. In grade school, he was enrolled in special education classes and was noted to have some learning problems. His greatest difficulties arose in peer interaction; he was viewed as markedly eccentric and had no friends. His preferred activity, watching the weather channel on television, was pursued with great interest and intensity. On examination at age 13, he had markedly circumscribed interests and exhibited pedantic and odd patterns of communication with a monotonic voice quality. Psychological testing revealed an IQ within the normal range with marked scatter evident, with verbal abilities much higher than nonverbal ones. Formal communication examination revealed age-appropriate skills in receptive and expressive language, but marked impairment in pragmatic language skills. Tom has now gone on to college where he has, with considerable support, done well. He is currently employed as a computer programmer.

Comment: Preservation of language (if not always communication) skills in Asperger's presents some important strengths for treatment. There is a related issue of **neurodiversity**. The neurodiversity movement would tend to see all these things less as a disorder but rather a different way of viewing and interacting with people. This is a complex issue in and of itself but it is important to realize that some individuals, particularly those who function at the highest levels, would rather see themselves as different rather than disordered. The book *NeuroTribes* by Silberman addresses these complex issues and raises other issues.

WHY DID IT TAKE SO LONG TO RECOGNIZE AUTISM AS AN OFFICIAL “CONDITION”?

Autism was first described in 1943. Why did it take so long (1980) before autism was officially considered a diagnosis in the U.S.? In the first place, the early editions of the DSM guidebook to psychiatric diagnoses were not very helpful—they were theory driven rather than focused on the clinical phenomena. Only the term **childhood schizophrenia** was available to describe children with severe disabilities (other than intellectual disability) and, as we mentioned before, there was some early confusion around the word *autism* that had been used much earlier to describe self-centered thinking (not social problems) in adult schizophrenia. During the 1970s, several important lines of evidence made it clear that autism needed to be recognized in its own right:

1. Autism was clearly a brain-based disorder—with a high risk for developing seizures.
2. Children with autism was strongly genetic with a much greater risk for autism in identical, as opposed to fraternal, twins.
3. Autism responded best to structured teaching.
4. Children with autism did not exhibit features of schizophrenia. Rather, they exhibited a distinctive pattern of behavior and development from very early in life; in point of fact schizophrenia in children (before adolescence) is very rare.

All these lines of evidence supported the inclusion of autism within the new and totally revamped guidebook DSM-III in 1980.

NAMES FOR AUTISM AND RELATED CONDITIONS

Autism was first recognized as “**infantile autism**” in the DSM-III; a new term “**residual infantile autism**” was available for persons who had once met the strict list of diagnostic features or criteria for infantile autism but no longer did so. Over time there have been various changes in the way autism is termed and defined. The current term “**autism spectrum disorder**” was recently officially recognized by the American Psychiatric Association in 2013 in the 5th revision of its guide to diagnosis, the DSM-5. This term replaced the older term “**pervasive developmental disorder**” (**PDD**) that had been used from 1980 to 2013 to refer to the entire group of conditions, including autism. This was a term for the class of disorder to which autism belongs—autism is a kind of PDD like apples are a kind of fruit. The DSM-5 also dropped a number of other terms that had been included with autism, including Asperger’s Disorder and **pervasive development disorder not otherwise specified** (**PDD-NOS** or **atypical autism**). The PDD-NOS term had been used for several decades in one way or another to describe children who needed services but didn’t quite fit the autism label. The decision to drop both categories has been rather controversial. As we’ll discuss in more detail in Chapter 3, the current DSM-5 definition is more stringent than the previous one and because having a label is so closely tied (in the U.S.) with services, this can be problematic. DSM-5 did include a new category for a **social communication disorder** (**SCD**) but this category is not simply the same as either the older Asperger’s concept or PDD-NOS. We will talk more about some of the issues of diagnosis in the next chapter.

This gets us to the question of what an official diagnosis is. In the United States the DSM—the *Diagnostic and Statistical Manual* of the American Psychiatric Association—is the one most frequently used, and it is now in its 5th edition (DSM-5). This book is used for both clinical and research purposes. In contrast, there is an international system (also used in the U.S. and around the world) called the ***International Classification of Diseases, 11th edition (ICD-11)***. These systems are similar in some ways and different in others. Probably the major difference is that the ICD has two different guidelines, one for research and the other for clinical work; it also makes some differences and distinctions on subtypes based on presence/absence of functional communication and/or intellectual disability. Other diagnostic systems exist as well but these two are the major ones. These books give guidelines to physicians and other health care providers about diagnoses. We will talk more about the uses and limitations of these diagnostic systems in the next chapter.

A big complexity for autism is that, at least at present, there is no simple blood or laboratory test that can establish whether a child does or doesn't have autism. A search for such markers (the technical term is **biomarkers**) is underway and these may be helpful in simplifying the diagnosis in the future. One of these is the use of the EEG or brain wave response to viewing human faces. As we discuss later on in this chapter, much work has now been done on understanding the social brain in autism and this may be of value in developing new diagnostic approaches. We know now of a number of genetic risk factors for autism, and genetic testing has taken on an important role in medical assessments with some genetic differences/conditions identified between maybe 10% and 20% of the time (although sometimes the importance of any small genetic difference remains unclear!).

At present, doctors (both clinicians and researchers) have to focus more on the history and symptoms presented by the child. In Chapter 2, we'll talk about some screening tools that have been designed to help parents, teachers, and health-care providers look for early warning signs of autism. The various terms used for autism are listed in Box 1.5

BOX 1.5 TERMS USED FOR AUTISM AND RELATED DISORDERS

Current Name	Other Names for Essentially the Same Thing
Autism spectrum disorder	Autistic disorder , childhood autism, infantile autism, early infantile autism, residual infantile autism.
Asperger's disorder	Asperger's disorder (no longer an official term in DSM-5 but grandfathered in from DSM-IV for people with "well-established" diagnoses), Asperger's syndrome , autistic psychopathy , autistic personality disorder .
Childhood disintegrative disorder	Heller's syndrome , disintegrative disorder, disintegrative psychosis. No longer recognized in DSM-5.
Broader autism phenotype (BAP)	Atypical autism , pervasive developmental disorder not otherwise specified , atypical PDD , atypical personality . No longer specifically recognized in DSM-5 but "grandfathered" in for well-established cases.

Social communication disorder

A new disorder (in the communication section of DSM-5). This may overlap somewhat with the broader autism phenotype although this is a communication disorder rather than an autism spectrum disorder in the DSM-5.

SERVICES FOR CHILDREN WITH AUTISM

Until the passage of the Education for All Handicapped Children Act in 1975 (and its various successors) parents of children with autism often were at a loss as to how to educate them. Research began to suggest that structured educational programs were more effective than unstructured ones; that is, programs in which the adult had an agenda for teaching the child were better than if the child were left to his or her own devices to learn on their own. Before 1975, parents were often told by schools that there was no way their child could be educated. Often, parents were advised to place their child in a residential or large state institution where the child got little in the way of intervention.

Now schools in the U.S. are mandated to provide a free and appropriate education for all individuals with disabilities. This is a radically different approach. As programs have become more sophisticated, schools have done an increasingly better job of providing education for children with autism. This means that schools are often now the major focus of intervention for children with autism. As a result, it appears that more children are being identified in schools and receiving services and, importantly, it also seems like, as a group, children with autism are doing better. In subsequent chapters, we'll be discussing these issues and specific programs in greater detail.

HOW COMMON ARE AUTISM AND RELATED CONDITIONS?

The first studies of the frequency or epidemiology of autism were conducted in the 1960s. Since that time many studies have been conducted. The most recent comprehensive review of the topic by Myers and colleagues (in 2019) suggests a rate of 1 in 145 children (and also notes the many problems in summarizing the available studies). Studies have been conducted in many countries and there are some important issues in diagnosis, including culture, ethnicity, and gender.

Is the Rate of Autism Increasing?

There is some concern that the rate of autism may be increasing as more recent studies tend to report higher rates. However, we really don't know if this is true for several reasons. First, it is clear that awareness of autism has increased dramatically, so cases are more likely to be noticed. When one of us (FV) moved to New Haven in 1980 to work on autism at the Yale Child Study Center with Donald Cohen, people would ask what my research was about and when I said, "Autism," they would frequently say something like, "Isn't that wonderful—we need more artistic children"; people didn't even know what the word meant! Today, there are ads from the Ad Council about autism on radio and television and posted in the background of TV shows advertising autism-related groups. Another possible reason for an apparent (but not real) increase is changes in the diagnostic guidelines for autism—the current systems (both DSM and

ICD) were designed to do a better job of detecting autism in more able children. Another problem has been the tendency to equate autism (strictly defined) with the much broader and much less well-defined autism “spectrum.” Finally, there is an unusual problem with autism, since the label often gets children more services than other labels; i.e., parents may push to get an autism label for educational purposes even if the child might be said to have something else (this is a problem called **diagnostic substitution** and one of the reasons we have to be skeptical about state-reported data based on school services). This is a real problem because states, and sometimes regions within states, vary widely (and wildly) in terms of how they provide services—in some states only the label of autism really gets needed services. This can also be a problem, as many higher cognitively able children are now being diagnosed, and many parents want a label of autism/PDD to get their child intensive educational services.

Sex Differences

It is clear that autism appears to be at least 3 to 5 times more frequent in boys. On the other hand, when girls have autism, they are more likely to have intellectual disabilities. We do not yet understand the basis for these differences. One theory is that, perhaps on a genetic basis, girls are generally somewhat less vulnerable to autism (hence the greater frequency in boys) and that for girls to have autism they must have greater genetic or central nervous system damage (hence the higher rate of intellectual disability in girls). This gender discrepancy is also noted in Asperger’s, but there does not appear to be a marked sex difference in the broader autism spectrum (what used to be termed PDD-NOS). A current focus of much research is whether girls may exhibit a milder version of autism and be part of the “broader autism phenotype.”

WHAT CAUSES AUTISM?

As we mentioned earlier, Kanner’s first paper on autism was very influential—in both good and bad ways. The good ways had to do with the unusually clear way he described what he saw in autism (problems in social interaction and unusual responses to the environment). He was also clear in suggesting that autism was congenital—that is, children were born with it. On the other hand, the bad thing about his description is that it included some of his speculations that proved incorrect as we discussed at the beginning of this chapter. Beginning in the 1960s, and particularly in the 1970s, research began to show that autism was a brain-based disorder with a strong genetic basis.

SEIZURE DISORDERS AND EEG ABNORMALITIES

One of the important things that happened to help doctors realize parents weren’t to blame for autism was an increasing awareness of the higher than expected risk children had for developing seizures. **Seizures disorders** (also referred to as **epilepsy** or as **convulsions**) are a group of conditions all of which result from abnormal electrical activity in the brain. The symptoms of seizure disorder are quite varied. They can range from brief episodes when the child seems to “tune out” to much more obvious convulsions when the child falls to the ground, loses consciousness, and has alternating periods of muscle contraction and relaxation. There are many different kinds of epilepsy as we discuss later.

One of the ways doctors look for seizure activity is through the EEG, which measures electrical activity in the brain. Both early and more recent studies suggest that as many as 50% of individuals with autism have abnormalities in their EEG; findings on the EEG are diverse and not specific to autism but the higher rates of abnormality are, of course, suggestive of some basic problem with how the brain is “wired.” Even more dramatic are the rates of seizure disorder in autism. In the “normal” population of children, rates of first seizure are highest around the time of birth and then greatly decrease over time. Figure 1.1 presents information on times when epilepsy (recurrent seizures) develop. These data are from two studies of children with autism and PDD-NOS as well as data from a large sample of normally developing British children. There are higher rates for first seizures in children with autism than in the normal population of children without autism.

Onset of seizures in autism

Rates of first seizure (excluding febrile seizures)

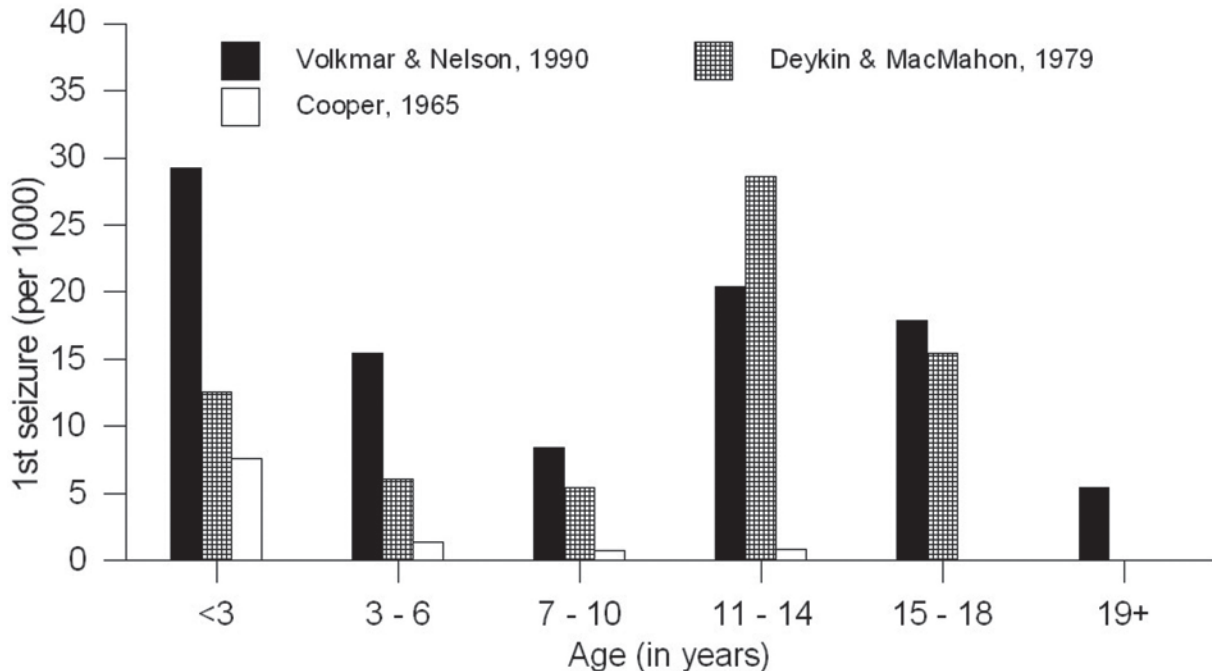


FIGURE 1.1

Rates of first seizure and epilepsy in two samples of individuals with autism (Volkmar & Nelson, 1990; Deykin & MacMahon, 1979) and a British sample of typically developing children (Cooper, 1965).

OTHER NEUROLOGICAL FEATURES

A number of other neurological problems are observed in autism. Again, these are of many different types; not every child has every sign and some children will have none. Some children with autism have delays in the development of such things as hand dominance (they may draw with either hand without having a preference for right or left). They can also have general decreases in muscle tone in the body and be somewhat “floppy” as babies (technically, this is called “**hypotonia**”). Sometimes individuals with autism have unusual reflexes; often these are reflexes that are usually seen only in very young babies but can persist into adulthood in individuals with autism. For example, with a newborn, if the doctor brings his reflex hammer toward the baby’s mouth, the baby may start to suck, as if anticipating the bottle or breast; this *visual rooting reflex* is sometimes seen even in adults with autism, whereas in most of us it disappears very early in childhood. Other problems may be seen in the way that individuals with autism walk, or with their posture.

NEUROCHEMISTRY

Nerve cells use different kinds of chemicals to communicate with each other. A number of these systems have been studied in autism and there is some suggestion of alterations in these systems in autism. Probably the most work has centered on the chemical **serotonin** (also sometimes referred to as 5-HT or 5-Hydroxytryptamine). A number of studies have shown that levels of serotonin in the blood (not necessarily in the brain) are often increased in individuals with autism. Unfortunately, the relationship between blood levels and brain levels of this chemical is not always clear. Other studies have focused on the chemical **dopamine**, which is involved in parts of the brain that control movement, and is part of a broader system that relates to levels of arousal. Many of the drugs used to treat symptoms of autism affect these chemicals (see Chapter 11).

NEUROANATOMY AND BRAIN IMAGING STUDIES

Various methods can be used to study the brain. These range from actual studies of brain tissues obtained at the time of death (postmortem studies) to studies of the living and active brain through **functional magnetic resonance imaging (fMRI)**. A number of findings deserve mention. Both autopsy and brain imaging studies have suggested that at least some individuals with autism have increased brain size. Several studies have suggested the possibility that there are some alterations in brain structure—particularly in those parts of the brain that process more emotional or social thinking, perception, and responding.

In the last several years a number of interesting findings have emerged from studies of functional neuroimaging in autism. A paper from our (Yale) group documented that children with autism and Asperger’s syndrome seemed to process the information in faces differently in the brain—basically, they don’t “see” faces in the special part of the brain that the rest of us use. Another, possibly related, finding is that higher functioning individuals with autism tend to look at mouths—rather than eyes and the upper parts of the face—when watching very intense social interactions. Similarly, other work,



FIGURE 1.2

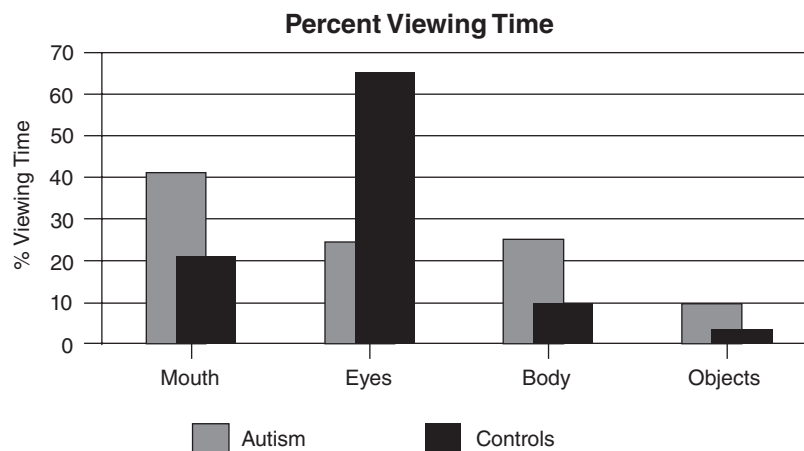
Visual focus of a man with autism (bottom line) as compared to a typically developing viewer observing a short clip from the movie class “Who’s Afraid of Virginia Woolf”. The typically developing person goes back and forth between the eyes in viewing a social scene; the high-functioning person with autism goes back and forth between the mouths of the speakers missing much of the social-emotional meaning in the scene.

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now being pursued as a potential biomarker for autism, notes that viewers with autism have, on EEG recordings, different times and patterns of looking at faces. This tells us that individuals with autism use different brain mechanisms and systems to process social information. See Figures 1.2 and 1.3.

GENETICS

As scientists began to look into the issue of the genetics of autism it became apparent that rates of autism were increased in the brothers and sisters of children with autism. Rates reported vary between

**FIGURE 1.3**

Data adapted, with permission, from A. Klin, W. Jones, R. Schultz, F. Volkmar, & D. Cohen. (2002). *Archives of General Psychiatry*, 59(9), 809–816. Percent viewing time spent focused on mouth region, eye region, and body region for viewers with and without autism. All differences are significant. Visual fixation patterns during viewing naturalistic social situations as predictors of social competence in individuals with autism.

1 in 10 and 1 in 50. This does not seem like a very high rate *unless* one realizes that the rate of autism is around 1 in 150 or so; although autism is by no means common in siblings of autistic children, the rate *is* clearly increased relative to the general population. It is clear that the role of genetics in autism is very strong and, that said, there is also some role for environmental factors (perhaps interacting with genetic factors) as well.

Autism is associated with some genetic conditions and genetic factors may be the most significant cause for the autism spectrum. As research has increased in its sophistication and we have been able to use very large samples of individuals with ASD, we've become aware of a range of genetic risk factors. These can include new mutations (called *de novo* mutations) in the individual. This could include mutations passed from older fathers (since sperm are produced through a man's life, they are more prone to contain mutations as men age) to their children. The growing sophistication of genetic work also made us aware of the potential contribution of copy number variations (CNVs)—losses or duplications of parts of the DNA. Other mutations may deactivate, or partially deactivate, genes in their functioning. Many of the genes identified may relate to certain aspects of autism or aspects of brain function, for example, nerve to nerve connections. The thought that many different genes or genetic mechanisms are involved is also supported by the observation that even when brothers or sisters do not have autism, they may be at increased risk for language and learning difficulties.

Still other factors, called epigenetic factors, might also be involved in autism. Epigenetic changes might be based on exposure to drugs, toxins, or other environmental factors, as we discuss shortly. Certain drugs are well known to have negative effects on the growth and development of the developing fetus. Congenital rubella was, at one time, thought to be linked with autism. But this congenital infection is so devastating it can be difficult to sort out the autism from the general and widespread array of problems. Certain parts of the human chromosome have seemed particularly sensitive to such

effects. In sum, there is clearly some potential role for environmental factors in autism but so far the work on genetic risk factors is much more advanced.

Although research has increasingly highlighted the importance of genetic factors in autism, final answers are not yet in. The genetics of autism is complex and it seems like multiple genes are probably involved; it is estimated that hundreds, perhaps up to a thousand, genes may be involved. To make life even more complicated, it may also be the case that not all forms of autism have the same (or any) genetic basis but might occur for other reasons; for example, there might be a specific problem at the moment of conception when some genetic material might be lost or a genetic change (mutation) might occur. There is also a suggestion that experience broadly defined (e.g., in the form of early birth difficulties) might interact with genetic predisposition to cause autism. Major efforts are now underway to identify potential genes in autism.

ENVIRONMENTAL RISK FACTORS

Although the first twin studies found a very strong genetic component, there was also some potential role for environmental factors (broadly defined) in causing autism—perhaps in association with specific genes. As noted above, these could include a range of things during the pregnancy—exposure to drugs, toxins, and other risks of pregnancy. Much interest has centered on the issue of whether autism could be caused by problems during pregnancy, labor, and delivery. A number of studies have looked at this question. Generally, they have employed some rating scale that looks at the degree of risk during the pregnancy and/or during labor and delivery. Early studies seemed to show that there was an increased risk based on the use of these rating scales. Factors that seemed to be associated with increased risk for autism included older age of the parent, prematurity, and some other problems during labor and delivery. Of course, it would be just as reasonable to assume that if there was something wrong with the child from the moment of conception, there might be problems at birth. Thus, it would be just as reasonable to assume that problems in the child cause difficulties in the pregnancy. The growing body of work on genetic factors in autism, which is discussed shortly, would be most consistent with this idea. Although a handful of studies suggest some potential increased risks, the largest studies of studies (that is, what are termed **meta-analytic studies**) haven't consistently produced a positive result. And, of course, it is clear that horrendous difficulties during labor and delivery, particularly when associated with severe fetal distress, won't help any child—and have the potential to cause further trouble for a child who was going to have autism.

PSYCHOLOGICAL MODELS OF AUTISM

The very earliest attempts to develop psychological models of autism were based on the misguided idea that experience and parental care caused autism. In the 1970s, it became clear this was fundamentally mistaken but over the subsequent decades various attempts have been made to develop psychological models or theories of how autism works in the psychological sense, i.e., of how individuals learn, remember, and respond. Their attempts are important because they help guide theory and research studies which, we hope, can advance treatment approaches and methods. Parents may hear about some of these referred to in passing, so it is good to know a little bit about them.

These approaches all have their pros and cons and none has, at least as yet, emerged as the “winner.” At present, they all have something to offer in terms of alternative models of how we might understand autism.

The **theory of mind** approach has emphasized the idea that there is a basic problem for children with autism in empathizing with others, that is, having a “theory of mind,” or theory of what motivations, intentions, and so on, impact on the behavior of others. This approach, first proposed by Simon Baron-Cohen (see reading list) has been remarkably productive in terms of research. The simplicity and elegance of this theory have added to its attractiveness. There are, however, two problems with this model. One is that the severe difficulties in social interaction impact behaviors seen in very, very young children—children of a few weeks of age. This is a time well before the ability to “put yourself into the other’s place” has really developed. Another problem is that many higher functioning individuals on the autism spectrum can do “theory of mind” tasks just fine, and yet these individuals are still very socially disabled.

Another approach, termed the **executive dysfunction hypothesis** (ED), emphasizes deficits in “executive functions” (a topic we discuss in greater detail in Chapter 6). The notion of executive functions refers, basically, to the whole range of abilities involved in planning and organization. For example, seeing the multiple steps involved in a complicated task, plotting a solution in terms of getting to the desired result, keeping the desired result in mind, and being able to work out alternatives when this is needed. Within this view, autism is related to difficulties in dealing with change and a tendency to engage in **repetitive behavior** and **perseveration** as well as to problems in developing planning and problem-solving abilities due to a lack of coordinated reasoning and ongoing adjustment to feedback. As we discuss later in this book, there is no question that children with autism spectrum disorders often have severe problems in this area. From the point of view of a more general theory, however, there are some difficulties. Probably most importantly, difficulties in this area are not unique and specific to autism; that is, children with attention deficit hyperactivity disorder also have problems with organization (but don’t have social troubles of the same type seen in autism).

A somewhat different theory proposes that the difficulties in autism relate to “**weak central coherence**.” The idea here is that people with autism have trouble getting the “big picture” issue (Happé et al., 2001); they don’t see the interconnections of things—a “not seeing the forest for the trees” problem. This theory would account for some of the people with autism who are gifted in one area but very deficient in another area. Although very attractive in many ways, the experimental evidence has been somewhat weak and contradictory. Other approaches, for example, Klin and colleagues (2003) focus more on the social difficulties being a primary cause of autism, with many of the symptoms arising from the limited interest in people and the negative consequences on brain and psychological development.

UNDERSTANDING THE CAUSES OF CONDITIONS RELATED TO AUTISM

Our understanding of the causes of conditions maybe related to autism is not as advanced as in autism—with the major exception of Rett’s disorder. Again, we understand that all these conditions have a basis in problems in the brain. This is suggested by such things as rates of seizure disorder and, occasionally, other abnormalities as well. The role of genetic factors in **Rett’s syndrome** is now clearly established, as a gene has been found to be involved. It is interesting that in the original description of Asperger’s

disorder, Asperger himself noted that the father often had similar problems in social interactions. This has been supported by some research but, to date, studies of the genetics of Asperger's have been limited.

SUMMARY

This chapter has given some background information on autism and related conditions. All these conditions share impairment in social interaction as a major feature. Autism/autism spectrum disorder are the terms that people know best. As a result of changes in terminology over the years other terms like Asperger's, pervasive developmental disorder-NOS, and the broader autism phenotype have emerged as well. These share important similarities to autism but differ from each other in various ways. We know that as we have developed better treatments and early diagnosis, it does appear that outcome is improving. We also know that autism is frequently associated with evidence of brain impairment, such as seizure disorders, and parents (and doctors) should be alert to the possibility of a child developing seizures. We know much more about how the social brain works both in typically developing children and those with ASD. Differences in social information processing likely are a major component of the problems in development and learning we see in autism—essentially these present areas we must “work around” in helping the child learn from people.

We have also now come to appreciate that genetic factors are very much involved in autism. In some ways this has turned out to be a surprise, since early work did not seem to suggest a strong genetic basis. But this early work did not take into account the frequency of autism and only when the first studies of twins were done was the possible genetic basis of autism recognized. Studies of twins showed that if the twins were identical (with exactly the same genetic make-up) and if one had autism, there was a very high chance the other twin would as well; if the twins were “fraternal” (not exactly the same genetic make-up but sharing as many genes as any siblings would) the rate was much lower. As time went on, it also became clear that a range of other problems—in language and learning and social interaction—might be inherited. Active research around the world is being conducted to look for the genes that cause autism.

■ REFERENCES AND SUGGESTED READING

- Altevogt, B. M., Hanson, S. L., & Leshner, A. I. (2008). Autism and the environment: Challenges and opportunities for research. *Pediatrics*, 121, 1225–3000.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). American Psychiatric Press.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual (DSM-5)* (5th ed.). American Psychiatric Press.
- Anderson, G. M., & Hoshino, Y. (2005). Neurochemical studies of autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 453–472). Wiley.
- Asperger, H. (1944). Die “autistischen psychopathen” im Kindersalter. In U. Frith (Ed.), *Archive für psychiatrie und Nervenkrankheiten* (pp.117, 76–136). Reprinted (in part) in (1991). *Autism and Asperger syndrome*. Cambridge University Press, 1991.
- Attwood, T. (2006). *The complete guide to Asperger's syndrome*. Jessica Kingsley.

- Baron-Cohen, S. (1995). *Mindblindness*. MIT Press.
- Baron-Cohen, S. (2003). *The essential difference: The truth about the male and female brain*. Basic Books.
- Baron-Cohen, S. (2004). *The essential difference: Male and female brains and the truth about autism*. Basic Books.
- Baron-Cohen, S., Tager-Flusberg, H., & Cohen, D. (Eds.). (2000). *Understanding other minds: Perspectives from developmental neuroscience* (2nd ed., pp. 357–388). Oxford University Press.
- Bashe, P. R., & Kirby, B. L. (2001). *The OASIS guide to Asperger syndrome*. Crown.
- Cohen, D. J., & Volkmar, F. R. (1997). *Handbook of autism and pervasive developmental disorders* (2nd ed.). Wiley.
- Donvan, J., & Zuker, C. (2016). *In a different key: The story of Autism*. Penguin/Random House.
- Exkorn, K. (2005). *The autism sourcebook: Everything you need to know about diagnosis, treatment, coping, and healing*. Regan Books.
- Folstein, S. E., & Rutter, M. (1977). Genetic influences in infantile autism. *Nature*, 265, 726–728.
- Fombonne, E. (2005). Epidemiological studies of pervasive developmental disorders. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 42–69). Wiley.
- Frith, U., & Hill, E. (Eds.). (2004). *Autism: Mind and brain*. Oxford University Press.
- Grinker, R. R. (2007). *Unstrange minds: Remapping the world of autism*. Basic Books.
- Happé, F. (2005). The weak central coherence account of autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 640–649). Wiley.
- Happé, F., Briskman, J., & Frith, U. (2001). Exploring the cognitive phenotype of autism: Weak “central coherence” in parents and siblings of children with autism: I. experimental tests. *Journal of Child Psychology and Psychiatry*, 42(3), 299–307.
- Hermelin, B. (2001). *Bright splinters of the mind: A personal story of research with autistic savants*. Jessica Kingsley.
- Howlin, P. (1998). *Children with autism and Asperger syndrome: A guide for practitioners and carers*. Wiley.
- Huerta, M., Bishop, S. L., Duncan, A., Hus, V., & Lord, C. (2012). Application of DSM-5 criteria for autism spectrum disorder to three samples of children with DSM-IV diagnoses of pervasive developmental disorders. *American Journal of Psychiatry*, 169(10), 1056–1064.
- Ingersoll, B., & Wainer, A. (2014). The broader Autism phenotype. In F. R. Volkmar, R. R. Paul, S. J. Rogers, & K. A. Pelphrey (Eds.), *Handbook of autism and pervasive developmental disorders* (4th ed.). Wiley.
- Jackson, S. L. J., & F. R. Volkmar (2019). Diagnosis and definition of autism and other pervasive developmental disorders. In F. R. Volkmar (Ed.), *Autism and pervasive developmental disorders* (pp.1–24). Cambridge University Press.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Klin, A., Jones, W., Schultz, R., & Volkmar, F. (2003). The enactive mind—from actions to cognition: Lessons from autism. *Philosophical Transactions of the Royal Society, Biological Sciences*, 358, 345–360.
- Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. J. (2002). Defining and quantifying the social phenotype in autism. *American Journal of Psychiatry*, 159(6), 895–908.
- Klin, A., McPartland, J., & Volkmar, F. R. (2005). Asperger syndrome. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 88–125). Wiley.
- Klin, A., Sparrow, S. S., & Volkmar, F. R. (Eds.). (2000). *Asperger syndrome*. Guilford Press.
- Mesibov, G. B., Adams, L. W., & Klinger, L. G. (1997). *Autism: Understanding the disorder*. Kluwer Academic/Plenum Publishers.
- Mesibov, G. B., Shea, V., & Adams, L. W. (2001). *Understanding Asperger syndrome and high functioning autism*. Kluwer Academic/Plenum Publishers.
- Minshew, N. J., Sweeney, J. A., Bauman, M. L., & Webb, S. J. (2005). Neurologic aspects of autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 453–472). Wiley.
- Myers, S. M., Voigt, R. G., Colligan, R. C., Weaver, A. L., Storlie, C. B., Stoeckel, R. E., Port, J. D., & Katusic, S. K. (2019). Autism spectrum disorder: Incidence and time trends over two decades in a population-based birth cohort. *Journal of autism and developmental disorders*, 49(4), 1455–1474. <https://doi.org/10.1007/s10803-018-3834-0>
- Neisworth, J. T., & Wolfe, P. S. (2005). *The autism encyclopedia*. Brookes.
- Odom, S. L., Morin, K., Savage, M., & Tomaszewski, B. (2019). Behavioral and educational interventions. In F. Volkmar (Ed.), *Autism and pervasive developmental disorders* (pp. 176–190). Cambridge University Press.

- Ozonoff, S., Rogers, S. J., & Hendren, R. O. (2003). *Autism spectrum disorders: A research review for practitioners*. American Psychiatric Press.
- Ozonoff, S., South, M., & Provençal, S. (2005). Executive functions. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 606–627). Wiley.
- Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 37, 51–87.
- Powers, M. D. (2000). *Children with autism: A parent's guide* (2nd ed.). Woodbine House.
- Powers, M. D., & Poland, J. (2003). *Asperger syndrome and your child: A parent's guide*. HarperCollins.
- Romanowski-Bashe, P., Kirby, B. L., Baron-Cohen, S., & Attwood, T. (2005). *The OASIS guide to Asperger syndrome: Completely revised and updated: Advice, support, insight, and inspiration*. Crown.
- Russell, J. (1997). *Autism as an executive disorder*. Oxford University Press.
- Rutter, M. (2005a). Aetiology of autism: Findings and questions. *Journal of Intellectual Disability Research*, 49(4), 231–238.
- Rutter, M. (2005b). Genetic influences and autism. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 425–452). Wiley.
- Schreibman, L. (2005). *The science and fiction of autism*. Harvard University Press.
- Schultz, R. T., & Robbins, D. L. (2005). Functional neuroimaging studies of autism spectrum disorders. In F. R. Volkmar, A. Klin, R. Paul, & D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (Vol. 1, pp. 515–533). Wiley.
- Siegel, B. (1998). *The world of the autistic child: Understanding and treating autism spectrum disorders*. Oxford University Press.
- Silberman, A. C. (2015). *NeuroTribes: The legacy of autism and the future of neurodiversity*. Penguin Random House.
- Smith, I. C., Reichow, B., & Volkmar, F. R. (2015). The effects of DSM-5 criteria on number of individuals diagnosed with autism spectrum disorder: A systematic review. *Journal of Autism and Developmental Disorders*, 45(8), 2541–2552.
- Szatmari, P. (2004). *A mind apart: Understanding children with autism and Asperger syndrome*. Guilford Press.
- Thompson, T. (2007). *Making sense of autism*. Brookes.
- Towbin, K. (2005). Pervasive developmental disorder not otherwise specified. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 165–200). Wiley.
- Van Acker, R., Loncola, J. A., & Van Acker, E. Y. (2005). Rett syndrome: A pervasive developmental disorder. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 126–164). Wiley.
- Vivanti, G., Yerys, B. E., & Salomone, E. (2019). Psychological factors in autism spectrum disorders. In F. R. Volkmar (Ed.), *Autism and pervasive developmental disorders* (pp. 61–89). Cambridge University Press.
- Volkmar, F. R. (Ed.). (2007). *Autism and pervasive developmental disorders* (rev. ed.). Cambridge University Press.
- Volkmar, F. R., Klin, A., & Pauls, D. (1998). Nosological and genetic aspects of Asperger syndrome. *Journal of Autism and Developmental Disorders*, 28, 457–463.
- Volkmar, F. R., Koenig, K., & State, M. (2005). Childhood disintegrative disorder. In F. R. Volkmar, R. Paul, A. Klin, & D. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders* (3rd ed., pp. 70–86). Wiley.
- Volkmar, F. R., Paul, R., Klin, A., & Cohen, D. (Eds.). (2005). *Handbook of autism and pervasive developmental disorders* (3rd ed.). Wiley.
- Weber, J. (2000). *Children with fragile X syndrome: A parents' guide*. Woodbine House.
- Wetherby, A. M., & Prizant, B. M. (2000). *Autism spectrum disorders: A transactional developmental perspective*. Brookes.
- Whitman, T. L. (2004). *The development of autism: A self-regulatory perspective*. Jessica Kingsley.
- Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine*, 11(1), 115–129.
- Wing, L. (2001). *The autistic spectrum: A parent's guide to understanding and helping your child*. Ulysses Press.
- Wing, L., & Potter, D. (2002, August). The epidemiology of autistic spectrum disorders: Is the prevalence rising? *Intellectual Disability & Developmental Disabilities Research Reviews*, 8(3), 151–161.
- Yuenn, R. K. C., Szatmari, P., & Vorstman, J. A. S. (2019). The genetics of autism spectrum disorders. In F. R. Volkmar, *Autism and pervasive developmental disorders* (pp. 112–128). Cambridge University Press.