Autism Spectrum Disorders

From Theory to Practice

Third Edition

Pearson

Laura J. Hall

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From Theory to Practice

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LAURA J. HALL
San Diego State University

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About the Author

Laura J. Hall, Ph.D., is a professor and chair of the Department of Special Education at San Diego State University (SDSU). She has been working with individuals with autism spectrum disorders and their families for more than 35 years and is responsible for the creation of the master's degree program with a specialization in autism at SDSU. Her current research interests focus on factors that facilitate the implementation and sustained use of evidence-based practices by educators and paraprofessionals working with learners with autism spectrum disorders. Dr. Hall's involvement with several boards of nonprofit organizations supporting autism is another way in which she is working to develop a solid community of practice in San Diego.

Dedicated to

Pat Krantz, Lynn McClannahan, and Sam Odom

Inspirational role models and mentors

Preface

While researchers are trying to determine the currently unknown cause of autism spectrum disorders (ASD), there is a need for effective strategies to address the characteristics displayed by individuals with this classification. The need for knowledgeable and skilled educators who can identify, implement, and sustain the use of evidence-based practices is critical and is likely to remain so well into the future. *Autism Spectrum Disorders: From Theory to Practice* is a comprehensive text that provides information about ASD from up-to-date research on brain development and genetics to working with families to prepare adolescents and young adults for transition. Descriptions of common practices used by educators are organized by theoretical perspective. How to determine whether there is research support for a practice, or whether a practice is evidence-based, is explained. The research support, or lack thereof, is provided following each description of the approach, programs, and current practices.

New to This Edition

The passing of the Combating Autism Act in 2006 and the increase in prevalence of ASD that continues to be reported by the Centers for Disease Control and Prevention have resulted in increased funding for research and increased attention by researchers. Each chapter in this revised edition has been updated to reflect the most current research outcomes. This edition also uses the results from several publications of literature reviews in 2015 that have identified evidence-based strategies and comprehensive programs (e.g., from the National Standards Project and the National Professional Development Center on Autism Spectrum Disorders) as the basis for reviewing common practices. The criteria used for describing autism spectrum disorder are taken from the revised diagnostic manual from the American Psychiatric Association (DSM-5) and relate current research and practice to these criteria. The following is a list of changes and updates in the third edition of *Autism Spectrum Disorders*:

- Literature reviews of all content areas have been updated, and description and emphasis on the identification of practices and strategies with sufficient research evidence, as determined by the National Standards Project and the National Professional Development Center on ASD, will provide state-of-the-art information about which practices are scientifically based.
- New to this edition is the highlighting of *Sensory Considerations* found in each chapter. Examples of practices that have been used to address the possible sensory issues such as hyper- or hyposensitivity to sensory stimuli and the associated challenges for individuals on the spectrum can be found throughout the text.
- New to this edition are the video examples in each chapter to assist the reader to *Learn more about* the content covered. Videos include examples of described interventions, experts speaking on the topics addressed, individuals with ASD providing recommendations, and discussions from model developers about the focus of their approach.

- A chapter on systems of support for individuals with ASD and their families (Chapter 10) has been added. Content on the influence of culture and how to work with families from diverse backgrounds is a main focus of this chapter. The importance of collaboration within and across systems, such as between families and school personnel and between school personnel and agencies, is emphasized. The importance of advocating for policy and a history of examples of parent-initiated efforts resulting in policy changes are included.
- Cultural and linguistic considerations are emphasized throughout the text, and especially so in Chapter 10, so that all practitioners are sensitive, knowledgeable, and aware of the influences of culture and language and can implement culturally responsive strategies.
- Each chapter contains suggestions for future research, and these updates take into account research questions that have been addressed in recent years and new questions that have arisen as a result of ongoing research. Students planning to conduct research will find the summaries of current research and recommendations for future research helpful in designing projects.

Organization of the Book

The text is organized into three parts. Part I includes three introductory chapters. Chapter 1 focuses on the current approaches to determining the causes of autism spectrum disorders and provides a description of strategies used to address this disorder on a physiological level, such as through prescription medication, diet, or activities to arouse or calm the sensory systems. Chapter 2 provides information regarding assessment that is organized by purpose (screening, diagnosis, educational planning). The emphasis in this chapter is on curriculum-based assessment that links assessment results to educational practice. The identification and effective implementation of evidence-based strategies are addressed in Chapter 3. A description of the individualized education program (IEP) process is provided. The importance of working in collaboration with families is emphasized with recommendations for effective practice.

The following three chapters describe the programs and practices used to increase the skills of individuals with autism spectrum disorders and comprise Part II of the book. Each chapter is organized by theoretical perspective. Chapters 4 and 5 focus on principles, programs, and strategies based on applied behavior analysis. Programs and practices influenced by the developmental, social-relational, transactional, and cultural theories are included in Chapter 6. An emphasis on working collaboratively with families is discussed in all approaches, regardless of theoretical perspective. Each approach or program model includes a brief description of the theoretical perspective, a historical overview of strategies used by educators working with individuals with autism spectrum disorders, definitions and descriptions of key concepts of the approach, examples and illustrations of strategies based on key concepts, a description of a model program or classroom based on the approach, and a summary of the research evidence for the approach and practices.

Part III contains four chapters. The first two put together the information across the theoretical approaches described in Part II to address two areas that are frequently a focus for educators: communication (Chapter 7) and social relationships (Chapter 8). Chapter 9 focuses on preparing adolescents and young adults for transition. Best practices are summarized with examples of high-quality programs. Chapter 10 focuses on the systems of support necessary to achieve optimal outcomes for individuals with ASD and their families. Strategies for working in collaboration with diverse families, colleagues in schools and agencies, paraprofessionals, and

administrators are emphasized. This last chapter (and the book) concludes with a description of the major contributions to the field made by families and the importance of working in collaboration with families to influence policy, change systems, and build communities of practice.

Features of the Text

In Their Words

Boxes called In Their Words, found in each chapter, feature input and quotes from professionals and parents of individuals with autism spectrum disorders and interviews with adults identified on the spectrum. These features augment the chapter content by enabling the reader to obtain the perspective of professionals using various theoretical approaches (e.g., Drs. Krantz and McClannahan, Dr. Mesibov, Dr. Wolfberg) from various disciplines such as speech-language pathology (Colleen Sparkman), occupational therapy (Janinne Karahalios), psychology (Dr. Natacha Akshoomoff, Dr. Brooke Ingersoll), and special education (Dr. Dean Fixsen, Dr. Bonnie Kraemer, Dr. Eleanor Lynch, Dr. Angela McIntosh, Sheila Wagner). Special education teachers (Cindy Bolduc, Penelope Bonggat), parents (Laura Wood, Juan and Sharon Leon), and adults with autism spectrum disorders (Erik Weber) provide their perspective on effective practices, diagnosis, and suggestions for preparing for postsecondary education.

Sensory Considerations

Boxes called Sensory Considerations, found in each chapter, highlight assessments and interventions used to address the sensory issues experienced by individuals with ASD, such as hyper- or hyposensitivity to stimuli, one of the diagnostic criteria added to the DSM-5.

Evolution of Practices

The evolution of practices in special education is included in most chapters to reflect the development of approaches and practices over time and to capture the historical progression of events or strategy development. Educational practices with learners with autism spectrum disorders are far from static. These practices are influenced by changes in philosophy, policies, and research outcomes.

Research Emphasis

The importance of research is emphasized throughout the text, including a review of the evidence for practices and suggestions for future research. This emphasis will be particularly helpful to master's degree and doctoral candidates, but it will provide direction for all practitioners who are working to further the field with information on the effectiveness of intervention strategies. Understanding the research evidence, or lack thereof, is important for all educators who are required to use only scientifically based practices.

Learning Outcomes, Suggestions for Discussion, Resources

Each chapter begins with a list of learning outcomes for the information gained by reading the chapter content. Chapters conclude with two features. The first is a list of suggestions for discussion that are focused on key points from the respective chapter. These suggestions for discussion include a suggestion for a debate (usually number 3 in the list) on a particular issue from the chapter. A list of resources (books and websites) provides additional information on various topics found in the respective chapter.

Acknowledgments

This book would not have been possible without the education, professional guidance, and encouragement provided by my professional mentors Pat Krantz, Lynn McClannahan, Beth

Sulzer-Azaroff, Gail McGee, and Sam Odom. I am also grateful to the individuals with autism spectrum disorders who taught me a great deal about how to address educational challenges with clarity, consistency, and humor. I am appreciative of the parents of the learners with ASD who have sought and supported my skills and have served as excellent partners. A special thank-you goes to my students, who have been exceptional research collaborators and who inspire me with the energy, enthusiasm, and affection that they bring to their work with learners and their families. I continue to learn from each of them.

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I would also like to express my utmost gratitude to Megan Ledoux for the assistance provided throughout the preparation of this edition, including searching for articles, finding for the best video examples from YouTube, and organizing the multitude of references. Much appreciation goes to Marianne Bernaldo, who provided some of the content for the revision of Chapter 9. Special thanks goes to my family, especially my mother Phyllis Hall, who is a constant source of support and encouragement, and above all to Sam, my fulcrum and sage.

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CHAPTER ONE

Classification and the Physiological Approach

LEARNING OUTCOMES

At the conclusion of this chapter, the reader should be able to:

- State the criteria for the diagnosis of autism spectrum disorder (ASD) according to the classification system developed by the American Psychiatric Association, the DSM-5.
- Explain the possible causes of autism spectrum disorders currently under investigation by researchers.
- Discuss physiological interventions for individuals with ASD, provide a rationale for the use of sensory activities, and consider the implications for the lack of evidence to support sensory-based treatments.
- Describe the ways in which an occupational therapist can be a helpful collaborator for addressing the sensory needs of individuals with ASD.

The Autism Society of America (ASA) uses a ribbon of puzzle pieces as the symbol of autism spectrum disorders. This symbol is used because so much about autism spectrum disorders (ASD) remains unknown, or a puzzle.

Written on the About Autism page of the ASA website is the comment: ASD is defined by a certain set of behaviors and is a "spectrum condition" that affects individuals differently and to varying degrees. There is no known single cause of autism, but increased awareness and early diagnosis/intervention and access to appropriate services/supports lead to significantly improved outcomes (Autism Society of America, 2016).

In this chapter the currently used classification systems of the *Diagnostic and Statistical Manual of Mental Disorders* and the *International Classification of Diseases* and the classifications under the Individuals with Disabilities Education Act will be described. Each of these systems represents the consensus opinions of the authors at the time of publication, and all are revised as additional information about autism spectrum disorders is learned.

The evidence for a genetic influence on autism spectrum disorders and the current areas of focus in determining the cause will be explored. Much of the effort to find the cause focuses on the biology and physiology of individuals with ASD and their families. Interventions that address the biological or physiological aspects of an individual with autism spectrum disorder, such as the prescription of medication, recommendation for a special diet, and organizing activities that address arousal (calming or stimulating) such as exercise and sensory interventions, will be reviewed. In summary, this chapter includes a description of various classification systems for autism spectrum disorders; an explanation of the genetic, biological, and physiological areas under investigation to determine a cause; and descriptions of interventions aimed at changing the physiology of the individual.

Classification Systems of Autism Spectrum Disorders and Prevalence

Classification of the characteristics associated with autism spectrum disorder (ASD) into a specific category occurred in the 1940s by Leo Kanner,

a U.S. psychiatrist (1943), and Hans Asperger, a German pediatrician (1944). Influenced by adult psychiatry, the term **autistic** (derived from the Greek word *autos* for "self") was used as the description of negative symptoms when someone was entirely uninterested in the outside world (Houston & Frith, 2000). Kanner described a triad of impairments (social difficulties, communication problems, and repetitive and restricted activities) in his paper about 11 children from the Child Psychiatric Unit at Johns Hopkins University where he worked (Mesibov, Shea, & Adams, 2001).

Lorna Wing was one of the first people to use the term **spectrum** when describing a group of individuals displaying the characteristics of autism. In their project conducted in the 1970s (the Camberwell study), Wing and Gould (1978) found that individuals with autism varied in the degree of severity of their displayed characteristics as well as in the form of differences and delays, leading the authors to begin to discuss this spectrum of disorders (Houston & Frith, 2000). Included in this spectrum were individuals with no cognitive delays, referred to by the researchers as individuals with high-functioning autism, and individuals with Asperger syndrome (Wing, Leekam, Libby, Gould, & Larcombe, 2002). Wing was aware of Hans Asperger's work, and when she began to publish her research in the early 1980s, she used the term **Asperger syndrome**, resulting in international awareness of this classification. Prior to this time, Asperger's work was not well known due to the fact that it was published only in German (Mesibov et al., 2001).

Diagnostic and Statistical Manual of Mental Disorders

One of the main classification systems used internationally is the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) published by the American Psychiatric Association (APA). Details of the characteristics that classify individuals with varying diagnoses are made by a group of psychiatrists, psychologists, and physicians with expertise in the area. The criteria for the fifth edition of the manual were published in 2013. The focus of the revised DSM version is on the variability in severity of characteristics that comprise one category of autism spectrum disorder compared with the five distinct disorders of the pervasive developmental disorders in the DSM-III and IV (American Psychiatric Association, 1980, 1987, 1994, 2000) such as autistic disorder, Asperger syndrome, Rett's disorder, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified (PDD-NOS) (Volkmar, Reichow, Westphal, & Mandell, 2014).

There are two domains with criteria used for autism spectrum disorder reduced from three in the previous version (Volkmar et al., 2014) that include (a) persistent deficits in social communication and social interaction across contexts and (b) restricted, repetitive patterns of behavior, interests, and activities. In addition, (c) symptoms must be present in childhood that (d) limit and impair everyday functioning. Individuals must have deficits in all three subcategories of social communication and social interaction (deficits in [1] social-emotional reciprocity, [2] nonverbal communicative behaviors, [3] developing and maintaining relationships) and two of four subcategories of restricted and repetitive patterns of behavior ([1] stereotyped or repetitive speech, motor movements, or object use; [2] excessive adherence to routines or ritualized patterns of verbal or nonverbal behavior; [3] highly restricted, fixated interests; [4] hyper- or hyporeactivity to sensory stimuli) to receive a diagnosis (American Psychiatric Association, 2013). Children with this diagnosis may have skill areas where they are functioning similarly to typical peers or may even exceed the skills of peers in areas such as music, math, or reading.

The criteria for the DSM-5 represent a change in the classification category as well as the sub criteria that comprise the symptoms compared with earlier versions of the manual (American Psychiatric Association, 1980, 1987, 1994, 2000). Table 1.1 shows some of the changes in language and age of onset for the versions between 1980 and 2011. It appears from the changes in diagnostic criteria over

TABLE 1.1 Sample changes in diagnostic criteria for autism from the DSM

DSM III-1980	DSM IIIR-1987	DSM IV-1994 and TR-2000	DSM-5 2013
Onset before 30 months	Onset before 36 months	Delays or abnormal functioning in one area (social interaction, language or play) before 36 months	Symptoms in early childhood
Gross deficits in language development	Qualitative impairment in both verbal and nonverbal communication	Qualitative impairment in communication	No separate impairments for communication
Pervasive lack of responsiveness to others	Qualitative impairment in reciprocal social interaction	Qualitative impairment in social interaction	Deficits in social-emotional reciprocity and social relationships

time that children can get a diagnosis if fewer characteristics appear later. Hyper- or hyposensitivity to sensory stimuli was included in early diagnostic versions (DSM III, 1980) and again in the latest version (American Psychiatric Association, 2013).

The DSM-5 criteria were created under the assumption that deficits in communication and social interaction are inseparable and it is more accurate to consider them together and influenced by contextual and environmental variables. This decision was made based on literature reviews, expert consultation, and workgroup discussions (American Psychiatric Association, 2013). Because distinctions among the disorders in previous versions of the manual were found to be inconsistent over time and the use of diagnostic categories varied across sites, the use of a single diagnostic category (autism spectrum disorder) was adopted in the latest version. In the DSM-5, it is recommended that the level of severity for social communication and restricted interests and repetitive behaviors is considered on a scale of 1 (requiring support) to 3 (requiring very substantial support).

International Classification of Diseases

The *International Classification of Diseases,* published by the World Health Organization (WHO), is in its tenth version and is referred to as the ICD-10. The ICD-11 revision will be an online tool that has a target completion date of 2017. This classification system is used widely in Europe as well as in other countries worldwide. Autism first appeared in the ICD in 1967, when it was listed as *infantile autism* under one of the subtypes of schizophrenia. In 1974, infantile autism was classified under behavior disorders of childhood but without defining diagnostic criteria (Leekam, Libby, Wing, Gould, & Taylor, 2002). The ICD-11 will use the term *autism spectrum disorder* (7A20) under the category of mental and behavioral disorders with the subclassifications of with a disorder of intellectual development or without, with or without functional language, and with or without loss of previously acquired skills and all combinations of these factors. It is defined as a neurodevelopmental disorder as such:

> Autism spectrum disorder is characterized by persistent deficits in the ability to initiate and to sustain reciprocal social interaction and social communication, and by a range of restricted, repetitive, and inflexible patterns of behaviour and interests. The onset of the disorder occurs during the developmental period, typically in early childhood, but symptoms may not become fully manifest until later, when social demands exceed limited capacities. Deficits are sufficiently severe to cause impairment in personal, family, social, educational, occupational or other important areas of functioning and are usually a pervasive feature of the individual's functioning observable in all settings, although they may vary according to social, educational, or other context (World Health Organization, 2016).

Individuals with Disabilities Education Act (IDEA)

Autism was added as a separate category of disability that may require special education services in 1990 under PL 101–476, the Individuals with Disabilities Education Act (Knoblauch & Sorenson, 1998). To receive special education, the child (1) must have one or more of the disabilities from a list that includes autism and (2) must require special education and related services. Autism is defined by IDEA as a developmental disability significantly affecting verbal and nonverbal communication and social interaction, generally evident before age 3, that adversely affects a child's educational performance. Other characteristics often associated with autism are engagement in repetitive activities, stereotyped movements, resistance to environmental change or change in daily routines, and unusual responses to sensory experiences. Individual states have their own criteria for eligibility of early intervention and special education services. Educators should be aware of what their own state requires for eligibility.

Classification systems are important in order to determine the outcomes of research. Replication is a hallmark of good science. If an intervention is demonstrated as helpful for a group of individuals classified with ASD, it will be important to replicate the intervention with a similar group prior to claiming effectiveness. Without a classification system and the inherent definitions of characteristics, this replication would not be possible. Classification or diagnosis is also important for parents who are eager to understand the atypical behavior of their child.

Prevalence

The **prevalence** is the number of cases of a condition that exists at a particular time in a defined population. The estimated prevalence rates following the creation of the classification category for autism in the 1940s was considered to be 4 or 5 in 10,000 for decades (Stevens et al., 2007). This estimate increased in the mid-1990s to 10 per 10,000 (1 in 1,000) for autism and 22 per 10,000 for pervasive developmental disorder (PDD) (Mauk, Reber, & Batshaw, 1997). A review of 54 studies of prevalence completed internationally published between 1966 and early 2013 for 23 countries revealed a correlation between prevalence rate and publication year with higher rates in more recent years (Hill, Zuckerman, & Fombonne, 2014). The mean international prevalence rate from recent surveys is 66/10,000 or 1 in 152. Research findings indicate that low SES, minority, and immigrant populations experience problems of underdiagnosis (Hill et al., 2014). It is important to note that the increase in prevalence rates could be attributed to increased awareness and access to services and that "the possibility that a true change in the underlying incidence has contributed to higher prevalence figures remains to be adequately tested" (Hill et al., 2014, p. 90).

Research indicates that regression, or loss of skills such as use of language, occurs in 1 in 4 children diagnosed with ASD and is associated with more severe symptoms (Fombonne, Quirke, & Hagen, 2011). If regression occurs, it is likely to happen during the 6 months prior to age 2. Researchers have found that there is no difference on a range of outcome measures for children with or without regression (Ozonoff, Heung, & Thompson, 2011). The median percentage of individuals with autism spectrum disorders that have a co-occurring intellectual disability is 65% as calculated from 14 studies completed since 2000 (Dykens & Lense, 2011). It should be noted that estimates of how often intellectual disabilities occur with ASD vary widely and are influenced by definition and classification systems. Compared with the general population, epilepsy is 10 to 30 times more prevalent in individuals with ASD and occurs most often when there is a moderate to severe intellectual disability (Tuchman, 2011).

The Autism and Developmental Disabilities Monitoring (ADDM) Network funded by the Centers for Disease Control and Prevention estimates the prevalence of autism spectrum disorders in the United States, or the number of cases identified, to be 1 in 68 (Christensen et al., 2016).

This information is based on data obtained across 11 states in the United States during 2012. Researchers in each of the states collected data from health and school records to determine the number of 8-year-olds diagnosed with ASD as well as the ethnic background of these students. The authors comment that the 11 states included in the study (Arizona, Arkansas, Colorado, Georgia, Maryland, Missouri, New Jersey, North Carolina, South Carolina, Utah, and Wisconsin) are not representative of the nation and that the prevalence rates should be used with caution (Stevens et al., 2007). The prevalence rates vary among states, with an earlier study reporting lowest rates for South Carolina (1 in 81) and highest for New Jersey (1 in 41) (Christensen et al., 2016). The ratio of 1 in 68 remains the same as the prevalence reported from the 2010 data. Although the prevalence stayed the same for most of the states, there were increases in New Jersey and Wisconsin. The authors of the ADDM report state that it is too soon to say if the prevalence rate for ASD is stabilizing (Christensen et al., 2016). This high prevalence rate of less than 1 in 1,000 means that ASD is now considered a common disease (Bailey, 2016).

Consistently more boys than girls are classified with autism spectrum disorders, with a ratio of approximately 4 or 5 boys to 1 girl. White non-Hispanic children were 1.2 times more likely to be identified with ASD than Black non-Hispanic children and 1.5 more likely than Hispanic children (Christensen et al., 2016). Black and Hispanic children were also less likely to be evaluated for concerns by age 3 compared with White children, among whom 43% are evaluated for concerns prior to age 3. Approximately one-third (32%) also had an intellectual disability, 22% were considered borderline, and the remaining 44% had average to above average intellectual ability.

The majority of children do not receive a diagnosis by a community provider until age 4. In a prevalence study conducted by the ADDM in five of the participating states focusing on 4-year-olds, the authors found that those states that had both health and education records for review had twice as many children with ASD identified than those that relied on health records alone (Christensen et al., 2015). The authors conclude that the special education system has an important role in identifying children with ASD. Across the five states the prevalence rate for 4-year-olds was lower than for 8-year-olds, indicating increased identification over time (Christensen et al., 2015).

Whether the increase in prevalence rates represents a true increase in incidence of ASD has yet to be determined (Fombonne et al., 2011). The factors that may have contributed to the increase in prevalence include (1) an increased awareness by the public and by physicians and psychologists who make the diagnosis, (2) the broadening of the classification to include pervasive developmental disorder that requires minimum criteria for classification, (3) different methods for case finding, and (4) service availability (Fombonne et al., 2011). Epidemiologists also argue that ASD is a cultural phenomenon and that how it is conceptualized influences both diagnosis and treatment; they explain ASD as an interplay of biological, cultural, and psychological phenomena (Grinker et al., 2011).

What Causes Autism Spectrum Disorders?

History of Attribution of Cause

Autism has long been considered a classification of a mental disorder. Early individuals with moderate to severe delays in language and social skills would have been placed in institutions to be "treated" by the medical establishment of the time. Kanner, a psychiatrist, first wrote about what was once called infantile autism and the associated symptoms in his paper in 1943. It is likely that individuals with autism who were high functioning were considered odd loners who were not classified as needing intervention.

In the 1950s, psychiatrist Bruno Bettelheim attributed the symptoms of autism to uncaring and detached mothers who did not love their

1.1 IN THEIR WORDS

Laura Wood

Three Children on the Spectrum

My husband and I have three wonderful sons. They are all on the autism spectrum. Our oldest son Alex's diagnosis at age 2.5 came as a tremendous blow to us, as it does to every family grappling with this disorder. At that time our twins were 5 months old and we were thoroughly overwhelmed by our responsibilities. But we managed to launch a high-quality home intervention program for Alex and felt that we were doing everything we could do for him.

As the shock of Alex's diagnosis wore off a bit, I did start overanalyzing certain observations of the twins' behavior. Were they smiling? Paying attention to faces? During their first year of development, despite my hypervigilance, I believed it to be unlikely that they were autistic. I had not yet heard about the increased likelihood of autism in siblings of autistic children, so I told myself I was being unnecessarily paranoid.

But as the months went by and certain developmental milestones were missed or seemed ambiguous, the nagging feeling returned and slowly I realized that the nightmare scenario was coming to pass. The twins were both diagnosed with autism spectrum disorder shortly before their second birthday.

The most obvious impact of having kids "on the spectrum" is the financial commitment required for their home therapy. We are extremely lucky in Seattle to have a wonderful integrated preschool that all three kids attend for specialized instruction, but supplemental home programs are also recommended for two of our children, and the staggering cost of those programs is not covered by health insurance.

In addition, as many parents of children with special needs will tell you, guilt is ever-present. Sometimes the guilt has a specific source (Am I spending enough time encouraging communication and appropriate play? Have we set up *enough* hours of home therapy?). Other times the guilt is vague, intangible, and inexplicable.

I have times when I feel bitter and isolated. I look at other families with typically developing children with longing or even with anger. They can't know what it's like to take their children to countless therapy appointments and spend tens of thousands of dollars on essential therapy. How can I relate to these parents who take for granted their child's imaginative play skills or brag recklessly about their baby's first words? And how can they relate to me when I occasionally confess the reason why my kids don't always respond appropriately to a peer's invitation to play? Unless autism has touched their lives in some way, a blank look tinged with pity is all those parents can muster.

But in other ways I recognize the gifts sent our way by this unexpected path in life. There are several wonderful teachers and therapists who would never have been in our lives had it not been for the autism in our family. And my children have reminded me that there are different varieties of intelligence beyond what we think of as typical.

My kids are sweet, wonderful rays of sunshine in my life. They give me so much joy that all the worry and guilt and expense are absolutely worthwhile. I have already learned from them, and I'm sure they have more to teach me as we continue through our lives together.

children enough. Treatment consisted of removing the children from these so-called **refrigerator mothers**; offering psychoanalysis or counseling to the mother; and providing play therapy to the child, ideally in Bettleheim's institute in Chicago. The disorder was thought to occur in middle-class Caucasian families where both parents were educated. During this period of history, not only did the mothers of children with autism have to care for a child with challenging and unusual behaviors, they also experienced the added burden of being blamed as the cause.

In 1964, Bernard Rimland published the book *Infantile Autism: The Syndrome and Its Implications for a Neural Theory of Behavior*, which attributed the cause of autism to biology rather than poor parenting. Rimland dedicated his career to addressing the biological issues that contribute to and result from autism spectrum disorders. He had long hypothesized that there are brain differences in individuals with ASD compared to the brains of typically developing children. Rimland founded the Autism Research Institute (ARI) that supports the project Defeat Autism Now (DAN). Annual conferences are held where physicians present information on possible metabolic (interrelated chemical interactions that provide the energy and nutrients) contributions to symptoms and suggest diets that can be used to avoid the side effects of toxins for individuals with ASD.

Genetic Influences

> Autism is a behaviorally defined lifelong neurodevelopmental disorder, with strong evidence for a complex genetic predisposition. (Lamb, 2011, p. 669)

Autism is currently considered a **neurological disorder** that is influenced by both environmental (including the in-utero environment) and genetic factors (Sigman, Spence, & Wang, 2006). Initial evidence for a genetic contribution to ASD is found in a series of twin studies conducted across several countries (e.g., United Kingdom, Scandinavia, United States) with similar outcomes (Bailey et al., 1995; Ritvo, Freeman, Mason-Brothers, Mo, & Ritvo, 1985; Rutter, 2005; Steffenburg et al., 1989). When one twin in a monozygotic pair of twins (identical) was diagnosed with autism, there was a high likelihood that the second twin, with the same DNA, also was diagnosed with autism (Cook, 1998). However, in dizygotic twins (fraternal), the concordance for the diagnosis for autism was very low. In addition to the twin studies, it has been found that the risk of autism for siblings of those identified on the spectrum is 15% to 20%, considerably higher than the population risk (.05% to .1%) (Lamb, 2011).

Results of surveys have revealed that relatives have an increased frequency of lesser variants of autism, including social, language, and repetitive behaviors (Dworzynski, Happe, Bolton, & Ronald, 2009; Rutter, 2005). This variation of the spectrum is referred to as the broad autism phenotype (BAP) (Ingersoll & Wainer, 2014; Sasson, Lam, Parlier, Daniels, & Piven, 2013) that describes individuals that have personality profiles related to the expression of subclinical characteristics of autism (Volkmar et al., 2014). The rate of BAP in first-degree relatives is estimated to be 57% (Ingersoll & Wainer, 2014). In adults, difficulties with social functioning result in less desire for close relationships and lower quality friendships (Ingersoll & Wainer, 2014).

The findings of a study investigating the broad autism phenotype among 711 parents of children with autism and 981 comparison parents using the Broad Autism Phenotype Questionnaire (Hurley et al., 2007) concluded that the parents of children with autism were more likely to have one parent with BAP characteristics than two parents with BAP and that the severity of autism was associated with having a parent with BAP characteristics compared with parental pairs with neither parent with the broad autism phenotype (Sasson et al., 2013). Although molecular genetic research has revealed loci in inherited, familial forms of ASD, there is substantial between-family locus heterogeneity (Piven et al., 2013), and molecular genetic studies have not identified the inherited factors of autism (Bailey, 2016). "Efforts to uncover that risk genotypes associated with the familial

nature of autism spectrum disorder (ASD) have had limited success" (Piven et al., 2013, p. 1).

The current thinking is that "autism spans the genome" (Coleman & Betancur, 2005, p. 17), or is likely to be caused by multiple genetic loci on several chromosomes. Our genes are found on 23 pairs of chromosomes numbered from 1 to 22 and an X or Y chromosome. The larger the number of the chromosome, the smaller the size of that chromosome. When using specialized equipment, each chromosome appears in an X shape with the top arms, or p part, shorter than the lower and longer q portion.

New technology provides additional information about gene insertions, deletions, and the interactions related to autism; however, there remain approximately 100 genes that potentially create a risk for ASD, and these genes are involved in a wide range of biological mechanisms (Bailey, 2016). Some of the genes on the following chromosomes are hypothesized to contribute to ASD: chromosome 1p21.3 and 1q25.1-1q25.2, chromosome 3p14 and 3q26.31 (Kaymakcalan & State, 2011), chromosome 7q31.2 (Lamb, 2011), chromosome 13q21.32 (Kaymakcalan & State, 2011; Rutter, 2005), chromosome 15q11-q13 (Delahanty et al., 2011) (or the same region that is missing in Prader-Willi and Angelmann syndrome), chromosome 16p13.2 (Kaymakcalan & State, 2011), chromosome 19p and 19q (Rutter, 2005), chromosome 22q13.3 (Kaymakcalan & State, 2011), and the X chromosome (Coleman & Betancur, 2005; Rutter, 2005). Chromosome X is of particular interest due to the higher ratio of males to females with the disorder and because of fragile X syndrome (Hagerman, Narcisa, & Hagerman, 2011), which results in similar symptoms (Morrow & Walsh, 2011).

Although research has revealed possible links between genes and ASD, there has not been a set of genes that indicate a high risk for a large number of individuals, the heritability is not understood, and the sex difference remains unexplained. No firm conclusions can be drawn yet about the influence of genes on biological pathways (Rutter & Thapar, 2014). It may be that, similar to intellectual disability, there is no one unified neurobiology for ASD but multiple paths that result in similar biological and behavioral characteristics (Bailey, 2016). However, researchers are continuing to explore the influence of gene networks that may result in ASD. One promising line of research is analyzing blood samples to determine if biomarkers for ASD can be found (Pramparo et al., 2015). One study using blood samples from boys ages 1 through 4 with ASD and a control group identified differentially coexpressed genes in translation and immune/inflammation functions for the boys with ASD in 83% of the sample. The authors conclude that these results mean that a blood-based clinical test for at-risk male infants and toddlers could be created and routinely implemented in pediatric settings (Pramparo et al., 2015). Replication of these findings for the identified biomarkers is needed.

Differences in Structure and Function of the Brain

In Kanner's original description of autism, he noted that 5 of the 11 children had large heads (Minshew et al., 2005). At birth the head circumference of infants who later were diagnosed with autism is near normal; however, by 6 to 14 months of age (Hazlett et al., 2012), the head circumference becomes enlarged, reflecting early brain and cerebrum overgrowth (Courchesne, Webb, & Schumann, 2011; Hazlett et al., 2011) that continues during the first 2 years of life (Courchesne, 2011). In a study of 270 infants at high risk for ASD and 108 low-risk controls during the first 2 years of life, findings indicated a significant increase in the corpus callosum area and in thickness for children with ASD starting at 6 months of age compared with the control group and that these difference diminish by age 2 (Wolff et al., 2015). See Figure 1–1 for a model of a typical brain.

The brain overgrowth and dysfunction is probably due to a dysregulation of layer formation and layer specific neuronal differentiation that occurs during prenatal development (Stoner et al., 2014). The most likely cause is

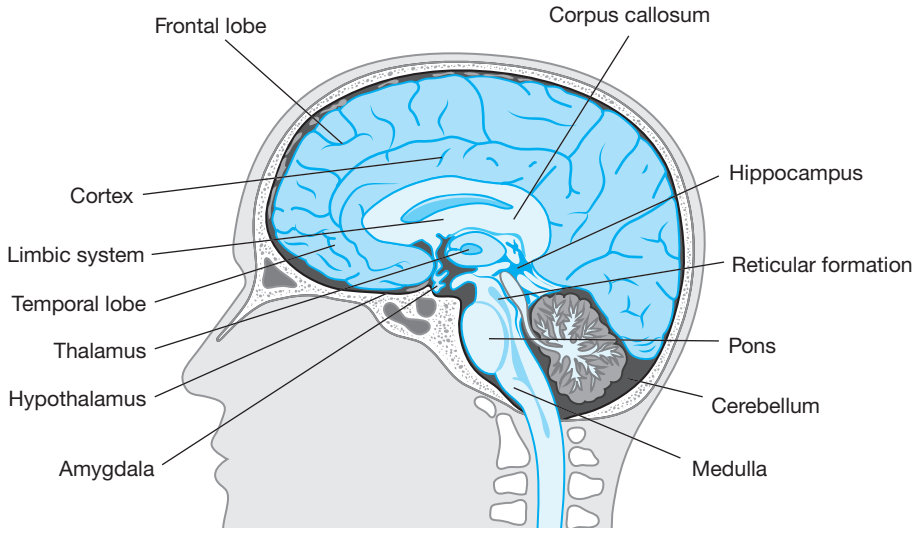

FIGURE 1–1 Key parts of the brain: The figure shows a vertical slice near the middle of the brain

excess neuron numbers due to dysregulation of neurogenesis, or the lack of the normal pruning and connecting of neurons. Researchers using an analysis of postmortem data in toddlers with ASD hypothesize that there could be axonal overconnectivity in the frontal brain lobes and amygdala (Solso et al., 2016).

Temple Grandin, professor of animal science at Colorado State University who is one of the most well-known adults with autism, writes the following in her book titled *The Autistic Brain* (Grandin & Panek, 2013, p. 27–29).

> Autistic brains aren't broken. My own brain isn't broken . . . They just didn't grow properly. . . . Autism researchers have contacted me over the years to ask permission to put me in this scanner or that. I'm happy to oblige . . . Thanks to a scan at UCSD School of Medicine's Autism Center of Excellence, I know my cerebellum is 20 percent smaller than the norm. . . . so this abnormality probably explains why my sense of balance is lousy. In 2006, I participated in a study at the Brain Imaging Research Center in Pittsburgh and underwent imaging with a functional MRI scanner and a version of MRI technology called diffusion tensor imaging (DTI). While fMRI records regions in the brain that light up, DTI measures the movement of water molecules through white matter tracts—the interoffice communication among the regions. The fMRI . . . showed a lot less activation in response to faces than (a control) did. The DTI imaging indicated that I am over-connected . . . way more connections than usual."

Autism does not result from a problem with one location in the brain but from abnormalities within one or multiple neural systems and with under-connectivity of cortical systems (Minshew, Scherf, Behrmann, & Humphreys, 2011; Coleman, 2005). Magnetic resonance imaging (MRI) studies have revealed the overgrowth in the frontal and temporal lopes of the brain and the amygdala (Courchesne et al., 2011). The frontal lobes are considered to play a role in memory formation and emotional expression, and patients with frontal lobe damage demonstrate a decreased ability to respond to stimuli in the environment (Reichler & Lee, 1989). The frontal lobes are the part of the brain where planning, organizing, self-monitoring, inhibition, flexibility, and working memory, or the cognitive construct of executive functioning, are considered to occur (Ozonoff, South, & Provencal, 2005). The amygdala is responsive to stimuli that are highly rewarding, such as a mother's face, for typical infants (Courchesne et al., 2011).