# Chapter 1
# Autism and Autism Treatment: Evolution of Concepts and Practices from Kanner to Contemporary Approaches


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Autism spectrum disorder (ASD) is a developmental disorder that requires lifelong support. As is often the case for conditions of uncertain etiology, many intervention approaches for ASD exist, only a minority of which have been empirically validated. Evidence-supported early interventions can result in substantial improvements in language, cognitive, social, and adaptive functioning, holding the potential to promote positive long-term outcomes, mitigate lifespan disability, reduce societal costs, and improve personal well-being and productivity of individuals with ASD and their families.

However, advising families in search for the best intervention to help their young children with ASD is challenging, due to the proliferation of intervention options and the heterogeneity of intervention needs within the ASD population. Additional elements of complexity include the striking variability in treatment response, with even the most established interventions producing different levels of success across individuals and contexts, as well as challenges with access to services and the financial impacts on families (Nahmias et al., 2019; Vivanti et al., 2014). Finally, scientific and philosophical divergences across the scientific community, advocacy groups, and practitioners further contribute to a difficult terrain for navigating intervention options. The aim of this book is to provide practical information to navigate this chaotic landscape, starting with an analysis of how knowledge and concepts about ASD and ASD treatment have evolved over the past decades.

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## From Kanner to the DSM-5: Evolution of the Diagnostic Concept of Autism and Current Diagnostic Issues

The diagnostic concept of autism was originally formulated in 1943 by an Austrian psychiatrist of Ukrainian origin, Leo Kanner. In his seminal report (1943), Kanner described 11 children affected by what he defined as "autistic disturbances of affective contact," a syndrome characterized by diminished or absent interest in other people, reduced social communication, and a striking "insistence on sameness." Although clinical reports of children with similar characteristics were published before Kanner's report (e.g., De Sanctis, 1906; Klein, 1930/1975; Ssucharewa, 1926), or at around the same time (most notably by Hans Asperger; 1944), Kanner's conceptualization of autism as a distinct clinical syndrome characterized by early onset social communication abnormalities and behavioral rigidity has proven the most influential and enduring.

The validity of autism as a distinct diagnostic entity separate from schizophrenia, language disorders, or general intellectual disability has been supported by subsequent research (Volkmar & McPartland, 2014). Five decades after Kanner's original description, autism was described in a report of the National Institute of Health Autism Working Group as "one of the most reliable diagnoses in psychiatric or developmental research" (Bristol et al., 1996). Following Kanner's first report, the diagnostic definitions of autism in subsequent decades have continued to include the notions of early emerging impairments in social communication (with a discrepancy between social and nonsocial abilities and deficits encompassing both verbal and nonverbal communication) as well as insistence on sameness/resistance to change. Despite this continuity, numerous shifts in the conceptualization and operationalization of autism have occurred, reflecting cultural changes as well as research advances.

In the decades after Kanner's report, the concept of autism was considered by most scholars to be overlapping with childhood psychosis or schizophrenia, with the terms "psychotic" and "autistic" being used interchangeably in research and practice throughout the 1950s, 1960s, and 1970s. Following research by Rutter (1972) and others documenting critical differences between autism and schizophrenia, autism was first included as a separate diagnostic category in the third edition of the *Diagnostic and Statistical Manual of Mental Disorders* in 1980, under the definition of "infantile autism" (DSM-III, APA, 1980). This was later changed to "autistic disorder" in 1987 in the revised edition (DSM-III-TR, APA, 1987). Asperger's disorder, a related diagnostic category described as being characterized by higher intellectual functioning and better language abilities compared to autistic disorder, was added in the fourth edition of the *DSM* (DSM-IV, APA, 1994) as part of five mutually exclusive diagnostic subcategories under the umbrella diagnosis of "pervasive developmental disorders" (APA, 1994). This category also included Rett syndrome, childhood disintegrative disorder, and pervasive developmental disorder—not otherwise specified.

In 2013, the DSM-5 grouped the diagnostic criteria for autism in two clusters that are reminiscent of Kanner's original description: "social communication deficits" and "fixated interests and repetitive behaviors." Additionally, the different subtypes of autism defined by mutually exclusive subcategories used in the previous DSM editions were replaced by a single diagnostic label, "autism spectrum disorder" (ASD). This conceptualization reflects the homogeneity in the core impairments, as well as the continuum of variability in the presentation of the clinical features (i.e., the notion of autism as a "spectrum," originally introduced by Wing and Gould in 1979). Additionally, the DSM-5 includes designations for the level of severity for each symptom cluster (on a 3-point scale ranging from "requiring support" to "requiring very substantial support") as well as relevant clinical "specifiers," including language and cognitive ability levels.

The terms autism and autism spectrum disorder (ASD) are now used interchangeably. The term "Asperger," while no longer used clinically, continues to be present in the mainstream vocabulary, most frequently as a cultural identity concept (e.g., "Asperger culture" or "Aspie culture"; Giles et al., 2014). The terms "high functioning" and "low functioning" autism are not formal diagnostic concepts, but are colloquially used in clinical practice to describe individuals with ASD with less severe versus more severe symptomatology and/or cognitive impairment. Importantly, however, both the DSM-5 and the autistic community suggest that "low support needs" and "high support needs" are more accurate and less stigmatizing, as functioning is often proportional to the amount of support provided.

As no biological marker for ASD has been validated to date, ASD diagnosis is based on the ascertainment of the behavioral manifestations listed in the DSM-5, or other formal classification systems such as the International Classification of Diseases (World Health Organization, 1993), which is largely overlapping with the DSM definition. When diagnostic referral occurs in childhood, this is achieved through a combination of direct observation and parent reports focused on the child's developmental history, often supported by standardized protocols such as the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2012) and the Autism Diagnostic Interview (ADI-R; Le Couteur, Lord, & Rutter, 2003).

In most cases, a diagnosis of ASD can be made at 18–24 months, although some children might not be fully symptomatic until 36 month and, more rarely, until school age (Lord et al., 2018; Ozonoff et al., 2018). Once a diagnosis is made, it is generally stable, (Ozonoff et al., 2015; Talbott & Rogers, 2016; Zwaigenbaum et al., 2015). However, cases of children who lose their ASD diagnosis at school age have been reported, although the frequency and nature of this phenomenon remain unclear (Fein et al., 2013; Moulton et al., 2016).

## Epidemiology

ASD was once considered a rare disorder, with estimates in the 1960s suggesting 4–5 cases in 10,000. Currently, however, ASD is considered to occur in about 1:59 cases (Baio et al., 2018). While the steady rise in the prevalence of ASD in the last decades has been described by some as an "autism epidemic," it is still debated whether the changes in prevalence rates reflect a true increase in ASD cases. Factors such as the progressive broadening of the diagnostic criteria, improvements in diagnostic procedures, and increased awareness as well as the changes in policies for access to services might have contributed to the apparent "epidemic."

The sex ratio (male:female) is 4.3:1, but there is suggestion that females are more likely to remain undiagnosed (Bargiela et al., 2016; Giarelli et al., 2010). In the United States, there is evidence of ethnic and racial disparities in the identification of ASD (Durkin et al., 2017; Mandell et al., 2009). Little is known about other parts of the world in this respect.

## Clinical Characteristics

Autism is a developmental disorder originating in infancy. Behavioral manifestations of ASD are affected by and affect the developmental level of the individual (verbal ability and mental age), and the way symptoms manifest themselves at different times is also influenced by the child's chronological age, history of interaction with the environment (including intervention history), and the presence of co-occurring conditions.

Early clinical presentations of ASD as well as changes in the symptomatology over the course of the lifespan are reviewed below.

### *Social Communication and Social Interaction*

Impaired social communication and social interaction are a defining feature of ASD (Carter et al., 2005; see Chap. 3). While early abnormalities in the social domain have been documented as early as the first 6 months of life (Jones & Klin, 2013; Chawarska et al., 2013), in most cases social symptoms become fully manifest during the second year, with research showing that by 18 months of age children with ASD show reduced eye contact (Chawarska et al., 2014); reduced responsivity to human voices, including to their name being called (Miller et al., 2017); reduced interest in observing social scenes (Chawarska et al., 2013); and reduced frequency of imitation (Vivanti & Hamilton, 2014) as well as impairments in joint attention (i.e., the ability to shift attention between a referent and a social partner; Adamson, 1995; Scaife & Bruner, 1975). Joint attention behaviors that are reduced or absent in ASD include using gestures and eye contact to direct another person's attention to an object or event (e.g.,

looking at the caregiver and pointing to a butterfly), as well as using others' gaze and head turns to identify the target of their attention (e.g., looking at a flower that the caregiver is pointing to). These behaviors, referred to as initiation and response to joint attention, respectively, are linked to subsequent communicative, social–emotional, and cognitive development in typical and atypical development (Bottema-Beutel, 2016; Mundy, 2016). Children with ASD show deficits in both initiation and response to joint attention (Sigman, 1998; Jones et al., 2014), with more pronounced impairments in the initiation component (Mundy, 2011). Recent research has documented that 10-month-old infants later diagnosed with ASD show fewer episodes of initiation of joint attention compared to their peers (Nystrom et al., 2019).

Importantly, many children with ASD show behaviors that are consistent with a classification of "secure attachment" with their caregivers, such as responding differently to caregivers as compared to strangers (Kasari et al., 1993; Sigman & Ungerer, 1984), seeking and maintaining proximity with caregivers after separation (Sigman & Mundy, 1989), engaging in more proximity-seeking behaviors toward their caregiver, and engaging in fewer explorative behaviors in the presence of a stranger (Dissanayake & Crossley, 1996). The presence of these social and affective manifestations toward caregivers may contribute to lower than expected referrals for evaluations by physicians even after a child screens as being at risk for ASD (Pierce et al., 2011). However, proximity-seeking behaviors with caregivers in children with ASD may be less frequent, less sustained, or less conducive to rich social exchanges (Vivanti & Nuske, 2017). Young children with ASD also show diminished engagement in social play routines such as peekaboo (Bolton et al., 2012), as well as reduced pretend and imaginative play (e.g., Barbaro & Dissanayake, 2013).

Additional features include reduced interest in and engagement with peers (McGee et al., 1997), reduced responsivity to others' emotions (Hutman et al., 2010), difficulties interpreting other people's behavior (such as incorrectly predicting behavior based on their own knowledge, rather than others' knowledge; Baron-Cohen, 1995; Senju, 2012), and fewer expressions of pleasure during social interactions (Wan et al., 2013). In many cases, children with ASD do initiate and respond to social contact but their interactions are idiosyncratic or "on their terms" (e.g., approaching unknown people to do "high five" multiple times; Rapin, 2002).

Spoken language is often delayed—and in approximately 25–30% of cases never develops (Tager-Flusberg & Kasari, 2013). While delayed or absent verbal communication does not discriminate ASD from other conditions, a distinctive feature of children with ASD is a lack of attempt to compensate speech deficits with gestures or other forms of nonverbal communication. Abnormal speech patterns, odd words or phrases, echolalia, and unusual tone or pitch in vocalizations or words are other distinctive characteristics of early language in ASD and may persist throughout the lifespan (Kim et al., 2014). Many children with ASD show an increase in their use of communicative speech from childhood to adolescence and adulthood, although the social use and social understanding of communication (pragmatics) is likely to remain impaired. For example, children with ASD might fail to understand others' communication when the communicative intent differs from the literal meaning of a sentence (e.g., irony and sarcasm; Happé, 1994).

Similarly, social and communicative deficits may manifest differently at different developmental stages and in different contexts. At school age, as the child is exposed to increasingly complex social demands, deficits in developing, maintaining, and understanding relationships with peers (such as friendship) become more relevant. Many individuals with ASD learn coping strategies to adapt to their social environment, but social symptoms typically continue to persist throughout the lifespan. For example, in adolescence and adulthood, social interactions may increase in frequency but retain a qualitative impairment (stilted, exaggerated, or "socially awkward" behavior). For many individuals with ASD interest in social contact increases during adolescence and adulthood, including the desire to have friends and romantic relationships, and difficulties with accomplishing these social goals might cause frustration and affect well-being (Sperry & Mesibov, 2005). Experiencing the social world as indecipherable, frustrating, or rejecting can lead individuals with ASD to "give up" on social interactions, which leads to fewer opportunities for developing social skills, further limiting social opportunities and exacerbating social difficulties. See Chap. 3 for an in-depth overview of social communication development and intervention in ASD.

## *Restricted, Repetitive Patterns of Behavior, Interests, or Activities*

Repetitive and restricted behaviors (RRBs) are a heterogeneous group of symptoms characterized by rigidity and resistance to change. These symptoms include behaviors characterized by stereotyped or repetitive movements (such as hand-flapping and repetitive use of objects) or repetitive speech (echolalia and idiosyncratic phrases) and restricted, fixated interests that are unusual in their intensity or focus (such as strong attachments to a specific object, circumscribed or perseverative interests, and excessive focus on one conversation topic). Other features of ASD that fall within this domain are the "insistence on sameness" originally described by Kanner (such as the desire to watch the same episode of a TV show repeatedly or to eat the same food every day) and the rigid adherence to routines or rituals (e.g., singing the song "five little monkeys" while lining up five objects every morning at childcare). Seeking of specific sensations (such as deep pressure), unusual sensory interests (such as peering at or smelling objects), and hyper- or hyporeactivity to sensory inputs (e.g., experiencing everyday sounds as being excessively loud or imperceptible) are also classified as RRBs.

RRBs appear to emerge during infancy and persist across the lifespan, although they might manifest differently at different ages, and their prevalence and severity are extremely variable in the ASD population (Ozonoff et al., 2008; Uljarević et al., 2017). Unlike social symptoms, RRBs are not a distinctive feature of ASD, as some types of repetitive behaviors are observed in intellectual disabilities, psychiatric disorders, neurological conditions, and other clinical conditions with sensory deficits,

as well as in typically developing toddlers (Evans et al., 1997). However, individuals with ASD appear to engage in RRBs more frequently compared to those with other conditions, and their RRBs are expressed across a wider range of behaviors (Leekam, Prior, & Uljarevic, 2011).

Several criteria have been proposed to classify RRBs into specific subtypes, such the distinction between lower order (which include finger flicking, body rocking, and hand flapping) and higher order RRBs (including circumscribed interests and fascinations/preoccupations for particular topics, such as trains, wasps, or the French Revolution; Bodfish, 2007) as well as the distinction between repetitive sensory-motor (such as motor body mannerisms, repetitive use of objects, and unusual sensory interests), and "insistence on sameness" behaviors (including rituals and resistance to change; Richler et al., 2007; Prior & Macmillan, 1973). Rather than being mutually exclusive, the proposed subtypes appear to be characterized by permeable boundaries, with cognitive and language functioning influencing the manifestations of different RRBs (Leekam et al., 2011). For example, repetitive use of objects (e.g., spinning the wheels of a toy car) or motor stereotypies such as finger flicking or hand flapping are more likely to occur in children with ASD with lower versus higher cognitive functioning, whereas circumscribed interests are more prevalent in individuals with normative IQ (Bishop, Richler, & Lord, 2006). There is emerging evidence that repetitive behaviors might decrease in frequency and severity with age, independently of the cognitive level, with the exception of stereotyped movements that tend to be more persistent in individuals with co-occurring autism and intellectual disability (Esbensen, Seltzer, Lam, & Bodfish, 2009). See Chap. 4 for a detailed review on the nature and treatment of RRBs in ASD.

## *Gender Differences in Clinical Presentation*

Research on gender differences in ASD phenotypes has mixed evidence, partly because fewer female participants have been involved in research studies compared to males. Evidence from large twin studies suggests that males may require greater familial etiologic load to manifest the ASD phenotype (Robinson et al., 2013). Consistent with this notion, a more severe presentation has been reported in females compared to males (Christensen et al., 2016; Lord & Schopler, 1985) and the sex ratio has been reported to be <2:1 in more severely impaired cases (Loomes et al., 2017). There is some evidence of increased social interest and approach in females versus males as well as female superiority in executive function and visuospatial processing tasks (Bölte et al., 2011; Dean, Harwood & Kasari, 2017; Harrop et al., 2018; Koyama et al., 2009)—however, counterevidence exists (see Ferri, Abel, & Brodkin, 2018, for a recent review). It is possible that gender differences in behaviors related to ASD diagnosis (e.g., social approach) and camouflage of autistic symptoms (i.e., imitation of neurotypical peers to mask ASD symptoms) may result in under- or misdiagnosing among females on the milder end of the spectrum (Mandy et al., 2012), although more research is needed to corroborate this notion.

## Co-Occurring Conditions

Autism frequently co-occurs with other developmental, psychiatric, or medical conditions, which might exacerbate the symptoms, aggravate impairment, and complicate diagnosis and treatment. Intellectual disability, which was once thought to be present in most individuals with ASD, is now estimated to occur in 30–50% of children diagnosed with ASD (Charman et al., 2011; Christensen et al., 2018; Postorino et al., 2016; Rydzewska et al., 2018). This change might reflect advances in early detection and improved access to early intervention, as well as increased awareness and diagnostic expertise improving the identification of ASD symptoms in individuals without intellectual disability. While cognitive functioning and ASD symptomatology are conceptually distinct constructs, recent research suggests that children with more severe ASD symptoms are more likely to have co-occurring cognitive impairments (Gotham, Pickles, & Lord, 2012; Vivanti et al., 2013), potentially reflecting a link between barriers to social learning posed by severe social/communicative impairments and developmental delays (Vivanti, Dawson, & Rogers, 2017).

The extreme heterogeneity in intellectual functioning within the ASD population, ranging from severe intellectual disability to above average IQ, results in different challenges and priorities with regards to intervention. Additionally, such variation can be a source of confusion for families, especially those with more cognitively impaired children, who might fail to see the connection between the needs of their children and those expressed by cognitively able individuals with ASD (e.g., self-advocates with ASD who emphasize intelligence as a key feature of ASD and challenge the utility of interventions focused on "normalizing" behavior; Cascio, 2012).

Additionally, ASD co-occurs with several psychiatric disorders (including anxiety, obsessive-compulsive disorder, depression, attention-deficit hyperactivity disorder [ADHD], and oppositional-defiant disorder) more frequently compared to rates in the general population (Rydzewska et al., 2018; Simonoff et al., 2013). The high frequency of mental health difficulties in ASD might reflect (1) shared pathophysiology (i.e., what causes ASD also causes or increases the risk for the comorbid condition); (2) the downstream consequence of growing up with ASD (e.g., coping with social demands that exceed processing capacity, peer rejection, and other negative life experiences stemming from the stigma associated with ASD symptoms); (3) shared symptom domains or overlapping diagnostic criteria; or (4) an interplay between these different factors (Postorino et al., 2017; Uljarevic et al., 2016). Additionally, ASD co-occurs at a higher rate than the typical population with tics and Tourette Syndrome (Canitano & Vivanti, 2007; Kalyva et al., 2016), sleeping disturbances (Souders et al., 2017), eating disorders and restricted and rigid food choices (Sharp et al., 2013), gastrointestinal issues (Holingue et al., 2018), elimination problems (Gorrindo et al., 2012), and most notably, epilepsy, which might be present in up to 25% of the ASD population and is more frequently present in females and more cognitively impaired individuals (Jeste & Tuchman, 2015; Volkmar & Wiesner, 2017).

## Etiology

Genetic factors play a critical role in the etiology of ASD, with recent research indicating that approximately 80% of ASD risk is heritable (Bai et al., 2019; Tick, Bolton et al., 2016). Twin and family studies suggest that siblings of children with ASD have a 20–50-fold increased risk of having ASD compared to the general population, and the risk is increased for those with more than one sibling with ASD, while the concordance rate in identical twins is up to 90% (Colvert, Tick et al., 2015; Rutter, 2000). Research has pointed to a large number of common and rare genetic variants associated with ASD risk, but none of them accounts for more than a minority of cases, suggesting that the risk of developing ASD might be predominantly related to the additive contributions from common and rare variants that, individually, are not sufficient to cause the condition (Arnett, Trinh, & Bernier, 2019; Ramaswami & Geschwind, 2018; Weiner et al., 2017).

While this body of research indicates that ASD is one of the most heritable neurodevelopmental conditions, the lack of complete concordance in monozygotic twins indicates that nongenetic factors also contribute to ASD. Environmental factors that have been associated with increased risk for ASD include advanced maternal age (≥40 years) and paternal age (≥50 years; Lyall et al., 2017) as well as preterm birth (Lampi et al., 2012). Additionally, there is some indication that short interpregnancy intervals (<24 months; Zerbo et al., 2015) and maternal hospitalization during pregnancy (Lyall et al., 2017) might be associated with the risk for ASD, although more research is needed to corroborate these initial findings.

The hypotheses that ASD could be caused by vaccines or poor parenting have been repeatedly tested and unequivocally disconfirmed (Capps, Sigman, & Mundy, 1994; Lord et al., 2018; Parker, Schwartz, Todd, & Pickering, 2004; Ventola et al., 2017).

## Neurocognitive Models of ASD

While the nature of the neurocognitive processes underlying the ASD phenotype remains unclear, several theoretical models have been proposed. Historically, these have included (a) the notion that ASD symptoms reflect difficulties in the ability to attribute mental states to others, or "theory of mind" (Baron-Cohen, Leslie, & Frith, 1985), (b) the "weak central coherence" account, i.e., a detail-focused processing style, whereby information is processed in terms of constituent parts, rather than global meanings (Frith & Happé, 1994), and (c) deficits in executive functioning which causes difficulties in the flexible organization of behavior across social and nonsocial domains (Ozonoff, 1997). While each of these accounts have contributed critical insight on several phenomena related to ASD, none of them is universally considered to provide a satisfactory account for constellation of features observed in ASD (Frith, 2012).

More recently, theoretical models have been steering away from "single deficit" explanations to focus on the developmental pathways that lead from early risk factors to specific features of ASD (Vivanti, Yerys & Salomone, 2019). Recent accounts informed by longitudinal research on infants at risk for ASD include the notion that social symptoms reflect the downstream consequence of early disruptions in the reflexive, orienting mechanisms that drive newborns' attention toward social stimuli (Di Giorgio et al., 2016), or, alternatively, disruptions in the developmental transition from reflexive orienting to volitional attentional engagement with the social world that occurs during the first 6 months of life (Klin et al., 2015; Shultz et al., 2018). Diminished attentional engagement with social stimuli has been proposed to originate either from domain-specific abnormalities in social–cognitive processing (whereby social stimuli are not experienced as meaningful or interpretable; Leekam, 2016), diminished social motivation (whereby social stimuli are not experienced as rewarding; Chevallier et al., 2012; Mundy, 1995), or widespread nonspecific abnormalities that disrupt social processing because of the inherent complexity and unpredictability of social stimuli compared to nonsocial aspects of the environment (Minshew & Goldstein, 1998; Johnson, 2017). Research does not provide unequivocal support to any of these explanations, with some findings pointing to widespread network abnormalities in infants with ASD, and other research pointing to specific abnormalities within the social brain network, including atypical brain activity in response to social versus nonsocial stimuli and rewards (Pelphrey et al., 2014).

While the causal pathways in ASD remain unclear, these converging lines of inquiry suggest that an altered engagement with social stimuli during early sensitive periods might affect neural specialization and behavioral expertise in the social domain, thus exacerbating initial abnormalities in an iterative fashion (i.e., as children with ASD are less engaged with the social world due to initial biological constraints, they have fewer opportunities to learn and practice social and communication skills, thus failing to build a behavioral repertoire from social experience). A corollary of this developmental perspective is that targeted intervention during early sensitive periods has the potential to mitigate or, according to some scholars, prevent this escalating deviance from typical social development (Dawson, 2008; Vivanti, Dawson, & Rogers, 2017). This topic will be the focus of the remainder of the chapter and of this manual.

## Treatment for Autism Spectrum Disorder: Evolution of Concepts and Principles

No pharmacological treatment has been shown to address the core symptoms of ASD, and currently recommended approaches to early intervention for ASD rely on various applications of behavioral, developmental, and special education principles (Lord et al., 2018; Volkmar et al., 2014). The historical evolution of early interven-

tion for ASD is marked by the influence of the different theoretical frameworks that have been prominent within disciplines concerned with ASD at different stages.

## *Influences from Psychodynamic Theory*

The earliest approaches to ASD intervention, in the 1950s and 1960s, were predominantly informed by psychodynamic theory, and in particular by the notions that mother–child dynamics play a critical role in psychopathology (Freud, 1911), and that autistic symptoms are the consequence of inadequate parenting (Bettelheim, 1967; Green & Schecter, 1957; Kanner & Eisenberg, 1956; but see Anna Freud, 1969/2015 for a dissenting voice within the psychoanalytic community). Interventions derived from this framework included psychodynamic psychotherapy to the child and the child's mother, as well as "parentectomy," which involved the separation of children from their supposedly unloving families and their placement in psychiatric residential institutions. The psychodynamic approach to ASD was discredited in the following decades both because of unsatisfactory scientific support and ethical considerations, although various elaborations of the same theoretical framework continued to be perpetuated throughout the 1990s (e.g., Tustin, 1991) and continue to be influential in many countries (Severson, Aune, Jodlowski & Osteen 2008).

## *Influences from Applied Behavior Analysis*

In the mid-1960s, applied behavior analysis (ABA; Baer, Wolf, & Risley, 1968) became a major influence on ASD intervention as Ferster (1964) and Lovaas (1968) and other ABA pioneers introduced the use of practices based on operant conditioning (Skinner, 1953) in the field of ASD. A key concept introduced by the ABA work is the notion that the behavior of children with ASD obeys the same laws that shape learning in typical development (Ferster & DeMyer, 1961; Lovaas & Smith, 1989; Mayville & Mulick, 2011), whereby behaviors followed by a positive consequence (reinforcement) strengthen and are more likely to be produced when the stimulus that triggered the behavior (antecedent) is presented, while those followed by a negative consequence result in an opposite effect. A critical corollary of this notion is that specific behaviors can be encouraged or discouraged through a careful manipulation of antecedents and consequences. Additional influential elements introduced by ABA include the emphasis on unambiguous operational definitions of intervention goals and targets (i.e., promoting the acquisition of clearly defined desirable behaviors and discouraging clearly defined maladaptive behaviors), and the use of empirical data as the platform for decision making and evaluation of outcomes. Early applications of these principles included procedures that have largely been abandoned because of ethical and scientific considerations, such as the use of

electric shocks to induce behavior modifications as described by Lovaas and colleagues (1965), which was later defined by Lovaas as "a mistake based on erroneous beliefs" (Lovaas, 1989). Nevertheless, the shift in focus from the psychoanalytic emphasis on unobservable unconscious dimensions to the data-driven observation and manipulation of well-defined behaviors provided the infrastructure for most contemporary approaches to ASD interventions, becoming the foundation for the highly structured approaches known as discrete trial teaching (Lovaas, 1981), as well as subsequent applications of ABA (Schreibman et al., 2015), including the early intervention approach known as early intensive behavioral intervention (EIBI; see Chap. 5).

## *Influences from Developmental Psychology*

The application of concepts from developmental psychology in the field of ASD became another major influence in ASD early intervention practices starting in the 1980s. A concept introduced by developmental literature that became relevant to ASD intervention is constructivism (Bruner, 1978; Montessori, 1912; Piaget, 1929; Vygotsky, 1930–1934/1978), i.e., the notion that skill acquisition during early development is built on the child's active, self-directed engagement with a stimulating physical and social environment, with knowledge gained at specific developmental stages supporting the transition to more advanced developmental stages which enable the acquisition of complex skills. The adult's role in this context is to facilitate skill acquisition through scaffolding (Bruner, 1978), i.e., supporting the engagement of the child in joint routines that build upon the child's initiative and current knowledge to generate opportunities to gain more advanced knowledge (a concept analogous to the construct of 'zone of proximal development', formulated by Vygotsky in the 1930s; Vygotsky, 1930–1934/1978). For example, during shared storybook reading routines, adults scaffold the child's acquisition of new knowledge by establishing a joint focus (the illustrations in the book), highlighting the relationship between the words, images, and emotions associated with the events in the story, eliciting children's verbal and nonverbal responses (i.e., emotional expressions, gestures, words) through pauses and questions, and providing feedback, such as asking for clarification or recasting the child's utterances (Ninio & Bruner, 1978). Pivotal skills in the domains of communication, joint attention, and imitation that are learned during joint activities provide the foundation for engagement in more complex social exchanges, which, in turn, enable the acquisition of further knowledge. A critical corollary of this framework is that advances in some pivotal or prerequisite skills will facilitate the acquisition of more advanced skills.

These concepts from developmental literature became increasingly influential in ASD intervention as research started to show that developmental sequences of skill acquisition in children with ASD appear to follow the same path observed in those with typical development. For example, development of verbal language is predicted

by joint attention skills both in typical children and in those with ASD, and improvements in joint attention result in language improvements (Bottema-Beutel, 2016; Kasari, Sigman, Mundy, & Yirmiya, 1990; Sigman et al., 1999; Whalen, Schreibman, & Ingersoll, 2006). Developmental concepts that continue to be prominent in ASD early intervention include the use of "developmentally appropriate" treatment targets and procedures (e.g., teaching developmental prerequisite to facilitate acquisition of more complex skills), the involvement of families and daily routines as a privileged context for learning, and the focus on scaffolding over didactic teaching to promote skill acquisition in early development (Rogers, 1999; Schreibman et al., 2015).

## *Influences from Neuroscience*

Starting from the late 1990s, ASD intervention has been influenced by social neuroscience research in typical development and ASD—i.e., the study of how social information and social experiences shape and are shaped by brain development (Lieberman, 2007). A notion from this field that is of prominent relevance in ASD early intervention is that the neural infrastructure of human sociability relies on an "experience-expectant" process. This means that "hardwired" brain mechanisms bias children to actively seek out opportunities for social engagement during early development. These early social experiences, in turn, provide the input needed for the brain to become specialized in social information processing, which then enables participation in and learning from more sophisticated social interactions (Brownell, 2013; Sullivan, Stone, & Dawson, 2014).

While research in this area is in its infancy, several interconnected brain areas and circuitries involved in this iterative process have been identified. This includes social–cognitive structures responsible for the quick processing of social–emotional cues such as gaze direction and emotional expressions (Frith & Frith, 2010), the social reward system, which underpins the experience of pleasure associated with social interaction (Dolen, Darvishzadeh, Huang, & Malenka, 2013), and the mirror neuron system (Rizzolatti & Sinigaglia, 2008). The mirror neuron system consists of a set of brain regions that activate during action execution as well as during the observation of the same action. This mechanism is thought to enable the observer to understand others' actions and emotions as if she/he would be doing a similar action or experiencing the same emotion, thus facilitating the processing of shared experiences. While the translation of neuroscientific findings into treatment practices is in the very early stages, the influence of social neuroscience perspectives on intervention includes the emphasis on engaging the child in typical social routines during early critical periods of brain plasticity, increasing the reward value of social exchanges, and supporting the child to register the correspondences between his/her and others' actions during shared experiences (Rogers, Vivanti, & Rocha, 2017; Vivanti & Rogers, 2014).

## *Influences from Implementation Science*

A more recent influence on ASD early intervention is the emerging field of implementation science, which studies the factors that facilitate adoption and successful implementation of interventions in the "real world" (Eccles et al., 2009; Green, 2012). This field focuses on features such as organizational systems of the context in which interventions take place, as well as beliefs, cultures, and the "buy-in" of professionals involved in the intervention. A critical notion derived from this research is that the commitment to use intervention techniques and implement them as intended is not only dependent on the level of evidence supporting the intervention, but also on the perceived fit between features of the intervention and stakeholder attitudes (whether they think that using the intervention is the right thing to do), norms (the extent with which they feel they are expected to use the intervention, or believe that their peers are using it), self-efficacy, and the perception of the cost-benefit of adding new approaches to their standard practice. These factors will affect the initial uptake of the intervention, maintenance over time, and the degree of integrity to which it is delivered, thus affecting intervention outcomes. Recent early intervention practices for ASD influenced by implementation science include the introduction of participatory research methods, in which administrators', practitioners', and end users' resources and preferences are examined and taken into consideration from the early stages of designing and piloting new interventions (Dingfelder & Mandell, 2011; Locke et al., 2016; Smith et al., 2007).

## *Influences from the Neurodiversity Framework*

A recent societal influence that has affected debate on intervention for ASD is the concept of neurodiversity, which refers to the notion that neurological differences are to be recognized and respected as any other human variations (den Houting, 2019; Baron-Cohen, 2017), rather than being seen as indicators of a pathology to be corrected or eradicated. Some self-advocates with ASD who adopt this perspective question the need for curing or "normalizing" autism, arguing that the goal of increasing "desirable" and decreasing "undesirable" behaviors in intervention reflects the parameters of what is desirable or undesirable set by a "neurotypical" majority. Further, this approach to treatment has been criticized as being designed to achieve conformity at the expenses of diversity, rather than serving the best interest of those with ASD. According to this perspective, ASD is a way of being in the world, or a culture, which requires support and appreciation, rather than prevention or treatment (Norbury & Sparks, 2013; see also Mesibov, Shea, & Schopler, 2004, for an early application of the concept of autism as a culture for treatment).

Importantly, other advocacy groups are not opposed to the idea of intervention per se, but emphasize the importance of practices aimed at increasing opportunities for self-determination and eliminating environmental and social barriers to

civil rights and inclusion in individuals in the autism spectrum. While the positions expressed by neurodiversity advocates are multifaceted and highly debated, especially in the context of the heterogeneity of intervention needs within the ASD population, there is a growing focus on researching treatment methods and outcomes that include preferences and perspectives of individuals with ASD (Pellicano & Stears, 2011; Vivanti, 2020).

## Conclusions

Despite the tremendous increase in research in the past decades, many aspects of ASD continue to remain enigmatic. As it has become clear that ASD does not result from a single disruption or etiological pathway, the challenge for early intervention research is to derive practical knowledge from the examination of the interplay between biological, neuropsychological, and environmental factors underlying the heterogeneous and seemingly unrelated clusters of symptoms and associated features that characterize the autism spectrum. Additionally, issues on appreciation of diversity, human rights, and self-determination are increasingly stimulating societal debate and challenging the established intervention paradigms. Despite these challenges, it can be concluded that the current landscape of early intervention for ASD has been shaped by progression from the widespread use of unethical and unsupported treatments to the increasing appreciation and integration of evolving scientific knowledge and more inclusive views on ASD.

While no area of early intervention for ASD is free from debate, and divergences among different schools of thought continue to exist, early interventions for ASD that have proven to be effective converge around a set of key features. These include the engagement of the child in a planned educational/psychosocial intervention that (1) starts early in life, (2) is developmentally appropriate, (3) is implemented throughout the child's day, (4) uses well-defined instructional strategies to target the core features of autism and address functional/adaptive skills, (5) includes systematic assessment procedures to define individualized sets of goals tailored to the individual child's profile of strength and needs, (6) includes a data collection system to monitor progress, and (7) involves caregivers in establishing treatment goals and delivering intervention strategies. Within this framework, different interventions exist, which will be described in detail in the following chapters.

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