Autism and Child Psychopathology Series
Series Editor: Johnny L. Matson

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Peter Sturmey
Editors

Handbook of Autism and Pervasive Developmental Disorder

Assessment, Diagnosis, and Treatment

Springer

Autism and Child Psychopathology Series

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Brief Overview

The purpose of this series is to advance knowledge in the broad multidisciplinary fields of autism and various forms of psychopathology (e.g., anxiety and depression). Volumes synthesize research on a range of rapidly expanding topics on assessment, treatment, and etiology.

Description

The **Autism and Child Psychopathology Series** explores a wide range of research and professional methods, procedures, and theories used to enhance positive development and outcomes across the lifespan. Developments in education, medicine, psychology, and applied behavior analysis as well as child and adolescent development across home, school, hospital, and community settings are the focus of this series. Series volumes are both authored and edited, and they provide critical reviews of evidence-based methods. As such, these books serve as a critical reference source for researchers and professionals who deal with developmental disorders and disabilities, most notably autism, intellectual disabilities, challenging behaviors, anxiety, depression, ADHD, developmental coordination disorder, communication disorders, and other common childhood problems. The series addresses important mental health and development difficulties that children and youth, their caregivers, and the professionals who treat them must face. Each volume in the series provides an analysis of methods and procedures that may assist in effectively treating these developmental problems.

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Part I
An Introduction and Overview to Autism and Pervasive Developmental Disabilities

Autism across the Ages: An Abbreviated History

Sarah J. Macoun, Buse Bedir, and John Sheehan

This chapter summarizes key scientific developments in autism, presented within the context of the world views of the time in which those developments were occurring. While an exhaustive review of every key scientific figure, clinician, advocate, and policy development is beyond the scope of this chapter, we highlight key influences on the evolution of scientific understanding in the field. The heterogeneity of autism has generated distinct conceptualizations, theorizations, debates, and unique challenges for research. It is clear that historical perspectives have strongly influenced conceptualizations of autism, including the questions posed and the lens through which data is interpreted. Research into autism has proliferated since the 1960/70s due to rising awareness that autism can respond to treatment, advances in diagnostic tools, and improved technologies for studying the brain and genetics. While there is still much to be learned, we can be encouraged by the significant progress that has been made since the disorder's formal introduction in 1943 (1943), that research into the disorder has not slowed (Graff et al., 2014), and that outcomes for individuals with autism have improved substantially (DeMyer et al., 1973; Howlin & Magiati, 2017; Howlin et al., 2013).

It should be noted that although this chapter has been organized into 'eras' this does not imply that the figures and developments reviewed within those eras are exclusive to that particular time. Although shifts from one paradigm to the next do not have sharp transition points, it is important to recognize how beliefs that have come before impact the formulation and ongoing interpretation of a given era's theories. Further, as historical accounts such as those by Grinker (2007) or Waltz (2013) point out, the persistence of differences amongst various cultures with respect to interpretation of symptoms, even with the growing standardization of norms in neurodevelopmental science, suggests that any reading of the historical

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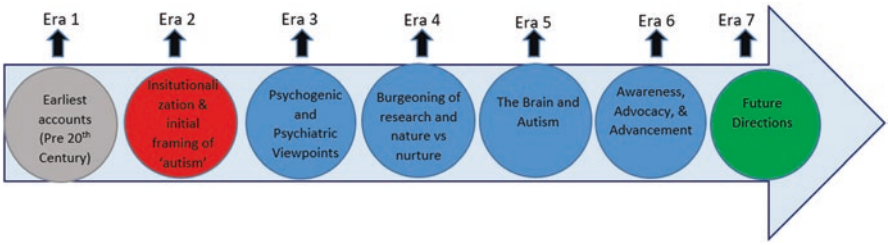

Fig. 1 Timeline of developments in autism

record should be done so with tremendous caution and an awareness of biases both known (e.g., skewing towards modernized and Westernized conceptualizations) and unknown (Fig. 1).

ERA 1: Earliest Accounts (Pre-Twentieth Century)

Although historical accounts of individuals believed to meet criteria for what is now considered an Autism Spectrum Disorder (ASD; what will hereafter be referred to as 'autism') are typically traced back only a few centuries, modern biological conceptualizations of autism would suggest its presence through much of human history. There are diverse reasons as to why historical accounts of autism do not stretch back further, one reason being that the signs and symptoms of autism are heterogeneous and overlap with other conditions thus complicating identification and interpretation. Only relatively recently has there been a transition towards more scientific approaches to understanding the underlying mechanisms of autism although this has, at times, been a non-linear and regressive endeavor. In pre-twentieth century accounts, aberrations from 'typical' patterns of development and behavior, including features of autism, were explained through the lenses of religious dogma and folklore. The *Age of Enlightenment* (approx. 1680–1799) brought a more medicalized understanding of disability with a shift towards the scientific method and away from religious- and folklore-inspired conceptualizations of disability (Waltz, 2013). While this shift represented an important advancement, a careful reading of subsequent history sheds light on both the limitations in how the scientific method was applied, as well as the resilience of earlier deep-seeded beliefs regarding the ultimate attributability of disability to parents or the individuals themselves.

Frith and Houston (2000) painstakingly created a 'case report' for Hugh Blair of Borgue (1708–1765), the son of a Scottish landowner who, at the age of 39, appeared in court for a legal dispute with his brother that called his mental capacity into question. As part of their work, Frith and Houston analyzed the depositions of 29 witnesses and re-constructed Blair's developmental and behavioural history, which they subsequently evaluated against the criteria for autism. There were no indications that Blair had been exposed to early neglect or serious illness, eliminating

common alternative explanations of the time. Blair attended school for many years and could accomplish some activities of daily living. He was reported to have limited social understanding, with atypical eye gaze and language (e.g., possible echolalia). Blair also displayed atypical motor movements/mannerisms, an unusual interest in collecting feathers and sticks, and a preference for sameness that extended to church and home. Other reports they reviewed, such as those describing a lack of common sense and a "silent madness," may have been alluding to general limitations in adaptive functioning and/or intellect. Beyond the itemizing of signs and symptoms, Frith and Houston (2000) also vividly illustrated what daily life was like for Blair, providing the reader a glimpse into how individuals with neurodevelopmental concerns were treated during Blair's time. Frith and Houston (2000) noted that Blair was given the latitude to engage in work that would typically be considered unfit for a 'gentleman, yet emphasized the diversity of outcomes for individuals with neurodevelopmental concerns. Circumstances for such individuals before the nineteenth century could include living with family, various forms of institutionalization, or even being left to 'wander aimlessly', as was the case for Jean-Marc-Gaspard Itard's (1775-1838) 'Victor.'

Victor, more widely known as The Wild Boy of Aveyron, experienced severe neglect and has since been proposed as an early example of autism. Initially described in Bonaterre's letter to a French journal (Itard et al., 1932), Victor was discovered in the woods in 1798 at the age of approximately 12 years. He was described as having a shifting gaze, a lack of expression, no language, being indifferent to the elements, and showing no preference for 'pleasing' (Uta Frith, 2003; Wing, 1997). Although he was described as generally melancholic, there were outbursts of laughter and joyous responses to unusual things (e.g., the sun, bright moon (Malson, 1972). Despite an initial determination by a physician that Victor's symptoms were immutable, Itard developed a behavioural program to assist with his emotional, social, and language development through carefully graded activities (Itard et al., 1932). As a result of this revolutionary program, Victor learned some verbal phrases, primitive written language, and to distinguish emotions, albeit with less success than Itard had originally envisioned. This led Itard to conclude that, although Victor's accomplishments provided some evidence for the efficacy of his techniques, perhaps there were 'critical periods' for learning social and language skills that Victor had missed due to extreme isolation (Frith, 2003; Wing, 1997).

Another well-known pre-twentieth century description of potential autism is represented in John Haslam's description of a young boy of about 6 years who presented with a history of infantile convulsions, severe measles, and motor/speech delays (Feinstein, 2010). In his 1809 book, *Observations on Madness and Melancholy*, Haslam described the boy as being restless and inattentive in hospital, albeit with a general curiosity relating to that environment, poor spatial distance judgement and, interestingly, as lying to hide his misdeeds (Wolff, 2004). Follow-up at age 13 did not identify abnormal gaze or echolalia, although he referred to himself in the third person. He did, however, display some atypical behaviors and had tendencies towards social isolation (e.g., watching others play, but not joining) and obsessive preoccupations (e.g., only wanting to discuss toy soldiers (Vaillant, 1962;

Wolff, 2004). While the case can surely be made that Haslam's description aligns with modern autism, others, such as Wolff (2004), have identified such alternative possibilities as a post-encephalitic syndrome.

While the case studies above help to illustrate some of the benefits that were emerging from the increasing utilization of a medicalized model of disability, it unfortunately remained entangled with longstanding folklore and/or religiously-inspired notions of sin, punishment, and/or a need to protect society from anyone with atypical presentations. The mixed conceptualization of neurodevelopmental and other medical concerns contributed to a shift towards institutionalization, given that either of these lenses (i.e., a medicalized need to be 'cured' or a dogmatic-inspired need to be 'cleansed') tended to inevitably result in the same recommendation; prolonged institutionalization (Waltz, 2013). Thus, throughout the eighteenth, nineteenth, and well into the twentieth centuries, asylums and workhouses became the mainstream answer to concerns relating to mental health, neurodevelopment, and/or 'immorality'.

Era 2: Institutionalization and the Initial Naming & Framing of 'Autism'

There are crucial and nuanced ways in which institutionalization impacted the initial and subsequent conceptualizations of autism and had lasting effects upon client-clinician dynamics. Institutions were utilized primarily for adults for centuries prior to their more widespread use with children. This was due, at least in part, to a general lack of understanding regarding the 'typical course' of normative child development and a lack of methods for comparison to developmental norms. It was not until the early twentieth century that children, outside of those displaying the most severe behavioral or medical concerns, also started to be institutionalized more commonly (Nadeson, 2007). In fact, there is a compelling argument to be made that it was actually this era of institutionalization, sometimes referred to as 'The Great Confinement' (Waltz, 2013), that is most responsible for how and why autism became identifiable as a disorder by the clinicians working within these institutions. This connection between setting, discovery, and conceptualization, however, has created a number of complicating and confounding factors that have required disentanglement ever since. While an exhaustive exploration of the specific history of institutionalization is beyond the scope of this chapter, the prominent and lasting role that it played within the history of this field warrants consideration (e.g., see Waltz (2013).

Originally coined by Swiss psychiatrist Paul Bleuler (1911), *autismus* (or, in English translation, *autism*) eluded to idiosyncratic or self-centered tendencies that Bleuler considered to precede social withdrawal in adult patients with schizophrenia (Goldstein & DeVries, 2013). This German term stemmed from the Greek *autos*, meaning 'self,' and *ismos*, denoting an 'action' or 'state.' Thus, Bleuler was

utilizing 'autism' to describe patients with schizophrenia who were intentionally departing from reality to cope with illogical or distressing thought processes (Maatz et al., 2015). In 1924, over a decade after Bleuler brought 'autism' into the lexicon of the psychiatric community, a Russian doctor named Grunya Efimovna Sukhareva examined a highly intelligent 12-year-old boy who was generally uninterested in the company of others and did not play with toys, but enjoyed engaging in philosophical discussions and had taught himself to read at age five (Manouilenko & Bejerot, 2015). Sukhareva went on to describe this boy as "an introverted type, with an autistic proclivity into himself" and, over the following year, she would identify five more boys with "autistic tendencies." These tendencies included social isolation, preference for their own inner worlds, and a unique talent and/or characteristic (e.g., giftedness in music or numeracy). Sukhareva published systematic observations of the six boys, which was translated in 2013, and is now considered to be an accurate and systematic description of DSM-5 criteria for ASD (Manouilenko & Bejerot, 2015). Finally, just prior to Kanner's writings in 1938, an American Psychologist, Louise Despert, published a series of case studies on 23 patients whom she classified as having childhood onset schizophrenia. Her clinical accounts described communication problems, emotional difficulties, and abnormal ways of thinking, without hallucinations (Fellowes, 2015; Louise Despert, 1938). Although recognition of autism as a disorder of childhood was clearly developing, treatments were extremely limited. Looking back, however, Itard could arguably be credited with reporting one of first behavioural approaches for teaching language and social conventions in autism (Itard et al., 1932).

In 1943, Leo Kanner described a previously unidentified, or at least unnamed, syndrome in his seminal paper, A*utistic Disturbances of Affective Contact*, which unquestionably elevated autism into the clinical and scientific literature (Kanner, 1943). Kanner, an Austrian Child Psychiatrist at John Hopkins, published clinical descriptions of 11 children who displayed a pattern of atypical behaviours that he termed 'infantile autism.' Kanner described a range of clinical features including social difficulties (e.g., difficulty relating to others, 'extreme autistic aloneness'), language anomalies (e.g., pronominal reversal, literal language, echolalia, inability to communicate, muteness, monotone speech), and repetitive/restricted behaviours (e.g., insistence on sameness, fascination with objects, repetitive behaviours). He believed that social isolation was a core feature of 'infantile autism' and noted secondary symptoms such as sensory aversions, strengths in rote memory, and enlarged head size. Kanner viewed 'infantile autism' as an innate psychiatric illness due to 'profound emotional disturbance'. Although he initially distinguished 'infantile autism' from childhood schizophrenia, due to differences in disease onset and progression, Kanner would later change his perspective and align with more common psychiatric viewpoints of the day (Kanner, 1949). Kanner's detailed clinical descriptions have stood the test of time and are surprisingly consistent with current conceptualizations of autism, except for his claims of normal intelligence. Kanner even went so far as to note similarities in behaviours between parents and children with autism, noting "a resemblance between their makeup and that of their children" and

foreshadowing research into the broader autism phenotype and heritability (Wolff et al., 1988).

In conjunction with his colleague, Leon Eisenberg, an American child Psychiatrist from Johns Hopkins, Kanner provided a methodology for the scientific study of autism. These authors would present over 100 cases in subsequent decades, adding to early scientific enquiry into autism on prognostic indicators, long-term outcomes, and differentiation of autism from other developmental disabilities (Eisenberg, 1955; Kanner, 1949; Kanner & Eisenberg, 1957). As an innate condition, Kanner believed that autism was essentially untreatable, noting that, although he believed that long-term institutionalization was a solution, it "cut short any prospects of improvement" (Kanner, 1964). Although there is no doubt that Kanner was a pioneer in the field, his work did contribute to misconceptions that would persist and create controversy for decades. First, by appropriating Bleuler's term 'autism' and by equating 'infantile autism' with childhood schizophrenia (Kanner, 1949), Kanner contributed to the perspective that autism was a disorder of childhood psychosis. Further, Kanner was influenced by psychoanalytic views of the day and his writings were, in fact, the first to introduce the misguided notion that cold and rejecting parenting caused autism (Kanner, 1943) .

Within a year of Kanner's, 1943 paper, Hans Asperger published an independent series of case studies (Asperger, 1944; Frith, 1991). Asperger was an Austrian Pediatrician and Professor who conducted research on mental disorders in children. His behavioural descriptors were eerily similar to that of Kanner's, although it is reported that the two never communicated directly with each other and it is uncertain as to whether they were familiar with each other's work (Lyons & Fitzgerald, 2007). Asperger used the term 'autistic psychopathy' and also likened the disorder to adult schizophrenia. Similar to Kanner's earlier views, Asperger differentiated 'autistic psychopathy' from schizophrenia, noting that 'autistic psychopathy' consisted of atypical personality features from birth, whereas childhood schizophrenia developed over time. Asperger's descriptions were of individuals who were more mildly affected, whereby he noted difficulty with social-emotional relations, narrow/restricted interests, extraordinary (savant) skills, intact language abilities, and atypical social-communication patterns. Asperger also observed that the parents of these children had similar traits to that of their child. Asperger further theorized that that 'autistic psychopathy' was a polygenetic phenomena that would lead to diverse presentations. Asperger's writings on autism were, in fact, decades ahead of his time but would prove to be much less influential than Kanner's. His works were not widely read for approximately 30 years until they were brought into the English science literature in the 1980s by Lorna Wing (Wing, 1981) and later translated from German to English in the 1990s by Uta Frith (1991). Also notable is that Asperger took somewhat of an educational and service oriented approach, as opposed to medical approach, in treating autism. He recruited his client's help to develop tailored strategies for teaching them and other children with similar tendencies, cognitive profiles, and learning preferences (Frith, 1991). These treatment recommendations would have deviated significantly from medical psychiatric approaches of the day, and as such were quite progressive.

Era 3: Psychogenic and Psychiatric Viewpoints

A key contributor to autism research in the 1950s was an American Child Psychiatrist working out of Bellevue Hospital (NY), Lauretta Bender (Faretra, 1979). Bender's views were influenced by the mindset of her generation and, like many researchers and clinicians before her, she considered autism to be an early form of childhood schizophrenia (Faretra, 1979). However, Bender also noted that autism and associated behaviours may be seen in children with other types of developmental and neurological disorders (Lauretta Bender, 1959). She further indicated that the type of 'schizophrenia' developed by the child depended on genetic factors, life experiences, age of onset, and other factors (Faretra, 1979). Bender extensively documented the behaviour of children with what she termed the 'autistic type of schizophrenia,' including deficits in speech and communication, inflexibility, withdrawal, and preference for aloneness (Bender, 1969). One of Bender's additional contributions was her observation of heritability patterns in mental illness, particularly schizophrenia and developmental disabilities, across families (Faretra, 1979). She noted that autism had a "chronic course" and conducted longitudinal studies which indicated that symptoms of 'autistic type schizophrenia' persisted into adulthood (Bender & Faretra, 1972). Bender's studies documented low intelligence test scores and poor language as predictive of a more severe and chronic course of the disorder (Bender, 1970). Bender was one of the earliest researchers to study possible therapy options for children with autism, with her group studying the effectiveness of intensive psychotherapy for children with autism using a close physical relationship and adjunct physiological therapy (Bender & Gurevitz, 1955). She was also a proponent of more invasive psychiatric treatments, including psychotropic drugs such as LSD (Bender et al., 1963) and electrically induced convulsions (Bender, 1947). Although these treatments would often prove more harmful than helpful, they do represent early attempts to treat autism and would set the stage for later treatment investigations.

Discussions of autism in the late 1950s and 1960s were often centered around the work of the famous, but highly controversial, Bruno Bettelheim (Severson et al., 2008), who was neither trained as a psychologist nor therapist. Born in Austria, Bettelheim's book, *The Empty Fortress* (Bettelheim, 1967), shifted the focus of autism research and treatment back to a parental blame model by discussing the concept of the "refrigerator mother." His argument was that the difficulties seen in children with autism were caused by emotionally rejecting parenting, especially cold mothers. In fact, Bettelheim's ideas were an extension of Kanner's, 1949 writings in which Kanner first claimed that most children with autism were exposed to a parenting style that was cold, obsessive, and mechanistic by only tending to the most basic needs of the child (Kanner, 1949). Bettelheim was a rigid proponent for psychogenic causes of mental disability and, although he did not produce empirical support for his claims, his pop-culture status and referencing of psychoanalytic principles, which were popular at the time, enabled his narrative to play a major role in autism research and treatment for an extended time. Decades of family trauma

and misguided treatment approaches, including the 'parentectomy,' whereby children were removed from their homes to residential facilities to be rehabilitated (Creak, 1963), would prove extremely damaging to these children and their families (Mesibov et al., 2000; Waltz, 2013).

In contrast to Bettelheim's claims about 'refrigerator mothers', UK-based child psychiatrist Mildred Creak studied organic contributions to child psychiatric and behavioral concerns. She published work that established a nine-point criteria for the diagnosis of autism, called "schizophrenic syndrome of childhood" at the time, which she considered to be a broader spectrum of infantile psychosis (Creak, 1963; Creak & Ini, 1960). Creak argued that autism was primarily due to genetics and was not influenced by parent behaviour, a deviation from the prevailing ideas of the time, but one that would set the stage for future research into organic causes of autism (Creak & Ini, 1960). The nine criteria established by Creak further allowed clinicians and researchers to more clearly identify autism symptoms and led to the development of more systematic and rigorous diagnostic criteria (Creak, 1963). Still, autism-related symptoms fell under the 'childhood schizophrenia' diagnosis in the first and second editions of the Diagnostic and Statistical Manual (DSM I/II 1950/68) (*Diagnostic and statistical manual of mental disorders: DSM-II*, 1968). In later years, as research methodologies were increasingly able to distinguish the defining features of social relatedness amongst affected children, it became clear that autism was a separate and distinct disorder from schizophrenia. Autism as a diagnosis in its own right first appeared within international disease classification systems in 1975 in the World Health Organization's International Classification of Diseases (Rutter et al., 1969). Here it was termed 'infantile autism' and fell under the boarder category of infantile psychosis (Rutter et al., 1969). Shortly thereafter, 'infantile autism' was introduced in the third edition of the DSM (American Psychiatric Association, 1980).

Although its distinction from schizophrenia was a significant milestone for autism, several questions remained regarding its proper conceptualization that would require parsing out over subsequent decades via revisions of diagnostic manuals. For example, the original conceptualization of infantile autism (described as a Pervasive Developmental Disorder, PDD) in DSM-III (American Psychiatric Association, 1980) emphasized structural language deficits, as opposed to the social use of language, and lacked a lifespan perspective regarding its course and impact (Goldstein & DeVries, 2013). Therefore, the subsequent revision of DSM-III (American Psychiatric Association, 1987) clarified that the newly termed 'autistic disorder' should be defined in relation to a child's mental age, while also removing diagnostic language that previously precluded older (i.e., dropping the required onset before 30 months) and higher functioning (i.e., addition of PDD-Not Otherwise Specified for milder presentations, a first move towards a 'spectrum' approach) individuals from diagnosis (Factor et al., 1989). The DSM-III revision (American Psychiatric Association, 1987) stands out as a crucial benchmark in the history of autism and present day researchers often use this point in time as essentially representing the birth of 'modern autism,' the implications of which will be addressed below.

Era 4: Burgeoning of Research and Nature Vs Nurture

The 1960s and 1970s saw a proliferation of Autism research as advances in behaviourism, neuroscience, and genetics facilitated a renewed motivation to investigate early identification, diagnosis, and treatment. The nature versus nurture debate, which weighed psychogenic versus organic causes for autism, was a crucial debate that also ignited during this time period. In 1961, Ferster and DeMyer (1961) published an account on the application of behavioral principles for increasing behavioural repertoires and reducing problem behaviours in two children with autism. Risley and colleagues used principles of operant conditioning to treat echolalia and tantrums in children with developmental delays (Wolf et al., 1967; Wolf et al., 1964). Wolf et al. (1964) proposed delivering behavioural interventions in naturalistic settings (e.g., home and school) and trained parents/teachers in behavioural modification. Baer subsequently published work outlining specific principles of Applied Behavior Analysis (ABA), including examples of applying these techniques to children with behavioural/developmental concerns (Baer et al., 1968). Although not the first, perhaps the most famous pioneer of the ABA approach was Ivar Lovaas, whose research focused on the outcomes, maintenance, and generalization of ABA treatments (Lovaas et al., 1973; Smith & Eikeseth, 2011). Lovaas would later examine the effects of ABA in children with autism making the bold, albeit unsubstantiated, claim that he was able to fully 'cure' 40% of his clients (Lovaas, 1987). Other treatments at the time included early special education approaches (Bartak & Rutter, 1973; Elgar, 1965; Schopler & Reichler, 1976), parent management techniques (Schopler, 1976), and structural therapies (Ward, 1968).

One of the most important contributions towards putting purely psychogenic theories of autism to rest occurred when Bernard Rimland (1928–2006), an American scientist, advocate for children with developmental disabilities, and parent with a child on the autism spectrum, published *Infantile Autism; The Syndrome and its Implications for a Neural Theory of Behaviour* (Bernard Rimland, 1964). Rimland made a strong argument for an organic, neurological cause for autism, albeit not without some errors (e.g., erroneously negating any environmental contributions). Reports of neurological abnormalities in autism, including seizures and EEG anomalies, added to the evidence supporting a neural basis for the disorder (Creak, 1963; Rutter et al., 1967). At the same time that Rimland and others were challenging the psychogenic theories, there were also efforts to invalidate an original point of confusion in the field, the idea that autism was a form of childhood schizophrenia. To do this, Kolvin (Kolvin, 1971) demonstrated that the age of onset for childhood psychosis (i.e., middle childhood to adolescence) was different than that of autism (i.e., early childhood). Additionally, Rutter and Bartak (Rutter & Bartak, 1971) proposed abandoning the incorporation of autism with schizophrenia on the basis of differences in age of onset, gender ratio, presentation, and course. In their follow-up work on children with 'infantile psychosis,' they further highlighted key differences between autism and schizophrenia via distinct cognitive profiles and neurological status, which was supported by other work distinguishing autism from

control subjects and those with other psychiatric conditions (Hermelin & O'Connor, 1970; Michael Rutter & Bartak, 1971; Schopler, 1966). Folstein and Rutter also conducted the first study of twins and autism by investigating 21 twin pairs and documenting a higher incidence rate of autism in identical versus fraternal twins, thus pointing to a strong genetic liability (Folstein & Rutter, 1977). Belief in a neurological basis for autism led to medical trials of vitamin treatments (Rimland et al., 1978; Bernard Rimland, 1973) and a range of psychotropic medications (Ritvo, 1983; Varley & Holm, 1990), although no drug treatment would be shown to effectively treat the core symptoms of autism (Siegel & Beaulieu, 2012). Other research into organic etiologies, such as the search for biomedical contributions, largely failed to yield consistent or meaningful results (Cohen, 1974; Cohen et al., 1977).

During this era, there began to be more specific consideration as to the impact of comorbidities upon symptoms of autism, including symptom severity. Creak and colleagues (Creak, 1961) suggested that intellectual impairment was commonly associated with autism and that autism was characterized by a diverse range of intellectual function. Other studies also documented links between autism and intellectual impairment (Gittleman & Birch, 1967; Lockyer & Rutter, 1969; Lotter, 1966; Rutter et al., 1967), suggesting variability in intellectual level and cognitive profiles (Lotter, 1974; Rutter et al., 1967) and leading to the recognition that it was important to control for intellectual level in autism research (Hermelin & O'Connor, 1970; Schopler, 1966). Other predictors of long-term outcomes subsequently emerged, including research suggesting that intellectual level and speech/language ability were important predictors of long-term outcomes (Lotter, 1974). Furthermore, the impact of common psychiatric comorbidities was also recognized, including attention problems, anxiety, aggression/tantrums, etc. (Creak, 1961; Lockyer & Rutter, 1969; Lotter, 1966; Rutter et al., 1967; Rutter & Lockyer, 1967), shedding light on just how complex autism is.

Towards the end of this era, the concept of autism finally began to officially expand and shake off decades-old misconceptions. In 1981, Lorna Wing and her German-speaking husband, self-described as loving and affectionate parents to their autistic child, translated Asperger's works, drawing particular attention to his writings on individuals at the higher end of the autism spectrum, as well as his inclusive approach to working with the children (Wing, 1981). In an effort to pressure the British government to increase supports for children and families living with autism, Wing had organized and conducted an impressive epidemiological study in the UK that eventually helped to expand autism into a spectrum, with individuals at both the higher and lower ends of intellectual functioning (Wing & Gould, 1979). This would provide key epidemiological data on the percentage of individuals with autism who have both intellectual impairments and pockets of giftedness and would help to differentiate children with autism from those with social-communication impairments that were attributable to other causes. By also highlighting key symptom patterns that were specific to autism, she further differentiated autism from intellectual delays and communication disorders (Wing, 1979). Wing proposed a classification system based upon the quality of social interactions and a clustering of social, language, and behavioral atypicalities, which she argued would help to

distinguish autism from other clinical groups. Wing's work would eventually form the basis for the 'triad of autism' that made its way into subsequent diagnostic systems.

Era 5: The Brain and Autism

The 1980s brought about the popularity of cognitive psychology and the search for cognitive etiologies for autism. These theories had their origins in cognitive testing and attempted to find causes for autism that were rooted in core cognitive deficits. During this time, Simon Baron-Cohen, Uta Frith, and Alan Leslie built on Premack and Woodruff's (1978) work relating to theory of mind in primates, and proposed that children with autism may be understood as lacking this basic function. In other words, they are unable to attribute mental states (e.g., beliefs, desires, emotions, intentions, etc.) to others and to apply that knowledge in social contexts (Baron-Cohen et al., 1985). Their research would demonstrate that individuals with autism perform more poorly on false belief tasks due to purported failures in theory of mind (Baron-Cohen et al., 1985; Wimmer & Perner, 1983). This generated the "Theory of Mind" hypothesis of autism wherein it was proposed that children with autism suffer from a deficit in the cognitive mechanisms required for representing mental states, leading to impairments in social understanding (Baron-Cohen et al., 1985). Other social processing accounts of autism proposed core deficits in emotion processing (Moldin et al., 2006) and social orienting (Klin et al., 1992). Francesca Happé, a graduate student of Uta Frith, built upon this by evaluating the relationship between language and theory of mind in autism, specifically around pragmatics in language use. Together, Happé and Frith proposed that language deficits in autism were a result of impaired pragmatics, particularly around understanding and using language to affect mental states (Frith, 1989; Frith & Happé, 1994). This generated new possibilities for treatments directed towards improving navigation within social situations (Bishop, 2008).

Uta Frith later synthesized two decades of work on the cognitive bases of autism(U. Frith, 1989), arguing that autism is a neurobiological disorder whereby individuals with autism have what she called "weak central coherence," or a tendency to focus on specific details and small pieces of information at the expense of the bigger picture or gestalt (Frith, 1989; Happe, 1999; Happé, 1996). This theory again stemmed from performance on cognitive tasks (Happe, 1999; Happé, 1996; Shah & Frith, 1983, 1993), where it was observed that individuals with autism seemed to process information in a detail-focused or 'local manner' (i.e., seeing wholes as a collection of individual 'parts') while neglecting the global perspective (Pring & Heavey, 1995). Subsequent research would suggest that individuals with autism may have a cognitive preference and enhanced processing of local details, rather than a deficit in central coherence (Grant & Davis, 2009; Plaisted et al., 2003).

In the 1990s, Sally Ozonoff, an American Psychologist, investigated cognition in autism with a focus on executive function (Ozonoff et al., 1995). This work gave

rise to the "Executive Dysfunction Theory" of autism or the idea that autistic symptoms are a result of core deficits in executive functions. Ozonoff demonstrated that performance of children with autism on the Wisconsin Card Sorting Test (WCST), a measure of planning and cognitive flexibility initially developed for adults, was consistently lower than that of typically developing children (Ozonoff, 1995). She then focused on identifying specific executive function profiles in children with autism (Ozonoff & Jensen, 1999). Subsequent research would support consistent deficits in aspects of executive function in autism, although no one 'autism-specific profile' would be found (Happé et al., 2006a; Hughes et al., 1994). Ozonoff further built upon Uta Frith's work around autism and pragmatic language, and demonstrated that children with autism performed more poorly on pragmatic language tasks, supporting the presence of core social and communication deficits in the disorder (Ozonoff & Miller, 1996).

Although each of the groups described above clearly hoped that their cognitive theory would prove causal in explaining autism, no single/unified theory was able to demonstrate the requisite standards of sensitivity, specificity and universality to be the fundamental deficit in autism (Rajendran & Mitchell, 2007). As such, researchers largely abandoned the search for a single cognitive cause favoring, instead, a view that perhaps a combination of cognitive theories would hold greater explanatory power. For example, proponents might contend that the different cognitive theories could account for different symptom domains in autism, that cognitive profiles could differ across the spectrum, or that distinct cognitive deficits may result in the same end point (Bishop, 1993; Charman & Swettenham, 2001).

Era 6: Awareness, Advocacy, and Advancement

In the 1990s and 2000s autism awareness increased in physicians, mental health clinicians and the general public. Although the voices of individuals with autism had been present throughout history, the rise of disability advocacy, the Internet, and greater skepticism for medical authority led autism communities to take a more active role in investigation and treatment (Silverman & Brosco, 2007). In 1986, Temple Grandin presented one of the first mainstream firsthand accounts of living with autism in her book, *Emergence-Labeled Autistic* (Grandin & Scanano, 1986). Shortly after, in 1988, the movie "*Rain Man*" was released, portraying an adult living with autism and thrusting autism into the public eye. Since this time, autism has been increasingly present in popular media with a 2008 review of 3500 health-related news pieces in the U.S. placing autism within the top five disorders most commonly reported on (Pew Research Center, 2008). In part in response to popular awareness, various autism-related organizations and advocacy groups arose to provide support, programs, and education for individuals with autism and their families (e.g., Global and Regional Asperger Syndrome Partnership (GRASP), Autistic Self Advocacy Network, etc.). In 2006, then President George Bush signed the Combating Autism Act (CAA), which provided funds for autism related research,

diagnosis, treatment and surveillance, stimulating an rise in autism-related publications (McKeever, 2013). In fact, a review of the PubMed database indicates an exponential increase in publications related to autism in the U.S. from 1998 to 2009 with a noticeable surge after 2004 (Thompson, 2013). Finally, the number of diagnosed autism cases started to grow (Matson & Kozlowski, 2011) igniting the search for a cause and cure for this 'autism epidemic'. Within this climate, a 1998 *Lancet* publication led by Andrew Wakefied (a gastroenterologist) would suggest that the measles-mumps-rubella vaccine (MMR) causes autism in children, instilling a wide-spread fear of vaccinations (Wakefield et al., 1998). Future studies would refute this finding and Wakefield's paper would be formally retracted in 2010 on the basis of fraudulent and unethical research methods. While it became well accepted in scientific circles that vaccines do not cause autism (Brown et al., 2012; Institute of Medicine, 2012), 'vaccine skepticism' in the general public has unfortunately persisted. Other environmental candidates, such as diet and the potential relationship between gluten sensitivity and autism were also investigated, although findings were not convincing (Batista et al., 2012; Molloy & Manning-Courtney, 2003).

Technological advances in the 1990s and 2000s permitted more nuanced investigations into the genome and brain in autism. With widespread acknowledgement that autism is heritable, along with advances in biomedical technology, researchers scoured the brain and genome in the search for an 'autism blueprint' (Miles et al., 2005; Szatmari et al., 1998). Research into family concordance patterns attempted to identify familial subtypes of autism, subclinical symptoms in families, and a broader autism phenotype (A. Bailey et al., 1995; Le Couteur et al., 1996; Losh et al., 2008; J. M. Silverman et al., 2002; Szatmari et al., 1998). There was investigation of psychiatric comorbidities, across simplex and multiplex families with autism, and consideration of shared genetics between such psychiatric disorders and autism (Bolton et al., 1998; Larsson et al., 2005; Micali et al., 2004).

Researchers would later conclude that finding a single cause for autism was improbable (A. Bailey et al., 1995; Anthony Bailey et al., 1998), giving rise to the idea that the autism phenotype may represent a common end-point for many different combinations of genetic and/or environmental factors (Chaste & Leboyer, 2012). In the field of molecular genetics, progress was made in identifying causal loci for autism including medical genetic conditions (D. Cohen et al., 2005), copy number variations (Sebat et al., 2007), and de novo mutations (Ronemus et al., 2014). A range of candidate susceptibility loci were identified, with over 100 studies published on different gene candidates (Berg & Geschwind, 2012; Persico & Napolioni, 2013; Michael Rutter & Thapar, 2014). Further consideration was given to the notion that the genetics of autism may function in a dimensional manner, such that different symptom domains of autism may be linked to distinct genetic anomalies (Ronald et al., 2006). It became apparent that multiple genes were likely involved, in addition to complex gene-gene and gene-environment interactions (Abrahams & Geschwind, 2008; Berg & Geschwind, 2012; Chaste & Leboyer, 2012; Happé et al., 2006b; London & Etzel, 2000). Interest rose in investigating environmental triggers such as prenatal factors (Durkin et al., 2008; Larsson et al., 2005; Zerbo et al., 2013), perinatal factors (Gardener et al., 2011; Krakowiak et al., 2012), and

teratogens/pollutants (Dufour-Rainfray et al., 2011; Kalkbrenner et al., 2014). It was recognized that parenting factors do have an impact on autism trajectories through bidirectional biological-environmental interactions (e.g., developmental cascade models(Mundy et al., 2010), although this was a far cry from the 'refrigerator mother' theories of the 1960's (J. M. Silverman et al., 2002).

Advances in brain imaging improved understanding of structural (Stanfield et al., 2008) and functional (Philip et al., 2012) brain differences in autism in key brain networks associated with social, emotional, linguistic, and sensory/behavioural processing. Significant progress was made in understanding brain differences in autism and it became clear that multiple brain regions and their connections were involved, different brain regions were affected across individuals, and patterns of brain difference varied across the lifespan within the same individual (Fein, 2011). Brain studies were (and continue to be) further challenged by the fact that many of the psychological functions studied in autism are complex (i.e., rely on multiple brain systems), that autism unfolds developmentally through interactions between the individual and the environment (Mundy et al., 2010), and that autism is a highly heterogeneous both within and across individuals.

Advocacy, better treatments, and autism funding drove the search for more sophisticated identification tools for autism research and clinical practice. Although a limited number of autism specific tools existed before this time (one example is the Childhood Autism Rating Scale; CARS (Schopler et al., 1980), a major development in the 1990s was the publication and uptake of the Autism Diagnostic Observation Schedule (ADOS) and Autism Diagnostic Interview/Revised (ADI-R), companion observational and interview tools for diagnosing autism (Lord et al., 1999; Lord et al., 1994). These standardized tools made their way into research and clinical practice, providing a more objective means for documenting the presence, severity, and progression of autism symptoms (Le Couteur et al., 2008; Lord & Corsello, 2005). Although not without limitations (Frigaux et al., 2019; Jones et al., 2015), the ADOS and ADI-R provided a systematic means of differentiating autism from other disorders and for ensuring that researchers studying autism were using a reliable and common metric for inclusion of study participants. Motivated by the prospects of very early intervention, prospective and retrospective research methodologies were utilized to investigate very early behavioural markers for autism in infancy (Dawson, 2008; Landa et al., 2007; Osterling et al., 2002; Zwaigenbaum et al., 2005).

With respect to diagnosis, the DSM-III-R (American Psychiatric Association, 1987) gave way to DSM-IV (American Psychiatric Association, 1995), the DSM-IV-Text Revision (TR; American Psychiatric Association, 2000) and then DSM-V (American Psychiatric Association, 2013). The DSM-IV/TR systems placed autism under an umbrella category of Pervasive Developmental Disorders (PDD), with subcategories of autism that differed in symptom severity and presentation (e.g., Autistic Disorder, Asperger's Disorder, PDD-NOS, Childhood Disintegrative Disorder). There were several limitations with this categorical approach, in particular the assumption that different PDDs were mutually exclusive. This led to difficulties with knowing where to draw diagnostic boundaries between the different

'autism-related' disorders and overlooked considerations regarding etiology and/or differential response to treatment (Volkmar & Reichow, 2013). The DSM-IV/TR also did not allow for diagnostic comorbidities between autism and other commonly co-occurring conditions (Gillberg et al., 2004), leading to practical problems with identifying and treating such comorbidities. With these challenges in mind, DSM-V made a conceptual shift towards a blended categorical and dimensional approach to diagnosing autism(*Diagnostic and statistical manual of mental disorders (DSM-V)*, American Psychiatric Association, 2013). Here, subcategories of PDD were dropped in favor of an inclusive, spectrum diagnosis (i.e., 'Autism Spectrum Disorder') with key specifiers and diagnosis of previously excluded comorbidities was permitted. It is important to note that while many saw DSM-V as an improvement, these changes were controversial and not without criticism(Fung & Hardan, 2014). Particularly contentious was the elimination of Asperger's Disorder and PDD-NOS, which were perceived as less stigmatizing (Kite et al., 2013) and/or a gateway to service for those with milder or atypical symptom patterns (Fung & Hardan, 2014). Research has also been significantly challenged by the changes from DSM-IV to V, with DSM-V being less inclusive such that up to 40% of those individuals with DSM-IV autism diagnoses might not meet the criteria for autism under DSM-V (Fung & Hardan, 2014; Worley & Matson, 2012). It is very likely that these changes will have an impact on generalizability when extrapolating research findings from populations studied using DSM-IV to those diagnosed under DSM-V.

Finally, treatment options for autism expanded with a focus on evidence-based approaches (Silverman & Hinshaw, 2008; Volkmar et al., 2014) and the promise of early intervention (Dawson, 2008). In 2001, the National Research Council published guidelines recommending that treatment programs for autism must address core deficits in autism, deliver instruction in structured settings, have a low student-teacher ratio, and plan for generalization (National Research Council, 2001). Intervention research focused on establishing/expanding the evidence base for existing treatments, making adaptations to existing treatments, and developing novel approaches. Growth occurred in research on comprehensive treatment programs (i.e., with a broad focus), focal treatment approaches (i.e., focused on specific symptom domains), and alternative/experimental treatments. The ABA techniques developed by Lovaas remained popular with data supporting efficacy across a range of contexts and ages (e.g., Early Intensive Behavioural Intervention (Howlin et al., 2009; Reichow, 2012). Other comprehensive programs arose including hybrid cognitive/behavioural treatments such as Pivotal Response Treatment, an approach that intervenes at the level of pivotal skills with presumed collateral effects other areas of development and function (e.g., joint attention, imitation skills, etc. (Koegel et al., 1999; Koegel et al., 2003; Pierce & Schreibman, 1995; Vismara & Lyons, 2007). Naturalistic Developmental Behavioural Intervention programs (NDBI's; e.g., Early Denver Start Model (Dawson et al., 2010) were developed as an extension of ABA approaches, to enhance generalization of intervention effects to natural settings through use of natural reinforcers. Focal interventions were developed for a range of concerns including (but not limited to): (1) communication (Bottema-Beutel et al., 2014; Lang et al., 2009; Schwartz & Nye, 2006), (2) social skills (Gray

& Garand, 1993; Reichow et al., 2012; Reichow & Volkmar, 2010; Sperry et al., 2010; Wang et al., 2011; Winner & Crooke, 2009) and (3) challenging behaviours (Mahatmya et al., 2008; Mancil, 2016; Taylor et al., 2005). Consistent with research from previous decades, ongoing research into pharmacological treatments would demonstrate that pharmaceuticals were not generally effective in treating the core symptoms of autism, although they were helpful in mitigating certain comorbidities and secondary associated symptoms (Canitano & Scandurra, 2011; Siegel & Beaulieu, 2012).

Era 7: Future Directions

Substantial progress has been made in characterizing autism since its formal introduction to the clinical and scientific and literature in the 1940s. Advocacy, understanding, and treatment options continue to grow, vastly increasing the possibilities for individuals with autism and their families. Ongoing technological advances have expanded opportunities for autism researchers studying causes, consequences, and treatments for autism. To date no one genetic feature or environmental cause has proven etiological in explaining most cases autism or has been able to account for rising rates of autism. Although the questions to be asked and answered remain limitless, several trends are worth nothing.

Continued work on the genetics of autism holds significant potential for better understanding etiologies and symptom profiles in autism and the development innovative treatment approaches. This includes work on differentiating the genetic, neural and behavioural characteristics of idiopathic and syndromic autism to identify distinct autism subtypes that may respond differentially to treatment (Casanova et al., 2020; Glinton & Elsea, 2019). Investigations of the endophenotypic similarities between autism and other neurodevelopmental/ psychiatric disorders may improve understanding of the genetic underpinnings for different symptom domains in autism and common comorbidities, aiding the development of more sophisticated diagnostic tools and treatments. Technological advancement in the field of molecular genetics further raises the possibility of genetic treatments, including ‘gene therapy’, and preventive approaches for autism at the level of the genome (Benger et al., 2018). Concerning environmental contributions to autism and gene-environment interactions, the exploration of epigenetic markers and developmental cascade models in autism remain areas that are relatively uncharted but that have substantial potential for identifying biomarkers and establishing preventive treatments (Massrali et al., 2019; Mundy et al., 2010; Wiśniowiecka-Kowalnik & Nowakowska, 2019). More nuanced characterization of symptoms, cognitive profiles, and behavioural subtypes of autism, how these change across the lifespan, and how these may differ across different populations (e.g. males versus females, different ethnicities, etc.) are also important areas for future research. Giving the developmental nature of autism, continuing to extend research down into early infancy to identify very early

biomarkers of autism with the goal of changing trajectories through innovative early interventions is also critical.

Finally, a crucial consideration for autism research relates to the priorities of those with autism and their families. A person-centered research approach that invites key stakeholders within the autism community to generate and guide research questions is increasingly being adopted. Such research, which actively involves individuals with autism in research from its inception, will ensure that researchers are prioritizing issues that are of importance to the autism community. This type of collaborative research partnership is crucial in ensuring that the voices of individuals with autism remain at the forefront of scientific enquiry within the context of the prevailing scientific cultural and political views of our time.

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