

## AUTISM

---

# A Historical Perspective on Autism

**Adam Feinstein**

Autism Cymru and Looking Up, United Kingdom

July 2012

### Introduction

The history of autism is plagued by misconceptions and distortions. This is due to the condition's heterogeneity and to the fact that a diagnosis of autism spectrum disorders (ASD) is based on descriptions and observations of behaviour. Although there is much evidence that autism is a neurodevelopmental disorder with a very strong genetic component, there is not yet a valid biomarker or biological test for autism.<sup>1</sup>

### Research Context

Many of the symptoms first described by Leo Kanner in his seminal 1943 article providing clinical descriptions of autism<sup>2</sup> still apply to the way the term autism spectrum disorder is used now, although Kanner himself never accepted the broadening of what he saw as a narrowly-defined syndrome characterised by: a profound lack of affective contact with other people; an anxiously obsessive desire for the preservation of sameness in routines and environment; a fascination with objects; mutism or a kind of language that does not seem intended for inter-personal communication, and onset of the condition from birth or before 30 months.

In fact, Hans Asperger was working in the field in Vienna<sup>3</sup> long before Kanner was in Baltimore.<sup>4</sup> Asperger said the condition was never recognised in infancy and usually not before the third year of life or later. A full command of grammar was sooner or later acquired, but there might be difficulty in using pronouns correctly. The content of speech was abnormal, tending to be pedantic and often consisting of lengthy disquisitions on favourite subjects. Perhaps the most obvious characteristic was impairment of two-way social interaction, due primarily to an inability to understand and use the unwritten, unstated rules governing social behaviour. Asperger, like Kanner, also reported certain skills, as well as impairments, such as excellent rote memories and intense interest in one or two subjects. Unlike Kanner, Asperger recognised wide variability in his syndrome.

For many years after Kanner's 1943 paper,<sup>2</sup> there was confusion between the terms "autism" and "childhood schizophrenia." It is generally considered that a distinction was clearly laid down in the 1970s.<sup>5</sup>

Thirty years ago, autism was considered to be a rare childhood disorder most often associated with severe intellectual disabilities, lack of social awareness and absence of meaningful expressive language.<sup>6</sup> Today, the spectrum of autistic disorders (or ASD) is recognised as a set of common developmental disorders. The causes of most cases of autism remain unknown. Once thought to be the product "*refrigerator mothers*", this notion has generally been discredited – although sadly not in some parts of France, Italy and a number of Latin American nations. There is common agreement today that ASD is a neurodevelopmental disorder that has a genetic basis (perhaps in interaction with the environment).

## **Recent Research Results**

In the 1980s, less severe forms of autism were recognized as separate diagnostic categories within a broader class of what are now known as ASD denominated as "pervasive developmental disorders." Despite its earlier description, Asperger's disorder appeared in official nosographies only in the 1990s. The difference between high-functioning autism (affects individuals who exhibit autistic behaviours but are functional in a social context) and Asperger's syndrome remains a subject of debate. The validity of Asperger's disorder as a distinct syndrome from autism is unclear, partly because of the paucity of differentiating neurobiological evidence.

In the currently-used fourth edition of the Diagnostic and Statistical Manual (DSM-IV) published by the American Psychiatric Association,<sup>7</sup> Autistic disorder is defined by onset prior to three years of

age and the presence of deficits or unusual behaviours within three domains: reciprocal social interaction, communication, and restricted, repetitive interests and behaviours. The DSM's broader category of PDD-NOS (pervasive developmental disorder not otherwise specified) encompasses a wider range of conditions sometimes referred to loosely as "atypical autism." In recent years, the definitions of autism have been further broadened, with an increasing reliance on a dimensionalisation of the autism phenotype.

For the new fifth edition of the DSM, the revised criteria include only two symptom domains (social-communication and fixated, repetitive interests), eliminate subtypes of ASD, and describe individual differences in severity of symptoms in the two domains, relative to developmental levels and chronological age. Thus, an individual with an ASD diagnosis would be described in terms of dimensions of social-communication symptoms and severity of fixated or restricted behaviours or interests. This diagnosis could be associated with other known genetic or medical conditions (e.g., ASD and Rett's syndrome or ASD and Fragile X), language disorders, or other psychiatric conditions (e.g., ASD with ADHD, ASD with intellectual disability).

## Research Gaps

Although the concept of ASD has become more familiar, important questions remain about its aetiology, the most accurate and efficient procedures for diagnosis, the apparent increase in prevalence of autism spectrum disorders, and the most effective treatments.

Despite ongoing attempts to "unpack" autism into separable components,<sup>8</sup> certain commonalities strongly define this group: significant and early-arising difficulties in basic aspects of social communication and restricted, repetitive behaviours or interests.

Some distortions and misconceptions about autism remain strong. For example, in terms of interventions, *spurious* pseudo-scientific claims of effective treatments have included *hyperbaric chambers*, chelation (the removal of heavy metals) and wrapping in cold wet sheets ("le packing" in France). Fortunately, there is also a tremendous amount of very solid scientific research now addressing questions about aetiology, epidemiology, diagnosis and treatment.

## Conclusions

The research to date clearly indicates that ASD affects a diverse group of children and adults whose needs are varied across the lifespan, both in terms of assessment and intervention. Given

the current variability in rates across states and the disparities in diagnosis across ethnic groups and parental education levels, it seems most likely that if disparities decrease, the numbers of children with ASD will rise even more.<sup>9,10</sup> Careful assessment is needed to determine the most appropriate services for different children, as well as for the same child at different points during the life course.

## Implications for Parents, Services and Policy

Because the prognosis and treatment of individuals with ASD are strongly linked to cognitive and language levels, assessments must include standard developmental measures in addition to autism-specific measures. In terms of public policy, it is important to recognise that, as ASD has become a more heterogeneous category, one-size-fits-all approaches to diagnosis and assessment are not appropriate.<sup>11</sup> The variability within rates of subtypes of ASD has significant policy implications. Because of the heterogeneity of the population, it is a difficult number for which to plan. A single program may not be appropriate for the majority of the children in a classroom because of the range in their ages and developmental levels. Even between birth and the age of three, the range of skills and needs of young children with ASD are variable.

## References

1. Abrahams, B., & Geschwind, D. (2008). Advances in autism genetics: On the threshold of a new neurobiology. *Nature Reviews. Genetics*, 9(5), 341-355.
2. Kanner, L (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217-250.
3. Asperger, H (1938). Das psychisch abnorme kind. *WienerKlinischen Wochenschrift*, 51, 1314-1317.
4. Feinstein, A. (2010). *A History of Autism: Conversations with the Pioneers* London:Wiley-Blackwell.
5. Kolvin I, Ounsted C, Humphrey M & McNay A (1971). Studies in the childhood psychoses, *British Journal of Psychiatry*, 118: 381-419.
6. Lotter, V. (1966). Epidemiology of autistic conditions in young children. *Social Psychiatry and Psychiatric Epidemiology*, 1(3), 124-135.
7. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed (text rev). (2000) Washington, DC: American Psychiatric Association.
8. Happé, F., & Ronald, A. (2008). The 'fractionable autism triad': A review of evidence from behavioural, genetic, cognitive and neural research. *Neuropsychology Review*, 18(4), 287-304.
9. Autism and Developmental Disabilities Monitoring Network Surveillance Year 2002 Principal Investigators; Centers for Disease Control and Prevention. Prevalence of autism spectrum disorders—autism and developmental disabilities monitoring network. *MMWR Surveill Summ.*, 56(1):12-28.
10. Fombonne, E. (2005). Epidemiological studies of pervasive developmental disorders. In F. R. Volkmar, A. Klin, R. Paul and D. J. Cohen (Eds.), *Handbook of autism and pervasive developmental disorders*, Vol. 1 (3rd ed.) (pp.42-69). Hoboken, NJ: Wiley.

11. Gotham, K., Bishop, S. L., & Lord, C. (in press). Diagnosis of autism spectrum disorders. In Amaral, Geshwind, & Dawson (Eds.), *Autism Spectrum Disorders*. New York: Oxford University Press.