Vignette


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Abstract

Autism, in a wider perspective Autism Spectrum Disorder (ASD), is perhaps the most prolifically researched of all child psychiatric disorders. The greatest contributions to our understanding about the disease have come from individual clinician researchers like Leo Kanner and Hans Asperger. The concept and definition of the disorder have changed greatly over the years, even socio-political shifts as well as research findings have radically altered our understanding of the syndrome as well as the care and treatment offered to people with autism. This paper provides an overview on the discovery of Autism Spectrum Disorders through philately.

Keywords: Autism, Autism Spectrum Disorder, Leo Kanner, Hans Asperger, history, philately

INTRODUCTION

Autism is considered as one of the five pervasive developmental disorders, which are characterized by widespread abnormalities of social interactions and communication, and severely restricted interests and highly repetitive behavior. Genomic research is beginning to discover that people with autism spectrum disorders probably share genetic traits with individuals with attention-deficit hyperactivity disorder, bipolar disorder, schizophrenia, or clinical depression. Autism is known as a complex developmental disability. Experts believe that autism presents itself during the first three years of a person's life. The condition
is the result of a neurological disorder that has an effect on normal brain function, affecting development of the person's communication and social interaction skills.\(^1\)

This syndrome was first described as the earliest form of schizophrenic psychosis. It is now considered to be a biologically based pervasive neurodevelopmental disorder affecting from the first days of life, social communication and adjustment to the environment.\(^6\) Advances in the understanding of its clinical neurofunctional and genetic aspects have progressively modified conceptions and practices for diagnosis, exploration and therapeutics. This live-long complex syndrome presents major challenges.\(^1\)

Over the last few years, the perception of autistic disorders, the theoretical references studied, diagnostic, assessment, care, educational and pedagogical practices have been transformed.\(^17\)\(^5\)\(^7\)\(^1\) (Figure 1)

The Latin word autismus was first used by the Swiss psychiatrist Eugen Bleuler in 1910 as he was defining symptoms of schizophrenia.\(^6\) Autismus is coming from the Greek word autos meaning self and it is used to mean morbid self-admiration, referring to the conditions in which a person is removed from social interaction; an isolated self.\(^3\)

We see two important doctors, in other words two milestones in the diagnosis and recognition of autism; Leo Kanner and Hans Asperger.\(^15\)

Leo Kanner (1896 – 1981) was an Austrian psychiatrist and physician known for his work related to autism. He is the founder of child and adolescent psychiatry in the U.S. and worldwide.\(^15\) Kanner was born in a small village north of Brody (Galicia) in Austria to an orthodox Jewish family. He started studying at the University of Berlin in 1913, his studies interrupted by service to the Austrian Army in World War I, finally receiving his M.D. in 1921. He immigrated to the United States in 1924 to take a position as an Assistant Physician at the State Hospital in Yankton County, South Dakota. In 1930 he developed the first child psychiatry service in a pediatric hospital at Johns Hopkins Hospital, Baltimore.\(^15\) (Figure 2)

Before Dr. Kanner started to write his paper titled "Autistic Disturbances of Affective Contact"; one of his child patients, Donald T., was an important case for his clinical observations. Donald T. was not like other 5-year-old boys. The 33-page letter which Donald's father wrote to Dr. Kanner described the boy by these details: “happiest when he was alone... drawing into a shell and living within himself... oblivious to everything around him.” Donald had a mania for spinning toys, liked to shake his head from side to side and spin himself around in circles, and he had temper tantrums when his routine was disrupted.\(^6\)

When Kanner met Donald, his suspicions were confirmed. In addition to the symptoms the letter described, Kanner figured out Donald's explosive, seemingly irrelevant use of words. Donald referred to himself in the third person, repeated words and phrases spoken to him, and communicated his own desires by attributing them to others.\(^3\)

Kanner described Donald and ten other children in his paper entitled Autistic Disturbances of Affective Contact in 1943 and this paper forms a very important basis of the modern study of autism. In this initial description of ‘infantile autism, which went on to become a classic in the field of clinical psychiatry, Kanner described a distinct syndrome instead of previous depictions of such children as feeble-minded, retarded, moronic, idiotic or schizoid.\(^15\)

Leo Kanner was the Editor for Journal of Autism and Developmental Disorders, then called Journal of Autism and Childhood Schizophrenia, from 1971 till 1974.\(^8\)

Kanner borrowed the term ‘autism' from Eugene Bleuler, who had used it to
describe the inward, self-absorbed aspects of schizophrenia in adults. But Kanner did not consider infantile autism an early form or prodrome of schizophrenia. The clinical signs were not identical and, unlike schizophrenia, Kanner's patients seemed to have autism from birth.\(^{15}\)

Dr. Kanner's humanism is evident in his lifelong fight against the abuse of children with autism and intellectual disability, and his enduring concern for their families. He also made extraordinary efforts to help physicians and scientists escape from Nazi-controlled territories.\(^{3}\)

In 1981, Kanner died of heart failure at his home in Sykesville, Maryland, when he was 86.\(^{15}\) (Figure 3)

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**Figure 1:** Stamp published in India in 2003; emphasising 'our world of special children'

**Figure 2:** Stamp published in Uruguay in 2013
Hans Asperger (1906 – 1980), in 1944, one year after Kanner's paper, described children that he also called 'autistic', but who seemed to have high non-verbal intelligence quotients and who used a large vocabulary appropriately.\(^{(7)}\)

Hans Asperger was born on a farm outside Vienna. He was the elder of two sons. He himself had difficulties in finding friends and was considered a lonely, remote child. As a child, Asperger appeared to have exhibited features of the condition subsequently named after him. He was talented in language; he was particularly interested in the Austrian poet Franz Grillparzer, whose poetry he would frequently quote to his uninterested classmates. He also liked to quote himself and often referred to himself from a third-person perspective.\(^{(7)}\)

Asperger studied medicine at the University of Vienna and practiced at the University Children's Hospital in Vienna. He graduated as a doctor of medicine in 1931 and became director of the special education section at the university children's clinic in Vienna in 1932.\(^{(5)}\) (Figure 4)

Asperger published a definition of autistic psychopathy in 1944. He identified in four boys a pattern of behavior and abilities that included "a lack of empathy, little ability to form friendships, one-sided conversations, intense absorption in a special interest, and clumsy movements".\(^{(15)}\) Asperger called children with this syndrome "little professors" because of their ability to talk about their favorite subject in great detail. Asperger noticed that many of the children he identified as being autistic used their special talents in adulthood and had successful careers. One of them became a professor of astronomy and solved an error in Newton's work he had originally noticed as a student. Another one of Asperger's patients was the Austrian writer and Nobel Prize in Literature laureate, Elfriede Jelinek.\(^{(12)}\)

Asperger died before his identification of this pattern of behaviour became widely recognised. This was in part due to his work being exclusively in German and as such it was little-translated. There was a resurgence of interest in his work beginning in the 1980s, and due to his earlier work which is regarded by some as
under the fold of autism spectrum disorders, Asperger syndrome (AS) was named after him.\textsuperscript{(7)} (Figure 5)

In the early 1990s Asperger's work gained some notice due to some research on the subject and translations, leading to the inclusion of the eponymous condition in the DSM-IV (Diagnostic and Statistical Manual of the American Psychiatric Association) in 1994, some half a century after Asperger's original research. Despite this brief resurgence of interest in his work in the 1990s, AS remains a controversial and contentious diagnosis due to its unclear relationship to the autism spectrum.\textsuperscript{(12)} Asperger's birthday, February 18, was declared International Asperger's Day.\textsuperscript{(8)} (Figure 6)

**Etiology and Genetic Background**

Starting in the late 1960's, autism was established as a separate syndrome by demonstrating that it is lifelong, distinguishing it from intellectual disability and schizophrenia and from other developmental disorders, and demonstrating the benefits of involving parents in active programs of therapy. As late as the mid-1970s there was little evidence of a genetic role in autism; now it is thought to be one of the most heritable of all psychiatric conditions.\textsuperscript{(8)} (Figure 7)

Autism has a strong genetic basis, although the genetics of autism are complex and it is unclear whether ASD is explained more by rare mutations with major effects, or by rare multigene interactions of common genetic variants. Complexity arises due to interactions among multiple genes, the environment, and epigenetic factors which do not change DNA but are heritable and influence gene expression.\textsuperscript{(2)} Deletion, duplication and inversion are all chromosome abnormalities that have been implicated in autism. However, most of the mutations that increase autism risk have not been identified. Typically, autism cannot be traced to a Mendelian single-gene mutation or to a single chromosome abnormality, and none of the genetic syndromes associated with ASDs have been shown to selectively cause ASD.\textsuperscript{(14)} Numerous candidate genes have been located, with only small effects attributable to any particular gene. The large number of autistic individuals with unaffected family members may result from copy number variations—spontaneous deletions or duplications in genetic material during meiosis. (Figure 8)

![Figure 4: Stamp published by United Nations in Vienna in 2012](image-url)
Figure 5: Stamp issued in Brasil in 2014

Figure 6: A special cancellation for Autism Day in Italy on 2.4. 2014

Figure 7: A special cancellation for Autism Day in Italy on 2.4. 2013
Although evidence for other environmental causes is anecdotal and has not been confirmed by reliable studies, extensive searches are underway. Environmental factors that have been claimed to contribute to or exacerbate autism, or may be important in future research, include certain foods, infectious disease, heavy metals, solvents, diesel exhaust, phthalates and phenols used in plastic products, pesticides, brominated flame retardants, alcohol, smoking, illicit drugs, vaccines, and prenatal stress.\textsuperscript{(16)} Blood serotonin (5-hydroxytryptamine or 5-HT) levels of autistic patients were evaluated as a biomarker in various studies since 1961.\textsuperscript{(13,18)} The significantly higher mean 5-HT levels were described in approximately 30% of autistic patients compared to control groups. Elevated 5-HT levels are seen as autism-specific because they were not elevated in cognitively impaired individuals, and are equally distributed in different Pervasive Developmental Disorder subtypes\textsuperscript{(9)}. It has been showed that autistic behaviors result from abnormal neurodevelopment.\textsuperscript{(4)} Additionally, reduced binding of antidepressant-sensitive 5-HT transporter and 5-HT receptors were also shown the abnormalities in brain 5-HT systems.\textsuperscript{(10,11)} The role of abnormal neurodevelopment on autistic behaviors seem to look promising for today, defining possible causes of autism, earlier diagnosis, estimating spontaneous developmental trajectories, and predicting treatment response.\textsuperscript{(5)}

**CONCLUSION**

We know that autistic people have their place in the organism of the social community. They fulfill their role well, perhaps better than anyone else could, and we are talking of people who as children had the greatest difficulties and caused untold worries to their care-givers. Although the rise of parent organizations and the destigmatization of childhood ASD have deeply affected how we view ASD, parents continue to feel social stigma in situations where their autistic children's behaviors are perceived negatively by others. A person with autism feels love, happiness, sadness and pain just like everyone else. Just because some of them may not express their feelings in the same way others do, does not mean at all that...
they do not have feelings. The main goals when treating children with autism are to lessen associated deficits and family distress, and to increase quality of life and functional independence. No single treatment is best and treatment is typically tailored to the child's needs. Families and the educational system are the main resources for treatment. Especially adults with ASD should be provided with appropriate information, and counseling to cope with changes in their own level of ability or health. Every year April 2 is celebrated as World Autism Day.

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