



Review

Autism, 75 years of history: From psychoanalysis to neurobiology

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Abstract: In this article we evaluated the lengthy seventy-five year history of Autism Spectrum Disorder beginning with the first diagnosis in 1943 and the first etiological orientations (psychodynamic factors) until today, in which the approach to behavioral problems has increasingly become neurobiological in nature. Abnormal relationships, verbal communication impairments and stereotyped behaviors, are no longer considered the cause of the problem, but rather the manifestation of this problem. Additionally, these factors must not be considered a “problem” and should be viewed as an “adaptation”. In other words, a way through which individuals with different anatomical and functional nervous system give “meanings” to their own bodies and the external world. Subjects with autism have a different connectome. Their neuronal network, under life experiences (learning), has undergone an abnormal selection. In modern words, talking about connectome, could mean direct our attention and studies on the body-brain relationship and chronic inflammation.

Keywords: autism spectrum disorder; neuroscience; neurological organization; sensory-motor dysfunction; connectome; neurodevelopment; neurobiology

Abbreviations: DSM: Diagnostic and Statistical Manual of mental disorders; PET: Positron Emission Tomography; CAT: Computed Tomography; TEACCH: Treatment and Education of Autistic and Communication related handicapped Children; ABA: Applied Behavior Analysis

1. The first diagnosis of autism, from the psychodynamic approach to the behavioral approach

In 1943 Kanner described clinical cases (eleven to be exact) of children/teenagers to whom he gave, for the first time in history, the diagnosis of **autism**.

The term autism (from the Greek *autos*, same) was introduced in medicine a few decades earlier in 1906 by Bleuler, to indicate some negative symptoms (closure) of schizophrenia.

From the first diagnosis, until the beginning of the 1980's, the genesis of the symptomatologic triad that characterizes the clinical presentation of autism in children (deficit in verbal communication, social interactions, stereotyped behaviors) was interpreted, by the scholars of that age, like a reaction of closure, more or less conscious, of the child towards the lack of affection of the mother (refrigerator mothers).

The treatment strategies suggested were: A psychoanalytical treatment for the mother and a removal of the child from the house with the admission to a psycho-pedagogic institute.

As mentioned, during the 80's (1980 and after that 1987), with the publication of DSMIII and DSM-IIIr, scholars, by substituting the term autism with developmental disorder, started to take, irreversibly, the distance from dynamic hypothesis to finally orient themselves toward "organic causes of autism".

Imaging studies (PET, CAT), the elevated presence of epilepsy in autistic subjects, and some neurological signs constantly present (dyspraxia, muscle tone unbalances, deficit of the motor coordination with awkwardness especially when trying to kick, throw or catch a ball, etc.), suggested to leave the old etiological hypothesis (psychodynamics) to orient toward a more "biological" approach of the problem.

However, although some researchers (Rimland (1971); Delacato (1972)) strongly sustained that autism was consequential to a biological cerebral damage, we had to wait twenty more years to definitely abandon the belief that autism could have a psychodynamic genesis (with all the inevitable consequences for the families of these patients).

At this point it's important to remember that during the 1990's the approach to the behavioral problems was predominantly behaviorist [1].

This meant that, after a short initial period (less than a decade), where the autistic child was prescribed a neurorehabilitation therapy (physiotherapy, psychomotricity, speech therapy) together with a series of therapeutic recommendations, influenced by the psychodynamic prejudice or, the more severe, "mental retardation" associated with autism (music therapy, horse therapy, occupational group therapies, water therapy, containment therapy through hugging and holding, didactical therapies or Portage), behavioral therapies (from TEACCH in 1996 to ABA these days) were the most prescribed.

2. Neuroscience shows the new way

Meanwhile, in the neuroscience field, from the 90's to today, cognitive psychology underwent a slow and progressive decline in favor of evolutionary biology as the referring science for the understanding of some processes: Perception, memory, learning, behavior, awareness, all atypical in the autism spectrum disorder [2].

The studies of many neuroscientists (e.g., Edelman, Eccles, Kandel, Changeux, Crick, Levi Montalcini, Zeki, Damasio, Tonioni, Calissano, etc.) led us to better understand not only how the organism learns behavior, but especially what happens in the organism's brain during the learning process [3].

Today, we better understand that behavior is influenced by both genetic and environmental factors [4]. These include evolutionary processes, development, education, social contacts, as well as his technological history.

When examining autism, the first signs and symptoms are often present about 14 months necessitating further exploration of the neuro-development aspect of the disorder.

3. What do modern neurosciences indicate with the term neurodevelopment?

If we were to observe the brain from the inside, we would see: Nerve cells, glia, synapsis, neurotransmitters, chemical modulators, electrical waves, as well as billions of active neurons that communicate between each other. Our thoughts, emotions, feelings, and our knowledge regarding the external and internal world depend uniquely from which nervous cells communicate and with who they communicate in that exact moment [5].

The term “development” explains exactly how this network of neuronal communication is “selected”, from birth throughout our entire life.

In fact, all our abilities and, therefore, our behaviors, will never depend on special characteristics of some components of the network (nervous cells or neurotransmitters), but rather on the interaction between these cells. It’s from their “organization” in forming the network that the specific abilities will “arise”.

The word development is preferred over neurodevelopment since the body-brain relationship will influence behavioral selection. This is because the primary purpose of the nervous system, including the human brain, remains to maintain the organism’s homeostasis [6].

This development of the nervous system starts in very premature age. Sure enough, during the third week of gestation there is already the progenitor nervous cell. From this, through a very fast process of cellular **multiplication**, it will form, in the human, hundreds of billions of nervous cells by the fifth month of gestation [7].

At the same time, all these nervous cells, through an additional process, called **migration**, will position themselves in specific cerebral areas, determining the typical conformation of our nervous system.

Cellular multiplication and migration represent the first two processes of neurological organization.

4. Genetics passes the baton to epigenetics

These processes are mostly regulated by chemical substances. This means that genetics determines the processes. At the same time, the mother’s health status, that is the environment in which the fetus develops, will interfere and will regulate the processes (epigenetics).

Because evolution determined that the human newborn comes to life excessively immature (biped position restricted pelvis’ size precluded labor after the fortieth week), the process of neurodevelopment and of neurological organization must continue and complete in an extrauterine environment.

The biological trait of not being able to organize our own nervous system before the seven /eight months of age, represents the primary condition for which genetic information is not sufficient for determining and regulating the entire process of “neurological organization” (development of neural

network). This is the biological limit (coming to the world extremely incomplete) that made us “free” from the gene’s determinism [8].

We can easily understand this concept through the study of Hubel and Wiesel’s experiments (they received the Nobel prize for these studies) on the development of cats’ visual system during the first ten weeks of extrauterine development. With this study, the two researchers demonstrated that, for a normal cerebral electric activity and, therefore, for the development of a “normal” neuronal map, the sensory experience is necessary.

Cellular multiplication and migration start the bases to build our nervous system but, for the formation of neuronal circuits, the sensory-motor experience is necessary [9].

5. Learning means to select the network

Successively, further studies demonstrated that the neuronal circuit is not built (creationist hypothesis), but rather, thanks to the sensory experience (pruning of synapses and circuits), it is **selected**.

Thanks to the biological property of nervous cells of being able to modify its own cytoskeleton for the entire life (neuroplasticity) and thought the biological properties of the nervous system (adaptation, sensibilization, de-sensibilization, classic conditioning, long term strengthening), the sensory-motor experience will select the neuronal circuit of the organism, in other words, the neuronal maps (experience selection) [10].

For whoever is interested in dealing with “behavior”, typical or atypical, understanding how neuronal maps form in our brain (neurological organization) is essential.

Indeed, in the last two decades, neurosciences have definitely clarified that the images, that “inhabit” our heads, are nothing more than the phenomenal aspect of our neuronal maps.

Whatever we do or experience (psychostatus) represents the corresponding of extensively integrated neuronal maps activated by our brain (neurostatus), selected during the development due to experience [11]. Therefore, we cannot express any psychostatus (capacity or learning) without having the corresponding neurostatus (neuronal map). These new findings help us understand the genesis of “atypical behaviors” that characterize the clinical pictures of autism spectrum disorder.

As a matter of fact, all the behavioral anomalies of the autistic child, like difficulties (verbal, concentration, motor coordination, etc.) and hyper-capacities (remember details, or remembering a specific itinerary, etc.), must be considered, exclusively, as consequences of an abnormal selection of neuronal maps during the neurodevelopment.

6. Why abnormal neuronal maps are generated during the development of an autistic child?

This important question needs to be divided into two discussions: Which is the *noxa patogena* of these anomalous neuronal maps (etiology) and how this determines the specific anomalies able to give these neurological signs and symptoms and not others (for example motor tics or obsessive-compulsive thoughts or attention deficit or more), in other words, what is the pathogenesis?

Since 1992 I have dealt with the pathogenetic aspect of autism, therefore, I prefer to omit the issue regarding the causes of autism and express a scientific consideration regarding its pathogenesis [12]. Clinical experience in neuroscience has demonstrated that the pathologies of the nervous system often have biological characteristic: Different *noxe patogene* (for example ischemia, hemorrhage, trauma, demyelination) and yet can present the same clinical symptoms, as the same “cause” often

presents with different neurological signs and symptoms when it affects different cerebral areas. In other words, observations in the neurologic clinic clinical signs and symptoms will depend more on the neuronal area involved than the cause of the disease.

A good anamnesis and a correct clinical observation are essential elements for the diagnosis. Usually, between the first and second year of life, members of the family first begin to observe, with concern, that the child speech development lacks behind peers. In other cases, always during the same time (essential factor to give the diagnosis of autism), the child manifests regression in previously acquired verbal abilities (repetitive sounds), non-responsive to verbal requests (when called child doesn't respond), or starts to manifest a "fleeing look".

But what happens in a human brain, between the first and second year of life, that allows the toddler to acquire the language and the relationship typical of that age (look sharing, appropriate mimics)? As evidence suggests, cellular quantity, migration, and neurological organization at that age (twelfth/twenty-fourth month of life) is already established.

Instead, what is being "selected" is the communication (synaptic connections) between neuronal modules of the cerebral cortex, as well as the communication of these cerebral areas and the subcortical structures [13].

Electroencephalographic recording on a human toddler between his first and second year of life we would demonstrate that, the alpha activity, progressively, would begin substituting the slower activities (δ and θ) which are present abundantly at birth. The meaning of this alteration of the electrical brain activity with age, is properly attributed to the increment of the neuronal integration in the sensory-motor and secondary associative cortexes (temporal-parietal-occipital circuits).

In terms of the anatomy and physiology of the nervous system it means that, both through retroactive axons (which go back to the thalamic neurons from sensory cortexes) and axons that connect submodules of visual, tactile and auditory areas, the cerebral areas (first between themselves and subsequently between cerebral lobes) establish precise connections to allow the organism to assign a "correct meaning" to the surrounding world [14].

The role of the nervous system is to: 1) coordinate the activities of all the cells that compose the organism (homeostasis); 2) allow the organism to attribute meanings to what surrounds him and, on how the latter, modifies the homeostasis.

This second role, at the beginning of our life, is very chaotic (we come to life uncomplete and for this we need assistance), because the areas of the brain are poorly integrated between themselves.

With the experience-based selection, stable connections will establish (learning) first between cortical and subcortical areas, then between submodules of the same sensory areas (it will guarantee sensorial meanings), subsequently between different sensory areas (it will guarantee perception meanings), and finally between posterior areas and frontal lobe (it will guarantee meanings that will go further than the here and now), in addition to connections between the two hemispheres [15].

When the thalamic-cortical and sensory sub-modules circuits, in addition to the ones between different sensory lobes, are not selected in a "typical" way, inevitably, between the first and second year of life, the child will begin to manifest atypical behaviors, both in the relationships and the communication, which will induce to a diagnosis of **autism**.

The clinical scenario will be characterized for the anomalous way to attribute meanings to the external world (psychostatus), which will reflect the atypical neuronal maps (neurostatus) selected with development.

7. Symptoms have an adaptive value

Inevitably, the child with atypical neurodevelopment to see better will have to look in a different way than the other children. For example, to hear better he will have to move away from loud noise. All these clinical manifestations, will all the others that the child will present, are not “problematic behaviors” but adaptations, extremely intelligent, inevitable to survive.

Indeed, starting from the beginning of the 70’s of the last decade, Delacato (*The Ultimate Stranger: The Autistic Child*, 1973) explained how, an anomalous sensory selection, could represent the central element of the pathogenesis of autism. The same was written a couple of years later by Temple Gradin in his autobiography (*The Autistic Brain*).

8. Conclusions

Declaring that autism is consequential to a disorder of the connections between neuronal areas (connectomes) contributes, in part, to shift the attention from the neuronal cell (gray matter) to the myelin (white matter), and in particular to microglia. For this reason, in my opinion, in the next years we will witness a true revolution: A pathology considered for decades as “dynamic”, not only it has been recently considered neurologic, but, shortly, it could be treated as systemic and, therefore, of pediatric interest.

The primary involvement of the immune system (microglia) in the process of pruning of the cerebral circuits could revolutionize our approach to autism. For certain, to improve care of a child with autism spectrum disorder it is essential to first consider the state of health of the child’s body. This statement finds support in at least a dual consideration: 1) The experience-based selection, essential for the synaptic pruning, doesn’t rely exclusively on what the brain does, but, especially on what the organism does; 2) Before receiving information from the external world to the organism, the nervous system receives information from the body in which it is located. This body-brain communication will affect both the evolutionary ancient aspect (homeostatic or visceral or interoceptive) and the less ancient (musculoskeletal structure or proprioception) [16]. This communication, different from sensorial communications (external world), will be extremely intimate and continuous, and lays the bases for the development of the mind.

Conflict of interest

The authors declare no conflict of interest.

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