# Consciousness and Attention in Autism Spectrum Disorders

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**Abstract**

Frequency of diagnoses of diseases, which belong to Autism Spectrum of Disorders (ASD) incerases with each year. The question is: is there a unifying mechanism at molecular, cellular or systems level? In the paper we present our point of view to problem of attention in autistic people. Simulations using neural network model with attractor analysis help assess system dynamics. Based on model of visual recognition, Fuzzy Symbolic Dynamics (FSD) visualization of attractors in semantic layer is provided. Simulations of structures like brain or other characterized by such order of complexity requires enormous computational power. The project announced herein will be implemented on cluster and grid-based architectures.

## 1 Introduction

In 1943 Leo Kanner defined autism as "extreme aloneness from the beginning of life and anxiously obsessive desire for the preservation of sameness". He paid particular attention to assuming that autistic children "have come into the world with innate inability to form the usual, biologically provided affective contact with people, just as other children come into the world with innate physical or intellectual handicaps". Autism is perceived as a behavioral (developmental) syndrome. Role of its initial cause can play psychodynamic theories, bad parents, refrigerator mothers etc. Common scientific deficit in the area is lack of the theory of mind. Because of it autism is perceived as multiple disease entities, multiple etiologies, including metabolic and immune system deregulation. However, as in many areas of neuroscience, we are "data rich and theory poor" [1].

In the paper we present our point of view to problem of consciousness and attention in autistic people and simulations using neural network model with attractor analysis to assess system dynamics.

# 2 Characteristics of Autism

## 2.1 Autism Spectrum of Disorders

Frequency of diagnoses of diseases, which belong to Autism Spectrum of Disorders (ASD) incerases with each year. ASD incidence is estimated to 6 per 1.000, but this incidence varies depend on form of autism, e.g.:

- incidence of a "typical" autism is estimated to 1-2 per 1,000;
- regressive autism (>2nd year) constitutes over 25% cases;
- incidence of Asperger syndrome, characterized by normal IQ but low social cognition, is estimated to about 0.3 per 1,000;
- pervasive developmental disorder not otherwise specified (PDD-NOS), when more specific criteria are not met – constitutes vast majority;
- Rett syndrome – rare.

Variability of ASD incidence depends also on sex – four times more boys suffer from autism than girls. Despite numbers metioned above – opinions differ in the area, if there is an autism epidemic.

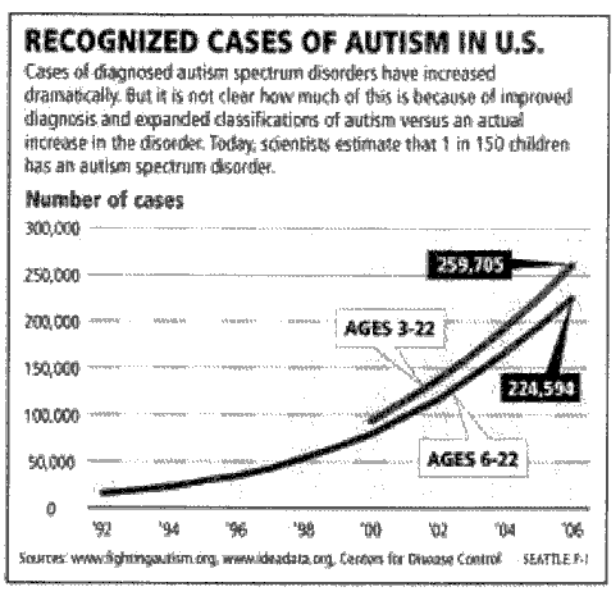


Fig. 1: Recognized cases of autism in U.S.

## 2.2 Patophysiology

Important question is whether there is a unifying mechanism at molecular, cellular or system level responsible for ASD? Autism may be a large set of disorders

with diverse mechanisms, like intellectual disability. Sixty gene mutations have been implicated [2, 3]. From the other point of view autism may result from:

- a few disorders caused by mutations converging on a few common molecular pathways, alteration of brain development soon after conception, influenced by environmental factors;
- abnormal formation of synapses and dendritic spines, poorly regulated synthesis of synaptic protein;
- an excess of neurons that causes local overconnectivity;
- unbalanced excitatory-inhibitory networks.

There are many conditions comorbid to autism spectrum disorders:

- increased 10-20% brain size in childhood, cerebral gray and white matter and cerebellum, disappears with age,
- mental retardation in 60-70% of cases,
- absence of spasticity or vision/hearing loss,
- 40% autistic children have some form of epilepsy, 30% seizures,
- lack of focal dysfunctions,
- distributed neocortical system disorder.

Moreover there is common belief that patients with ASD more frequently have problems with association, prefrontal cortex rather than sensory-motor areas.

### 2.3 Symptoms

Many different symptoms of autism have been identified (see Fig. 2), including:

- difficulty in mixing with other children,
- preference to be alone, aloof manner,
- inappropriate laughing and giggling,
- little or no eye contact,
- inappropriate attachment to objects,
- children may not want cuddling or act cuddly,
- apparent insensitivity to pain,
- spins objects, sustained odd play,
- insistence on sameness, resists changes in routine,
- noticeable physical overactivity or extreme underactivity,
- echolalia (repeating words or phrases in place of normal language) – have words but not meaning,
- not responsive to verbal cues, acts as deaf,
- tantrums – extreme distress for no apparent reason,
- difficulty in expressing needs, gestures/pointing instead of words.

## 3 Theories

Zimmerman [1] included about 20 theories of autism spectrum disorders in his book. Only few of them are explained below.


Fig. 2: Selected symptoms of autism.

### 3.1 Minicolumnopathy

Minicolumnopathy is a theory proposed by Manuel F. Casanova [4]. It is based on observation that average minicolumnar width is 25.7 mm in autistic patients, in contrast to 27.2 mm in controls. This abnormality may cause smaller minicolumns in dorsolateral prefrontal cortex, increased cell density and shorter connecting fibers, which favor local computation.

Minicolumnar variability is the result of genetic and epigenetic influences that reduce combinatorial diversity within overlapping networks, necessary for behavioral flexibility, leading to autistic symptoms.

### 3.2 Mirror Neuron System

According to the theory of Mirror Neuron System (MNS) multimodal neurons in motor cortex react also to visual observations, help to understand actions of others. Distortion in the development of the MNS interferes with the ability to imitate, leading to social impairment and communication difficulties. However

there is also abnormal brain activation in many other circuits in ASD. Moreover children performance on various imitation tasks may be normal.

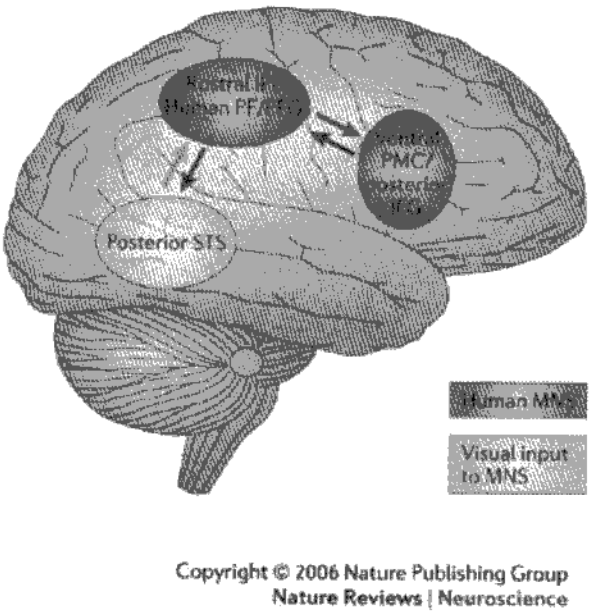




Fig. 3: Explanation of mirror neuron theory.

### 3.3 Underconnectivity Theory

According to the underconnectivity theory excess of low-level (sensory) processes, underfunctioning of high-level neural connections and synchronization, fMRI and EEG study suggests local overconnectivity in the cortex and weak functional connections to/from frontal lobes. Underconnectivity is mainly within each hemisphere of the cortex and that autism is a disorder of the association cortex. "Default brain network" (cingulate cortex, mPFC, lateral PC), shows low activity for goal-related actions; it is active in social and emotional processing, mindwandering, daydreaming. Activity of the default network is negatively correlated with the "action network" (conscious goal-directed thinking), but this is not the case in autism – perhaps disturbance of self-referential thought?

### 3.4 Empathizing-Systemizing Theory

Empathizing-systemizing theory is a newer version of the extreme male brain theory [5]: autism as an extreme case of the male brain, those individuals in whom systemizing is better than empathizing (according to psychometrical tests).

### 3.5 Executive Dysfunction Theory

According to this theory autism results mainly from deficits in working memory, planning, inhibition, and other executive functions, with superiority of locally oriented and perceptual brain operations.

### 3.6 Function Connectivity Theory

This model is developed since over 20 years by Nancy J. Minshew: autism as widespread disorder of association cortex, development of connectivity, only secondarily as a behavioral disorder.

### 3.7 Imbalanced Spectrally Timed Adaptive Resonance Theory

Theory iSTART [6], based on Adaptive Resonance Theory, assumes breakdown of interactions of cognitive, emotional, timing, and motor processes involving prefrontal and temporal cortex, amygdala, hippocampus, and cerebellum as cause of autistic symptoms. Autistic people have vigilance fixed at such a high setting that their learned representations are very concrete, or hyperspecific. It is interesting theory but also complex and hard to connect to molecular level.

## 4 Project Performance

### 4.1 Neural Model – Simple Mechanism

Our hypothesis is as follows: key role play processes of attention. These processes are a result of synchronization of groups of neurons, resultant from existing feedbacks, competition, inhibition and multiple constraint satisfaction. Neurons do not accommodate (desynchronize), creating deep and narrow attractors, that make attention shifts quite difficult. Most likely cause is damage of leaky ion channels. Attractor dynamics of two models implemented in the Emergent simulator [7] have been studied to verify this hypothesis.

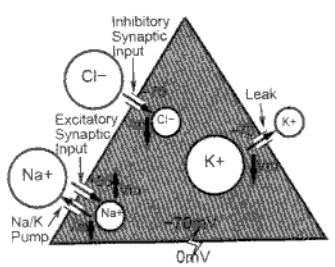


Fig. 4: Model of artificial neuron implemented in Emergent software [7], based on Hodgkin-Huxley model [11].

Our example, model of visual recognition, is defined as follows:

- recognition of two objects,
- processed by on-off cells,
- includes LGN, V1, V2, V4/IT, V5/MT layers (Fig. 5).

Spat1 has recurrent activations and inhibition, focusing on a single object. In normal situations neurons desynchronize and synchronize on the second object, and as a result: attention shift. Damage to leaky channels disables this process and the system cannot disengage attention from the first object. Autism may result from a simple low-level problem; all other problems may be a result of this disfunction influencing interest and development.

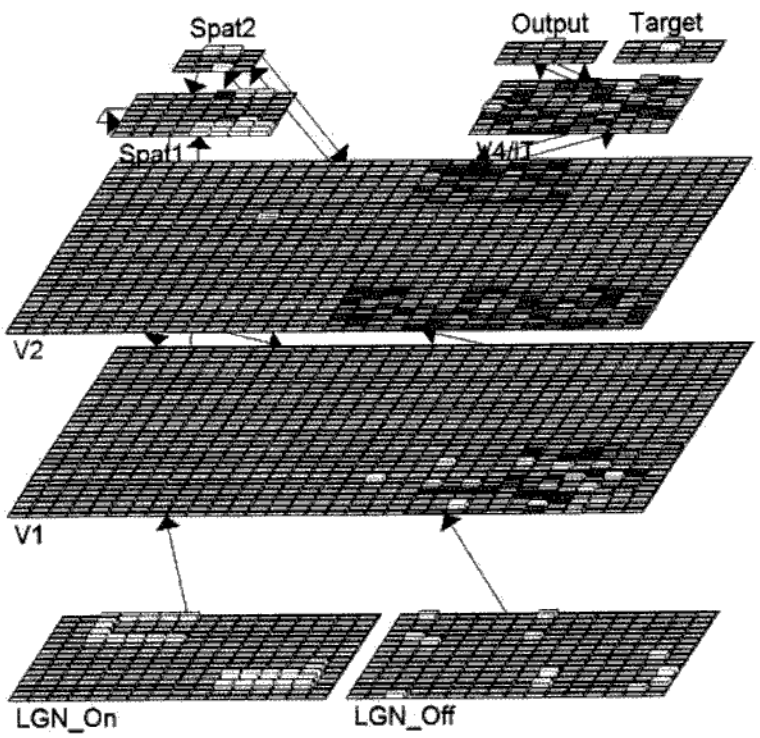


Fig. 5: Model of visual recognition based on [7].

However, there are one hundred billions ($10^{11}$) neurons in human brain and each of them may be connected with $10^4$ others which gives the degree of complexity by the order of $10^{15}$. Such structures cannot be simulated without supercomputing. Nevertheless, the good simulation of our relatively simple model requires high computational powers. Each neural cell is being simulated according to Hodgkin-Huxley model [11]. In the model each small part of cell (so called compartment) is represented by its equivalent electrical circuit. The behavior

of the circuit is described be set of several nonlinear differential equations. The model becomes more complicated if we take into consideration the influence of other ionic channels, different kinds of synapses and other parameters that may have influence on the behavior which is the point of our interest.

That is why more sophisticated simulations will be conducted in the parallel version of GEneral NEural Simulation System (GENESIS) [12] as some of the abovementioned parameters cannot be implemented directly into Emergent. Initial experiments have been already successfully completed on the local cluster, however, in future, for the big issues we are going to benefit from the support of the Polish Grid Project (PL-Grid) [13].

### 4.2 Attractors

Attention results from:

- inhibitory competition,
- bidirectional interactive processing,
- multiple constraint satisfaction.

Basins of attraction allow the system for object recognition for given input activations. In Fig. 6 Fuzzy Symbolic Dynamics (FSD) [8] visualization of attractors in semantic layer with 140 units is presented in weak (ASD), normal and strong (ADHD) accomodation case. In normal situation relatively large attraction basins lead to easy assasiations resulting in subsequent transitions from one basin to another, exploring the activation space. Weak accommodation prevent normal activation flow and in consequence form deep, narrow attraction basins making it hard to move out of them (i.e. assosiations are very weak). On the other hand strong accomodation lead to fast depolarization of neurons forming large, shallow attraction basins that prevent system from staying long enough in them resulting in inability to focus.

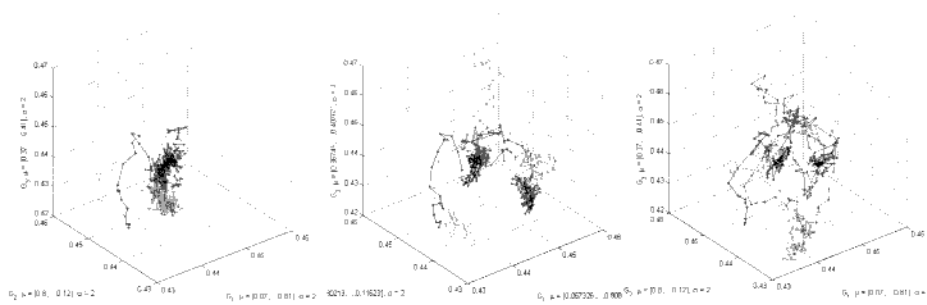

Fig. 6: FSD visualization of attractors in semantic layer with 140 units in weak (ASD), normal and strong (ADHD) accomodation case (left, center and rigth respectively).

### 4.3 Consequences of Deep Attractors

Deep basins of attraction may lead to the following abnormalities:

- deficits in attentional disengagement,
- precise memory for images, words, numbers, facts, movements,
- strong focus on single stimulus, absorption, easy sensory overstimulation,
- in motor cortex this leads to repetitive movements,
- generalization and associations are quite poor; integration of different modalities is impaired ⇒ development of connections;
- echolalia, repeating words without understanding (no associations),
- fast changes are not noticed (stable states cannot arise),
- faces are ignored (change to fast), contact with caretakers is difficult, gaze focused on simple stimuli; normal development relations, theory of mind, empathy - is impaired.

## 5 Concluding Remarks

Simple basic deficit can be a host of problems in the area of ASD because of severity and local expression, many insights from simple but general mechanism.

There are a lot of experimental evidences, because many studies verified attention deficits in autism [9, 10]. Simulations require high computational power. Their results will be presented during the future workshops.

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