

# Scientifically deconstructing some of the myths regarding autism

**Nouchine Hadjikhani**

Martinos Center for Biomedical Imaging, Harvard Medical School/MGH/MIT, Charlestown, USA; Gillberg Neuropsychiatry Centre, Göteborgs Universitet, Göteborg, Sweden

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## Summary

Autism Spectrum Disorders (ASD) represent a series of developmental conditions affecting social interactions and communication, that are accompanied by a number of co-morbidities. In the French-speaking European community, the myth that autism is caused by a bad mother-child relationship is still prevalent in some areas, despite all scientific evidence. Other myths remain about autism, and this paper addresses issues related to the prevalence, the associated symptoms in autism, and finally questions the notion of a lack of empathy in ASD.

*Key words: autism; brain imaging; aetiology; prevalence; empathy*

## Introduction

Several myths are frequently encountered regarding autism:

- Myth 1: Autism is caused by a poor mother-child relationship (a myth mostly restricted to the French-speaking European community).
- Myth 2: Autism is a rare condition and it is impossible to treat.
- Myth 3: People with autism cannot speak.
- Myth 4: Autism cannot be associated with a hyperactivity disorder (DSM IV TR).
- Myth 5: People with autism have no empathy.

In this paper, we attempt to present scientific evidence deconstructing these five myths.

Correspondence:

Nouchine Hadjikhani, MD, PhD  
Associate Professor in Radiology  
Harvard Medical School/MGH/MIT  
Martinos Center for Biomedical Imaging  
Building 75, Third Avenue, Room 1418  
Charlestown, MA 02129, USA  
Gillberg Neuropsychiatry Centre  
Göteborgs Universitet  
Kungsgatan 12  
SE-411 19 Göteborg  
Sweden  
nouchine[at]nmr.mgh.harvard.edu

## Myth 1: Autism is caused by a poor mother-child relationship

Autism spectrum disorders (ASD) are disorders that affect brain development, resulting in difficulties in social communication, repetitive behaviours and restricted interests. They are frequently accompanied by sensory disturbances in the domains of hearing, smell, touch, etc. [1].

When they described autistic symptoms for the first time in the 1940s, Leo Kanner in the U.S. and Hans Asperger in Austria had a clear organic conception of this condition [2, 3]. However, in the 1950's, Bruno Bettelheim imposed a psychoanalytic view of autism attributing responsibility for children's autism to parents, especially mothers, and comparing autistic behaviour to that of prisoners of concentration camps who coped with their extreme situation by withdrawing into themselves [4]. According to Bettelheim, like the camp prisoners, children who perceived their parents' desire that they not exist, coped with this lack of love and affection by locking themselves in an "empty fortress".

Autism made its entry in the DSM in the 1980s [5], yet in France, the Classification Française des Troubles Mentaux de l'Enfant et de l'Adolescent (CFTMEA) waited until November 2004 to stop classifying autism under the category of psychoses (although many psychiatrists/psychoanalysts continue to consider autism as a psychotic disorder, placing it in the category of schizophrenia and paranoia (see for example <http://www.courtil.be/feuillets/F29.html>). The French high authority of health published a report supporting a neuro-developmental conception of autism and evidence-based treatment, however, their recommendations are, unfortunately, not followed in many institutions.

ASD are neuro-developmental disorders with an onset during the gestational and early childhood period. The first evidence came from work by Beauman and Kemper [6], who showed in a series of autopsy cases the presence of abnormal neurons in the hippocampus, amygdala and cerebellum in people with autism, thereby dating a divergent developmental path before the 28th week of gestation. Nearly 20 years later, Casasova and colleagues [7] showed an anomaly in the structure of the cortex, with smaller and denser cortical columns (minicolumns) and more recently Courchesne et al. [8] showed a 79% increase in the number of neurons in the prefrontal cortex in autism. Yet, we know that cortical neurons only multiply during the prenatal period. In 2003, Courchesne et al. reported an anomaly in the growth of brain volume during the first two years [9], and Herbert et al. [10] described the presence of abnormalities in

the cerebral white matter that connects different parts of the brain. Finally, earlier this year Stoner and colleagues reported focal patches of abnormal laminar cytoarchitecture and cortical disorganisation of neurons in the prefrontal and temporal cortical tissue of children with autism [11], supporting the hypothesis of a dysregulation of layer formation and layer-specific neuronal differentiation at prenatal developmental stages.

The causes of autism are multiple and ASD are most likely a collection of endophenotypes that share clinical manifestations of socio-communicative disorders and repetitive behaviour disorders, and are associated with different co-morbidities. There are a number of aetiologies known to this day, that include genetic and environmental factors. Nearly a hundred genes (usually involved in synapse formation) have been associated with ASD [12]. Environmental factors include gestational exposure to toxicants, especially air pollutants and pesticides [13], and drugs (including valproate or SSRIs [14–16]), as well as maternal infection during the first trimester [17]. Finally, the important role of oxytocin during birth was recently demonstrated by Tyzio et al [18] in a genetic model and a teratogenic model of autism in rodents.

Brain imaging studies have shown differences in the brains of people with ASD compared with typically developing controls. In 2000, Zilbovicius et al. showed abnormal perfusion of the superior temporal sulcus [19]. Hadjikhani et al. [20, 21] and Dapretto et al. [22] have demonstrated abnormal spontaneous activation of brain areas containing neurons that are involved in mirror neuron mechanisms, and Hadjikhani et al [23] reported the presence of thinning of the cortex in these same regions. These areas are important not only for imitation (impaired in autism), but also to understand facial and emotional expression of emotion. Without a normal functioning of these regions, it is much more difficult to decipher facial micromimicry that play a very important role in the dynamics of social interactions.

Joint attention is also affected in individuals with ASD, resulting in difficulties in language acquisition in a significant percentage of people affected. Recently, using fMRI Zurcher et al. [24] demonstrated a lack of differential activation in individuals with ASD in response to social indicators of the presence of danger (averted gaze in a scared face); in contrast, in ASD participants we also demonstrated an increase in activation of the sub-cortical pathway for direct gaze. This pathway is important for fast processing of emotions, in particular for the treatment of stimuli indicating the presence of danger. A similar observation of increased sub-cortical activation when attending to the eye-region was also reported in another experiment by our group [25]. Hyperactivation of the subcortical pathway could be underlying active gaze avoidance, a very common feature in ASD, and we are currently pursuing our research on that topic.

In summary, autism is a condition resulting from an interaction between genetic susceptibility and environmental factors (chemical, viral, birth condition, etc.), which starts during pregnancy and delivery, and which results in anatomical and functional differences in the brain. Poor maternal relationship is absolutely not responsible for autism, and it is important to finally stop blaming mothers.

## **Myth 2: Autism is a rare condition, and it is impossible to treat it**

Autism affects boys four times more than girls. The prevalence of autism was estimated to be 1:5000 in 1975, 1:2500 in 1985, 1:500 in 1995, 1:110 in 2009, 1:88 in 2012 [26], and today it is 1:68 (and 1:42 for boys) [27]. Today, autism is more common than childhood cancer, juvenile diabetes and paediatric AIDS combined, and poses important challenges both in public health and in education. If the numbers above can be trusted, they would mean that there has been more than a 700% increase over the past two decades. But what do these figures really mean, and how do we interpret them?

On one hand, this increase is mostly due to a better understanding of the syndrome and its manifestations, resulting in increased detection, as well as to a broadening of diagnostic criteria. However, on the other hand, a real increase seems to also have taken place, which could be due to environmental factors, in combination with genes increasing susceptibility to these factors. Most remain mysterious, but among those incriminated are air pollutants, pesticides and drugs (see above). Advancing age of the parents, especially the father, is an additional risk factor [28]. However, vaccines play no role in the causes of autism [29–32].

More than one in a hundred children have ASD. Early diagnosis and intervention are crucial to take advantage of brain plasticity and help the child to develop as smoothly as possible (for review, see [33]). Early identification is also important in order to inform the parents and the preschool teachers about the child's cognitive difficulties in order to provide adequate help. Early intervention methods are based on cognitive-behavioural techniques methods (e.g., ABA, TEACCH, Early Start Denver Model) and aim, through positive reinforcement, at improving the child's social and communication functions, as well as reducing their repetitive and obsessional behaviour. It is important that parents and teachers are involved in all approaches, because the child must learn skills that are essential to everyday life, and the number of daily hours spent working on these areas is predictive of the child's success – although intensive training with a therapist (>15 h per week) does not seem to be necessary to achieve improvement, compared with non-intensive targeted intervention based on ABA conducted over long periods of time [34]. A recent meta-analysis evaluating early intervention in autism has shown that children receiving these therapies were better than others in the areas of intelligence, language development and adaptive functions [35]. Objective EEG activity changes in response to social stimuli after Early Start Denver Model intervention have recently been reported [36], demonstrating the effect of behavioural intervention on brain activity.

It should be noted that none of the 40 papers written since 1969 on psychoanalysis and autism have reported any efficacy of this approach in improving symptoms related to autism. In particular, “Hôpitaux de jour” are institutions where children with autism stagnate and do not acquire the necessary skills to function in society. Worse, in some of them “body therapies” without any scientific basis are ad-

ministered, that include “pateaugoire” (observing a child’s behaviour when placed in a small pool and drawing all kinds of conclusions about their psychological functioning) or packing (wrapping the naked child in wet and icy sheets) – a practice that is in principle prohibited today.

Autism can be understood as a lack of social instinct, and behavioural interventions should be rewarding and should systematically teach nonverbal and verbal communication, as well as emotional expression understanding and production from the youngest age. Brain imaging and eye-tracking studies suggest that there is an instinctive aversion to the eyes and to the face in individuals with autism, therefore training should include paradigms that make social stimuli, and in particular faces, interesting and rewarding to the child. The lack of development of mirror neurons found in anatomical and functional imaging is most likely due to a lack of training in imitation and mimicry, probably resulting from a decrease of attention to faces and social stimuli. Future research will shed light on brain plasticity and the effect of behavioural therapies on the anatomy and function of the brain.

In addition, new promising studies have been published concerning the specific improvement of social symptoms of autism with two molecules: oxytocin and bumetanide. Oxytocin administration increases the time spent looking in the eye region in faces, and improves, although for a short time, the autistic symptomatology [37, 38]. Bumetanide treatment improves subjective symptoms of autism [39]. Its effect is also visible in brain imaging, where increased activation in response to emotions has been reported after treatment [40]. The putative mechanism of action of these two substances is the recovery of the inhibitory function of GABA through modulation of the amount of neuronal chlorine.

In summary, autism is a condition that affects more than 1% of the population, and 1 boy in 42. Early diagnosis is crucial for early intervention, which is the best predictor of a favourable development of the child [41]. Cognitive-behavioural therapies are currently the only treatment for autism, although some drugs may be useful for addressing comorbidities such as sleep disturbance (melatonin) [42], or hyperactivity. Treatment with a molecule long known for its diuretic properties, bumetanide, looks promising and the results of multicentre randomised double-blind studies are expected in the coming years.

### **Myth 3: People with autism cannot speak**

A complete absence of language has long been considered a hallmark of autism. While it is true that, by definition, there is a language impairment in ASD, there is a large variety within the spectrum. Some children will have normal language acquisition (although with some pragmatic language oddities) – these children were classified as Asperger Syndrome until the DSM 5. Of the other children showing language delay, only about a quarter of them will remain non-verbal. A recent study examining 535 children with ASD with a history of severe language delay reported that 70% of these children were using phrases by age 8, and that

47% of them were fluent [43]. This study showed that positive predictors of language achievement were a high non-verbal IQ and less social impairment. Interestingly, sensory disturbances and the presence of stereotyped behaviour and repetitive interests were not delaying language acquisition. The authors concluded by underlining the importance of evaluating and considering nonverbal skills in children with language delay, and in taking these into account in early intervention.

It is; however, interesting to observe that even in fluent adults with ASD with a history of language delay and normal intelligence, subtle grammatical difficulties can be observed [44].

### **Myth 4: Autism cannot be associated with a hyperactivity disorder (DSM IV TR)**

In the previous version of the DSM (DSM IV TR) [45], a hyperactivity disorder (ADHD) excluded de facto autism spectrum disorder. While it has been demonstrated that ASD and ADHD are distinct and recognisable disorders, there is growing evidence that these two conditions share common features, that may be present in the same family, and that they probably represent different aspects of the same underlying disorder. Moreover, ASD and ADHD are very often associated, as has been demonstrated in several studies. About 30% of children with autism meet the criteria for ADHD – and a large number of children with ADHD also meet the criteria for autism because of social deficits, language delays and stereotyped behaviours [46, 47]. Indeed, a large number of syndromes (including attention-deficit/hyperactivity disorder, oppositional defiant disorder, tic disorder, developmental coordination disorder, and autism spectrum disorder) may overlap in the first years of life. It is for this reason that the term ESSENCE (Early Symptomatic Syndromes Eliciting Neurodevelopmental Clinical Examinations) was introduced by Gillberg in 2010 [48]. This term is intended to describe the reality of the complexity of clinical presentation of developmental problems in children before the age of five years. These children may present developmental disabilities in the fields of: (a) general development, (b) communication and language, (c) social inter-relatedness, (d) motor coordination, (e) attention, (f) activity, (g) behaviour, (h) mood, and/or (i) sleep. When a child presents with one or more difficulties in these fields, he/she should be referred to and thoroughly investigated by a multidisciplinary team in all domains of development.

Brain imaging studies have demonstrated similarities between ADHD and ASD, which may share anomalies in the region of the vermis of the cerebellum as well as a reduced connectivity of the resting state network [49, 50]. Similarities have also been observed in both conditions in studies looking at attentional processes [51].

In summary, autism spectrum disorders can be and often are associated with other developmental problems, and before age five it is more appropriate to speak of ESSENCE than to try to provide a definite compartmentalisation in one of the many neurodevelopmental disorders. It is important to investigate systematically and thoroughly all aspects

of the development of these children (cognitive, motor, language, attention and social interactions), and to start behavioural therapies as soon as possible.

### Myth 5: People with autism have no empathy

One often reads that people with autism do not have empathy, that they are cold, without feelings, acting like robots. But nothing is further away from reality.

People with ASD indeed have difficulty understanding social situations, and their apparent reduced reaction in response to other people's emotional reactions can be interpreted as a lack of empathy. Empathy, or the ability to feel what another person might feel in a certain situation, can be divided into several categories; cognitive empathy (or theory of mind), social empathy, and affective (or emotional) empathy. These three categories can be investigated independently (e.g., with the Empathy Quotient test (EQ) developed by Baron-Cohen [52]). People with autism suffer especially from deficits in the areas of cognitive and social empathy. Examples of questions that demonstrate the types of difficulties people with autism experience are: "I can easily predict what others will do" (cognitive empathy) or "I often find it difficult to know if something is rude or polite" (social empathy). However, within the category of emotional empathy (e.g., "I am overwhelmed by the suffering which is shown in the news"), people with autism have scores that are much closer to those of typical people.

When we observe someone suffering, we activate the same brain areas as those that are activated when we perceive pain: a network referred to as the pain matrix [53]. In a recent fMRI study [54], we used short films demonstrating facial expression of pain in patients undergoing mobilisation of their painful limb in a shoulder clinic, compared with neutral facial expressions elicited by the mobilisation of their healthy arm. A large group of people with autism and controls participated in this study (36 ASD, 31 controls). To our surprise, we found that people with ASD and controls had similar brain activation in response to perceived pain in others in the pain matrix mentioned above. That means that both groups reacted in the same way to the perception of other people suffering. When we lowered the statistical thresholds to see whether there was really no difference between the two groups, we observed an increase of activation in the prefrontal cortex, an area playing a role for the control of emotions, in people with ASD. From these data we can conclude that people with ASD not only fully perceive pain expressed by others, but that in addition they have to control their emotions to avoid experiencing personal distress. In a typical person, mentalising is the mechanism that allows distancing oneself from a distressing situation such as the expression of pain by another person. Mentalising is a function that seems to be challenged in autism. Interestingly, in specific situations, mentalising is not enough, and lateral prefrontal control is needed as well to allow the expression of caring behaviour. It has been observed recently that doctors showed activation in that specific region of the lateral prefrontal cortex (observed here in the people with autism) when they were observing painful

medical procedures, probably reflecting the strategies developed to remain effective and help patients [55].

Based on these data, we can argue that different processes are involved in the perception of social stimuli compared to those that present a phylogenetic function, such as perceived distress or pain expressed by another person. Our results, showing similar activation in areas involved in emotional sharing between participants with ASD and controls are consistent with the hypothesis that emotional empathy is preserved in people with autism. Interestingly, the dissociation between intact affective empathy and impaired cognitive empathy (ToM) in autism is the exact opposite of the profile observed in psychopaths, who have impaired affective empathy and intact cognitive empathy.

In summary, it is wrong to think that because they do not exhibit caring behaviour, people with autism are insensitive and lack empathy. In fact, it seems that they are rather hypersensitive, and their apparent demeanour actually reflects their effort to escape the overflow of emotion generated by the observation of pain in others.

In conclusion, the understanding of autism has been greatly improved thanks to its scientific investigations, and it is now clear that this neuro-developmental disorder needs early diagnosis and intervention in order to provide the best possible future to the >1% of children who are affected with this condition.

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