

# Imitation and Theory of Mind in Autism: A Review of Mirror Neuron System

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## ABSTRACT

Individuals with autism spectrum disorder usually suffer a combination of social interaction impairments, imitation deficits, theory of mind difficulties, sensory hyperreactivity, and repetitive behaviours. Researchers strive to identify the etiology or risk factors of this disability by developing a variety of theories, while the broken mirror theory, the broken mentalizing theory, and the intense world theory are three main candidates for explaining these symptoms. Specifically, the broken mirror theory regards the dysfunction of the mirror neuron system as a cause of imitation and theory of mind impairments in autistic individuals, whereas the broken mentalizing theory claims that the symptoms of autism result from the patients' mentalizing disability. Besides, the intense world theory blames the symptoms demonstrated above on the hyperreactivity and hyperplasticity of neural circuits in people with autism. In this paper, eighteen relevant articles or books were reviewed, indicating that current data regarding these three theories are very mixed. Overall, each of them can only provide partial explanations for these symptoms. However, although the causes of autism spectrum disorder still remain debated, the evaluation of these explanatory theories can effectively assist in advancing more successful methods to diagnose and treat this disability.

**Keywords:** autism, mirror neuron, imitation, theory of mind.

## 1. INTRODUCTION

Autism spectrum disorder (ASD) or autism spectrum condition (ASC) is a developmental disorder that leads to levels of difficulties in individuals' social interaction and communication [1]. It is considered presenting from birth but can be diagnosed when child is around two years old [1]. Children who are diagnosed with ASD usually show a combination of persistent social interaction impairments and restricted, repeated patterns of behaviour [2]. The severity of mental disability ranges from different levels [3]. Although the DSM-5 does not include any neuronal diagnosis, many brain imaging studies have confirmed different activities of neurons in brain of ASD patients and that of typically developing individuals. Mirror neuron system (MNS) is a specific group of neurons which respond to self-produced actions of an individual and the similar actions done by others that he/she observed [4]. In a typical developed human brain, the mirror neurons locate in inferior frontal gyrus (IFG), lower part of the precentral gyrus, the rostral part of the inferior parietal lobule (IPL) and the temporal, occipital and parietal visual areas [5]. MNS in human

brain is involved in many neurocognitive functions, such as action understanding, imitation, speech and language, theory of mind (ToM), social interaction and empathy. Particularly, the lack of ToM and imitation skills are highly related to characteristics of ASD. Some studies have found the link between atypical activation of mirror neurons and ASD, suggesting that the dysfunctions in MNS contributes to deficits in imitation and theory of mind [6, 7, 8]. Those studies have supported the broken mirror theory which considers dysfunction of MNS is a causal factor of deficits in imitation and ToM of ASD individuals [9]. Moreover, the broken mentalizing theory provides a cognitive view on behavioural symptoms of ASD, considering the deficits of behaviours of ASD are caused by the impairment of mentalizing modules of ToM [10]. Given that this situation, this study aimed to review possible causations of ToM and imitation disability of ASD individuals under broken mirror theory and mentalizing theory, followed by criticizing the two theories. Finally, further implications on diagnose and treatment of ASD will be provided.

## 2. THEORIES APPLIED TO EXPLAIN DEFICITS IN AUTISM SPECTRUM DISORDER

### 2.1. Broken mirror theory

The broken mirror theory suggests that ASD is to some extent caused by dysfunction of MNS and it can be deeper explained by three slightly different visions. The first one emphasizes the combination of extensive behavioural evidence (ASD individuals' poor imitation ability) and the neuroimaging evidence (role of MNS in imitation learning) [7]. Thus, it leads to the perspective that impairments of MNS is a possible factor that contributes to weak imitation skills in ASD. The second version highlights the crucial role of internal stimulation mechanisms, especially MNS, in normal development of imitation and ToM, proposing that this dysfunctional stimulation of MNS might cause imitation and ToM deficits in individuals with ASD [11]. The third one is the chaining vision which claims that chaining neurons only discharge when specific sequence of actions are observed or performed. Abnormality of this type of mirror neurons may cause difficulties in social cognition in ASD [12].

### 2.2. Broken mentalizing theory

The broken mentalizing theory suggests that individuals with ASD have difficulty imputing mental states to oneself and to others, proposing that they lack the ability of ToM [10]. In an experiment known as the false-belief task, Sally initially places an item, such as a ball, in a basket and then leaves. Then, Anne moves the ball to a nearby box. When Sally returns, the child who has witnessed the whole process will be asked to predict where Sally would look for her object. Those who believe that Sally tends to look her ball in the basket are considered to have the ability of ToM. Children with ASD are unable to pass the false-belief task and are suggested to have a broken theory of mind [10].

### 2.3. Intense world theory

Initially proposed by Markram and Rinaldi, the intense world theory suggests that people with ASD encounter the relevant symptoms mainly due to their hyper-functionality [13]. Specifically, since valproic acid (VPA) is believed to have the ability to cause autism, researchers made rats prenatally exposed to this drug and then studied their neuronal networks located in three brain regions, namely the somatosensory cortex, the prefrontal cortex, and the amygdala. As the results indicated, neural circuits in VPA-treated rats were more hyperreactive and hyperplastic than those in control rats [14]. In other words, for these VPA-treated rats, both the between-neuron connections within a local circuit and the persistent increase in synaptic strength became more significant following the electrical stimulation [14].

Based on this rat model of autism, Markram and Rinaldi therefore assumed that the hyper-perception, hyper-attention, and hyper-memory experienced by autistic individuals stemmed from their hyperreactive and hyperplastic local neuronal networks, which could be regarded as the core etiology of ASD [13].

## 3. DISCUSSIONS UNDER THE BROKEN MIRROR THEORY AND BROKEN MENTALIZING THEORY

The term imitation was defined as an individual's capacity to mimic or learn an observed performance [4]. ToM refers to the ability to attribute knowledge, intentions, beliefs and desires to self and others to further explain and predict behaviour [15]. Some studies have found neuroscientific evidence between abnormal MNS activation and impairment of imitation and ToM in ASD, supporting the broken mirror theory. For instance, Williams and colleague compared brain activation between typical developing individuals (control group) and individuals with ASD by using fMRI [7]. The findings indicated a reduced activity in anterior parietal areas in ASD brains during imitative actions, proposing that the impairment of MNS might be a causal factor to deficits of imitation skills in ASD [7]. Additionally, another study compared mu suppression (the sum of post-synaptic neuronal activity over an area) that measured by EEG between 14 ASD patients and 15 normal people during an imitation experiment. The findings reported a significant reduced mu suppression in ASD group [16]. Therefore, it is possible that less extensive activation between mirror neurons in ASD may have an impact on ToM and imitation, which supports the idea of broken mirror theory.

Although plenty of neuroscientific studies evident the crucial role of MNS in ASD, controversies related to its plausibility still exist [3]. The impairment of imitation and ToM in ASD might be influenced by deficits of other abilities that MNS support, such as language, empathy, goal understanding and emotion reading. This has greatly challenged the idea of broken mirror theory but the broken mentalizing theory could explain. For instance, in order to find connection between atypical activation of MNS and impairment of ToM and imitation skills in ASD, Cole and colleague tested the activation of MNS in young adults with ASD by using mentalizing tasks [8]. The results indicated a positive correlation between bad performances of intention-action task and reduced activation of MNS in participants' left hemisphere. Thus, the abnormal activation of MNS could be attributed to poorer connectivity between MNS and the sequence of mentalizing intentions to perform actions, which supports the theory of broken mentalizing modules in ToM [8]. Moreover, the false-belief task, a classic behavioural task that could

examine the ability of ToM of a person, presented that children with ASD are typically lack the ToM to pass this task [10]. Therefore, the findings fit the broken mentalizing theory which they have specific deficits in taking on the perspectives of others.

#### **4. DISCUSSIONS UNDER INTENSE WORLD THEORY**

Nevertheless, not all relevant experimental findings support the broken mirror theory or the broken mentalizing theory demonstrated above. For example, Southgate and Hamilton have reviewed a series of empirical evidence and have found that the broken mirror theory cannot fully account for the current experimental data [17]. Specifically, although autistic children are found to have a poorer performance on tests of the imitation of meaningless actions or facial expressions, they are able to correctly and effectively imitate goal-oriented actions when explicitly instructed to mimic, contradicting the broken mirror theory that individuals with ASD have a particular difficulty in imitating actions [17]. Critically questioned by some literature, the abnormal activation of the MNS does not provide effective reasoning for the idea that MN dysfunction leads to ToM and imitation deficits, but the sensory motor disorder in ASD individuals has an impact on producing a behaviour [3]. Although Iacoboni proposed from a cognitive perspective that imitative actions are defined by understanding and intention rather than motor properties, this theory was lack of neuroscientific evidence [6]. Similarly, some studies find that a proportion of individuals on autistic spectrum are able to pass the false-belief task [10,18]. The pattern of mixed results is difficult to reconcile with either a broken mirror module or a broken mentalizing model. Besides, neither of these theories can explain the symptoms of autism listed below: increased anxiety and sensory hypersensitivity.

Referred to as an alternative theory for autism, the intense world theory can bridge the gap between hypersensitivity, social disability, motor deficits, and theory of mind impairments [10,13]. According to this theory, autistic individuals can be characterized by hyperreactivity and hyperplasticity of neural circuits, both of which contribute to enhanced sensory sensitivity through hyperexcitable neuronal networks and long-term potentiation respectively [14]. Hyporesponse to social stimuli can also be explained by this theory: because ASD individuals usually undergo higher-than-average emotional intensity and hence suffer increased anxiety when interacting or communicating with others, avoidance behaviours are triggered, and ToM performance is also impaired during this process [10]. In addition to lack of social motivation and mentalizing ability, motor deficits, deemed to be another typical symptom of autism, can

be attributed to the prevention of hyperexcitable sensory stimulation [10]. Overall, this theory is worthy of researchers' substantial attention since it views ASD from a novel perspective, suggesting that these relevant symptoms emerge from a hyperreactive system rather than a hyporeactive one.

#### **5. IMPLICATIONS FOR DIAGNOSIS OF AND TREATMENTS FOR ASD**

In this paper, the connection between ASD and impaired MNS is analyzed, a comparison between the broken mirror theory and the broken mentalizing theory is made, the linkage between ToM deficits and imitation difficulties is explained, and an alternative theory for autism—the intense world theory is introduced. These theoretical considerations and discussions do have tremendous real-world implications, for example, a substantial improvement in diagnosis of and treatments for ASD. According to DSM-5 [2], the diagnostic criteria for ASD generally require the specification of symptoms at the behavioral level. However, the three theories discussed above, namely the broken mirror theory, the broken mentalizing theory, and the intense world theory, can additionally offer physiological, neural, and cognitive mechanisms for the symptoms of autism. If these theories are further supported by future experiments, dysfunctions in MNS, poor ToM performance, as well as hyperreactive and hyperplastic neural circuits can also be added to the diagnostic criteria for ASD. That is to say, when two people both develop the behavioral symptoms, the one with those physiological and neurocognitive manifestations demonstrated above should be particularly considered as having autism. Furthermore, interventions can be adjusted based on these theories, putting more emphasis on the treatments for abnormal MNS and hyperexcitable neural circuits.

#### **6. CONCLUSION**

This paper is to make a review of articles or books that were relevant to Dysfunction of MNS as a possible causal factor that may result in imitation and ToM impairments in ASD patients. Referring to broken mirror theory and broken mentalizing theory, many studies have found neuroscientific and cognitive experimental evidence to confirm that there is a link between abnormal activity of MNS and deficits of imitation and ToM abilities in ASD. As mentioned by Williams and colleague [7], reduced discharge of MNS was seen in ASD individuals than normal people during imitation and mentalizing tasks. This has supported the main idea of broken mirror theory. Another known task, the false-belief task, indicated that ASD individuals do not have the ability of ToM, which agreed with the broken mentalizing theory. Therefore, it could be concluded that the dysfunction

of MNS may cause the imitation skills and ToM ability to be impaired in ASD [10]. However, this view was questioned by its plausibility as the motor disorder could not be excluded to have an impact on producing a behaviour. What is more, the two theories were criticized by some researches that not all studies indicted the same result as the two theories suggest and the intense world syndrome has provided another possible reasoning of the impaired imitation and ToM in ASD. Generally, these theories could offer neurocognitive help for diagnose and treatment of ASD.

## AUTHORS' CONTRIBUTIONS

This paper is completed together by Yupeng Yuan and Chenxi Zhu.

Yupeng Yuan is responsible for reviewing, analyzing and summarizing studies related to the relationship between imitation deficits and theory of mind difficulties in autistic individuals and mirror neuron system impairments, by introducing and discussing statements and studies relevant to the broken mirror theory and the broken mentalizing theory.

Chenxi Zhu is responsible for introducing statements and studies related to the intense world theory, analyzing the mixed pattern of current experimental findings regarding the relationship between mirror neuron system impairments and autism, summarizing experiments whose results are opposed to the broken mirror theory and the broken mentalizing theory, as well as explaining the implications of this paper.

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