

RESEARCH GROUP: INTERDISCIPLINARY LABORATORY FOR RESEARCH IN AUTISM (LABIRINTO)

## A SYSTEMATIC REVIEW OF THE INFLUENCE OF MIRROR NEURONS IN AUTISM SPECTRUM DISORDERS

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### Abstracts

The objective of this study was to conduct a systematic review of experimental studies performed to assess the role of mirror neurons in the pathophysiology of autism. Four papers reported that areas of mirror neurons (MN) were under-active in autistic patients, giving weight to the theory of MN as a cause of ASD. Three papers indicated that MN were activated during the proposed activities, advocating atypical activation of MN, but not necessarily hypoactivation of these areas. One of the articles reported that only part of the MN system or just those areas of interconnected neurons are dysfunctional in autism. Analysis of the selected studies showed a correlation between dysfunction of the mirror neuron system and the main symptoms of autism such as deficits in social cognition, complete absence or a reduction in the individual's interaction with his/her social environment and a failure in the neural mechanisms of imitation.

**Keywords:** Mirror neurons; Autism disorder.

### INTRODUCTION

Autism is a developmental disorder that is characterized by clinical manifestations related to restricted and stereotyped interests, difficulty in social interaction and reciprocal social communication.<sup>(1)</sup> There are varying degrees of severity. One of the theoretical models developed to explain the pathophysiology of this syndrome is the “broken mirror” theory, which postulates that in autistic individuals there is a dysfunction in the neurons located mainly in the human frontal and temporal lobes, particularly in Broca's area.<sup>(2,3)</sup> Analysis of brain activation using techniques such as positron emission tomography (PET), transcranial magnetic stimulation (TMS) and

functional magnetic resonance imaging (fMRI) has indicated that mirror neurons (MN) are responsible for functions that include the ability to observe and imitate mechanisms of empathy, communication and social interaction, facets of human behavior that are impaired in autistic patients.<sup>(4,5)</sup>

## **OBJECTIVE**

The objective of this study was to conduct a systematic review of experimental studies performed to assess the role of mirror neurons in the pathophysiology of autism.

## **METHODS**

A systematic literature review was conducted to identify scientific articles published from 2006 onwards in the PubMed, Bireme, SciELO and Cochrane databases using the keywords: “mirror neuron”, “autism” and “human”. Inclusion criteria consisted of experimental trials involving human subjects; studies published in the English language; the presence of a control group that was compared to patients with an autism spectrum disorder; and the use of functional MRI as the evaluation method. In the absence of an ideal scale for specifically evaluating experimental studies, the methodology of all the studies meeting the inclusion criteria was assessed for quality using an adapted version of the Newcastle-Ottawa scale. Articles in which the quality of the methodology was deemed satisfactory were included in the study.

## **RESULTS**

The literature review initially yielded a total of 115 papers. Based on an analysis of the abstracts, 44 were found to meet the inclusion criteria and were then analyzed in their entirety. Of these, 8 were selected for inclusion. All the papers selected consisted of experimental studies involving a group of individuals with autism spectrum disorder (ASD) and a control group of typically developed individuals, with fMRI being used to analyze the degree of activation of areas of MN during both the observation and the performance of various activities. Four papers reported that areas of MN were under-active in autistic patients, giving weight to the theory of MN as a cause of ASD. Three papers indicated that MN were activated during the proposed activities, advocating atypical activation of MN, but not necessarily hypoactivation of these areas. One of the articles reported that only part of the MN system or just those areas of interconnected neurons are dysfunctional in autism.

## DISCUSSION

Specific and increasingly advanced experiments to assess activation of the mirror neuron system (MNS) in patients with ASD are of the utmost importance. The systematic literature review performed here involved studies conducted to evaluate the function of areas of the MNS in non-autistic and autistic patients, verifying the principal findings with regard to this correlation. The exact location of MN in the human brain remains a hotly debated issue; however, there is a consensus with respect to some areas such as the inferior frontal gyrus (IFG), also referred to as the pars opercularis or Brodmann area 44, the superior temporal sulcus (STS), the prefrontal cortex and the anterior intraparietal sulcus (IPS). Several papers have been published confirming the presence of MN.<sup>(6,7)</sup> Other regions have been associated with the presence and function of MN, the inactivation of these regions being associated with dysfunction in this group of neurons. One example includes face perception areas such as the amygdala and face-selective areas in the fusiform gyrus<sup>(8)</sup> and the limbic system, which appear to be modulated by the anterior insular cortex.<sup>(9)</sup>

## CONCLUSION

Analysis of the selected studies included in this systematic review showed a correlation between dysfunction of the mirror neuron system and the main symptoms of autism spectrum disorder such as deficits in social cognition, complete absence or a reduction in the individual's interaction with his/her social environment and a failure in the neural mechanisms of imitation.<sup>(5,10)</sup> Nevertheless, much controversy remains with respect to the dysfunctional areas, the degree of inactivation of these areas and, in particular, the stimuli used to test them. The main issue in analyzing MN is the impossibility of isolating them and evaluating their activity and function separately.<sup>(11)</sup> Many studies have been conducted with the objective of assessing this correlation; however, there are numerous biases in most of these studies. Consequently, there is a need for further, better-designed studies before mirror neuron dysfunction can be firmly established as the cause of autistic spectrum disorder.

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