# The Relationship Between the Autism Spectrum Disorder and the Mirror Neuron System

Autism is a difficult disorder that many children and their parents have to go through painfully. Recent hypotheses theorized that this has something to do with the brain's ability to imitate, which is a major function of the mirror neural system. The autism spectrum disorder is a complex disorder, which means that it is caused by many factors. Distortion in development of the mirror neuron system is one of the important factors. People with damaged mirror neuron system usually cannot imitate, which means that they cannot convert sensory stimuli into motor representations. They also have different white matter microstructure and cortical activity from normal people.

## Summary

In order to determine the neurological explanation for autism and autism spectrum disorders, the study of Martineau et al. aimed to test the truth of the hypothesis that children with autism show "dysfunctional mirror neuron activity" as triggered by the mu suppression (Martineau et al., 2008, p. 36). This hypothesis was based on the observation that children with autism have a problem with imitation of movements. Since the mirror neurons are in charge of this task, then it is assumed that the activity of the mirror neurons in children with autism is impaired. Mu expression is used to gauge and indicate mirror neural activity, thus this is expected to be observable in normal subjects and it is expected to be lacking in autistic subjects. Martineau et al. (2008) utilized data from the EEG activity of autistic children and 14 normal children with ages ranging from 5 years and 3 months to 7 years and 11 months (p. 36). Results showed that among the healthy subjects, there was desynchronization of EEG in the areas of the motor cerebral cortex and the frontal and temporal areas. On the other hand, there was no instance of desynchronization that existed among the autistic subjects. Instead, the autistic children had an increased cortical activity in the right hemisphere in the posterior region. Desynchronization means the synchronous firing of large numbers of neurons. This was present in normal children but absent in autistic children (Martineau, 2008, p. 38).

In the study by Oberman and Ramachandran (2008), the authors try to investigate whether children with autism showed a significant difference in the performance of the bouba-kiki task when compared to normal, or neurotypical, participants. The bouba-kiki task indicates mirror neural activity and seeks to measure and involve the activity of the Broca's area, angular gyrus and the superior temporal gyrus (p. 350). The method used to test the hypothesis was the bouba-kiki method, where subjects are made to "pair nonsense shapes with nonsense words" in order to determine the activity of the mirror neuron-like system, and thus the multisensory integration system in the child (p. 348). The results of the study by Oberman and Ramachandrean (2008) showed that normal children paired the nonsense shape with the nonsense word that had a phonemic structure that resembled the visual shape of the nonsense shape. The normal children did this 88% of the time, probably due to the active mirror neural system. Whereas, the autistic children group did so only 56% of the time (p. 351).

Lastly, based on the study by Frundt et al. (2017), the mirror neural system is connected with the severity of autism symptoms. However, Frundt et al. (2017) made it clear that the specific connection related to autism was the connection "between the right SMG [supramarginal gyrus] and the right IFG [inferior frontal gyrus]" in inverse proportion to the severity of the autism (p. 9). This specific connection corresponds to the mirror neural system's centers related to "multimodal integrative brain functions and language" (p. 9). Thus, for Frundt et al. (2017), the conclusion is that autism is not about the inactivity of the mirror neural system but a specific part of it (p. 10).

## Review and Evaluation

The studies by Martineau et al. (2008) and Oberman and Ramachandran (2008) both perfectly fit the meaning of the thesis statement, especially that a damaged mirror neuron system contributes to the lack of power of imitation and therefore becomes a significant basis of autism. However, latest information from Frundt et al (2017) proves that there are multiple connections the make up the entire mirror neural system, and that there is only one specific pathway that is linked to the severity of the autism disorder. According to Frundt et al. (2017), it is not the entire mirror neural system itself that is responsible for autism but the "right-hemispheric fronto-parietal MNS pathways" only that is connected with autism disorder severity (p. 10).

In fact, the findings do not contradict each other. The findings of Martineau et al. (2008) and Oberman and Ramachandran (2008) clearly reveal a more general discovery regarding the significance of the mirror neural system to children and adults with autism. The major finding in these two studies was that the mirror neural system determined autism, or that autism heavily depended on the activity or non-activity of the mirror neural system. The study by Frundt et al. (2017), which was conducted 9 years after those of Martineau et al. (2008) and Oberman and Ramachandran (2008), actually only negated the idea that the mirror neural system was totally impaired in children with autism. In fact, only a part of it was impaired. According to Frundt et al. (2017), this part was the "right-hemispheric fronto-parietal MNS pathways" (p. 10). Still, however, all three studies confirm the idea that the mirror neural system, or MNS, is the main factor behind the severity of autism or any autism spectrum disorder.

I agree with these findings because it is a fact that impairment with imitation is a major indicator of autism. Thus, those with autism actually have problems with this part of their brain, which corresponds to the ability to imitate.

The findings in the study by Frundt et al. (2017) is more significant than those of Martineau et al. (2008) and Oberman and Ramachandran (2008). This is because the findings of Frundt et al. (2017) dealt with the more specific part of the mirror neural system. Moreover, as implied from this study, the mirror neural study is actually a much larger system of neurons than previously thought. Thus, only one portion of it may be in charge with autism spectrum disorders. It is therefore possible that the other parts of the mirror neural system are associated with other memory diseases. Furthermore, since Frundt et al. (2017) does not entirely contradict Martineau et al. (2008) and Oberman and Ramachandran (2008), then it is clear that it merely adds to their information.

There are no interpretations that should be challenged here in these studies. However, it is important to know whether each autism spectrum disorder has differences in the way the mirror neural system is damaged. Another question would be whether a different damaged part would result in a different disorder.

## Conclusion

The autism spectrum disorder is determined by a problem or defect in the mirror neuron system. As proven by the studies of Martineau et al. (2008) and Oberman and Ramachandran (2008), total impairment of the mirror neuron system brings about autism. However, Frundt et al. (2017) clarified that only a problem with the "right-hemispheric fronto-parietal MNS pathways" in the mirror neuron system accounts for autism (p. 10).

## References

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