A critique of the mirror neuron hypothesis of autism

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Abstract
Since the discovery of multi-modal mirror neurons in the human brain, a broad range of socio-cognitive impairments and related disorders have been attributed to the dysfunction of the mirror neuron system (MNS). Specifically, one theory postulates that an impaired MNS in individuals with autism spectrum disorder (ASD) is responsible for deficits in socio-cognitive functions including action understanding, empathy, and imitation. Despite research on this connection, there is little direct evidence to support this mirror neuron hypothesis of autism in its current form. Rather, there is growing body of evidence that directly challenges this theory, suggesting that an impaired MNS is not a characteristic of ASD.

Keywords: autism spectrum disorders, mirror neuron hypothesis of autism, imitation, empathy, action understanding

The discovery of the mirror neuron system (MNS) was an important development in cognitive neuroscience, specifically in the study of social cognition. Mirror neurons are association cells that are activated by executing and observing an action (Dapretto et al., 2006). These specialized neurons allow for a multimodal representation of action by connecting seeing and doing in the visual and motor cortices (Williams, Whiten, Suddendorf, & Perrett, 2001). The MNS is theorized to underlie a broad range of higher-order social-cognitive abilities including action understanding, theory of mind, empathy, and imitation (Hickok, 2009). Conversely, disorders characterized by varying degrees of difficulty in understanding social interactions, verbal, and nonverbal communication, were then proposed to be related to deficits in the MNS (Rizzolatti & Fabbri-Destro, 2010).

A prominent example of this proposed connection between the MNS and disruption in behaviour involves autism spectrum disorder (ASD). ASD patients demonstrate symptoms aligning with impaired MNS functions, a theory often referred to as the mirror neuron system hypothesis of autism (Iacoboni and Dapretto, 2006). This theory is rooted in the controversial idea that intact mirror neurons lead people to understand others’ actions (Cattaneo et al., 2007). However, the true function of mirror neurons remains highly debatable, as researchers have not yet discovered their involvement in understanding actions (Hickok, 2009; Press, Richardson, & Bird, 2010). This paper will review the current literature on the role of the MNS in action understanding and imitation and its implications in ASD, and this paper will argue that the evidence for this proposed relationship lacks empirical tenability.

The Mirror Neuron System
Mirror neurons were first discovered over twenty years ago by a group of Italian researchers studying neurons in the ventral premotor cortex of macaque monkeys. These neurons fired not only when the monkey grasped an object, but also when the monkeys observed the researchers grasping the object (di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992). The discovery led researchers to investigate a homologous human system of mirror neurons (Rizzolatti, Fabbri-Destro, & Cattaneo, 2009; Rizzolatti & Fabbri-Destro, 2010). The first indirect evidence for a human correlate of MNS came from electroencephalogram (EEG) recordings. Specifically, mu wave blocking appeared when a person moved or when they observed movement (Altschuler et al., 2000). Likewise, PET scans exhibited patterns of activation in brain locations analogous to mirror neuron areas in monkeys (Rizzolatti et al., 2010). In particular, behavioral data indicates that observing someone else perform an action facilitates action execution (Bortolotto, Mattingley, & Cunnington, 2013). Thus, it was soon established that a mirror neuron system exists in human brains, wherein the mirror neurons are selectively activated when carrying out and viewing similar actions (Keysers, 2009).

The evidence for a human MNS has spurred re-
search into its possible role in cognitive and social processes (Fan, Decety, Yang, Liu, & Cheng, 2010). Several transcranial magnetic stimulation (TMS) studies show that mirror neurons map sensory information of observed actions onto the motor programs activated when an observer performs the same action (Borroni, Montagna, Cerri, & Baldissera, 2005; Gentitano, Mottaghy, & Pascual-Leone 2001; Maeda, Kleiner-Fisman, & Pascual Leone, 2002). To date, most research has focused on visual information as a subset of sensory processing. Following this discovery, various theories attempted to link this mechanism to social cognition and social abilities. This is because our interactions with others often involve understanding, and sometimes mimicking, the behaviours and actions of others (Fogassi et al., 2005; Iacoboni, 2009; Ramachandran, 2012). Researchers conclude that the human MNS is a circuit involving two main components: the parieto-frontal component, and the insular-cingulate component. The former, composed of the inferior parietal lobule, the ventral premotor cortex and the caudal inferior frontal gyrus, may be involved in action understanding, intention understanding and imitation (Cattaneo et al., 2007). The latter, composed of the insula and the anterior cingulate gyrus, may be involved in recognizing emotion (Cattaneo et al., 2007).

**Autism Spectrum Disorder and the Mirror Neuron System Hypothesis**

Autism spectrum disorder (ASD) consists of a group of neurodevelopmental disorders, including autism and Asperger’s disorder, childhood disintegrative disorder, and pervasive neurodevelopmental disorder, with an expression of symptoms ranging from mild to severe (Lauritsen, 2013). Although individuals with ASD often display inconsistent clinical profiles, there are common symptoms in three domains: difficulty with social interactions, verbal and non-verbal communication deficits, and a narrow range of behaviors and interests (Gallese, Rochat, & Berchio, 2013). According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-V; American Psychiatric Association, 2000), people with ASD often have difficulties in simple imitation tasks (Fan et al., 2010).

There is some evidence of a disruption of neural pathways in ASD, specifically in the connectivity between the MNS and its projections to the association cortex (Just et al., 2007). Indeed, fMRI studies reveal reduced functional connectivity between cortical regions (i.e. frontal and parietal areas) during task execution for a standardized executive function task and for the Tower of London Puzzle (Just et al., 2007). Notably, individuals with ASD usually show activation of brain regions similar to those of healthy controls during working memory tasks, but there is often a difference in the timing and patterns of activation across these regions (Minshew & Williams, 2007). This abnormal stimulation is primarily seen in individuals with high-functioning autism who have deficient processing for tasks that require integrating information from multiple neural systems (Minshew & Williams, 2007).

This evidence of neuropathology then led to the suggestion of a disruption in the neural machinery that aids in the development of social cognition (Fan et al., 2010). However, understanding all aspects of the neural correlates of ASD may be difficult due to the heterogeneity of the spectrum disorders. The varying symptom severity seen in different diagnoses for ASD may be commensurate with the multiple etiologies for different parts of the ASD spectrum. Additionally, the neurobiology of social cognition itself may be difficult to map given our incomplete understanding of the complex and multi-layered nature of social cognition (Southgate & Hamilton, 2008). For this reason, the excitement surrounding the discovery of mirror neurons and the focus on their role in social cognition was central to the development of the mirror neuron system hypothesis of autism (Press et al., 2010). This hypothesis postulates that the socio-cognitive deficits observed in ASD are due to the failed development of the MNS in early life, causing an abnormal representation of observed actions (Oberman & Ramachandran, 2007; Press, Richardson, & Bird, 2010). Although some research shows a link between mirror neurons and social cognitive functions, including empathy, action understanding, and imitation (Marsh & Hamilton, 2011; Rizzolatti & Craighero, 2004; Southgate & Hamilton, 2008), this paper will discuss significant gaps in the literature.

**Empathy and Action Understanding**

Theories linking the MNS to social cognition assume that the dual activation of observing and execut-
ing actions provides the observer with information on what actions occur and why (Iacoboni et al., 2005). These theories use the MNS to mechanize the complex process of intention understanding and, if taken a step further, to mechanize the empathy process (Cattaneo, 2007; Ramachandran, 2012). In theory, the insular-cingulate component of the MNS could transform the emotions an observer infers from other people's actions into the visceromotor responses the observer feels, which could be a possible mechanism of empathy (Fabbri-Destro & Rizzolatti, 2008). However, linking these basic mechanisms to empathy, a complex behaviour, is a significant claim to make. It requires more empirical evidence than is currently available as it involves understanding the thoughts, sensations, emotions, feelings, and beliefs of another person (Corradini & Antonietti, 2013). In fact, individuals with ASD have shown heightened emotional empathy, or the ability to understand the emotional mental state of another person, and motor empathy, or the tendency to automatically mimic another individual's movements (Bird, Leighton, Press & Heyes, 2007; Smith, 2009). Individuals with ASD exhibited heightened responsiveness measured with electromyography to happy and to fearful facial expressions (Magnée et al., 2007).

Action understanding is the ability to derive the meaning of an action through observation to direct future behaviour (Rizzolatti et al., 2001). The parieto-frontal component of the MNS has been proposed to be responsible for action understanding because the MNS maps a person's actions onto the observer's motor representation (Keysers, & Gazzola, 2006). Moreover, mirror neurons also code for goal-directed actions (Fogassi et al., 2005; Cattaneo et al., 2007), thus they are maximally activated when an action has a specific goal or an action is a part of a chain. Indeed, the mirror neurons of a monkey were differentially activated when a researcher grasped an object to eat it compared to when the researcher placed it in a container (Fogassi et al., 2005). This suggests that mirror neurons are goal-directed, which links action understanding to the MNS (Fogassi et al., 2005).

Despite these findings, evidence that the MNS allows for action understanding is indirect and empirically untenable (Cattaneo, 2007; Hickok, 2009). Action understanding is a complex process that requires the processing of context, perspective, and goals, among other situational factors. There may be a double-dissociation between the MNS and action understanding, which provides evidence against the theory of MNS as the basis for action understanding. Monkeys, the model animal in which researchers discovered mirror neurons, can view and process an observed action but they can not understand the meaning of the action (Hickok, 2009). However, humans can certainly understand actions they have not yet seen or performed, whereas monkeys can not. Yet mirror neuron systems have been found in both human and monkey brains. This casts doubt upon whether mirror neurons evolved for action understanding (Hickok, 2009). Mirror neurons may contribute to action processing, but mirror neurons are likely not the sole mechanism for understanding; they may be far too simplistic and reductionist to mechanize functions that require higher-order cognition, such as empathy and action understanding.

Furthermore, studies evaluating the connection between action understanding, the MNS, and ASD have been criticized for not distinguishing between the effects of an impaired MNS and the effects of impaired motor chains underlying action representation. For example, the motor chain for eating begins with neurons needed to grasp the food and ends with neurons needed for bringing the food to the mouth. Children with ASD lack the chained organization of motor programs seen in typically developing children. This could cause their reduced ability to understand the actions of others, rather than an impaired MNS (Cattaneo et al., 2007). Further, children with ASD do not show a significant decrease in their ability to understand the intentions of others (Carpenter, Pennington, & Rogers, 2001), a skill that has been associated with the MNS (Fogassi et al., 2005). Therefore, the theory that individuals with ASD possess poor action understanding skills is due to an impaired MNS lacks evidence to support its claim.

**Imitation**

In addition to understanding the actions and intentions of others, the ability to imitate involves activation of motor programs for similar observed actions (Hamilton, Brindley, & Frith, 2007). For example, if observing someone throw a ball activates the same motor
program involved in throwing a ball, this could explain how one prompts the necessary motor program to imitate this action. Action imitation activates MNS circuits at both single-cell and at neural-system levels (Keysers & Gazzola, 2006; Iacoboni, 2009). Indeed, observing actions when observers are told to imitate increases MNS activation compared to observing actions without being instructed to imitate (Decety et al., 1997). Similarly, a damaged MNS leads to impaired imitation of hand actions (Iacoboni et al., 1999, Southgate & Hamilton, 2008). This evidence, though indirect, for the involvement of the MNS in imitation (Iacoboni et al., 1999) has been connected to ASD in a meta-analysis study showing the disruption of imitative abilities as a core feature of ASD (Williams, Whiten, & Singh, 2004).

There may be a connection between MNS regions and imitation (Keysers & Gazzola, 2006). However, if mirror neurons allow for the transformation of sensory input into motor output, this does not fully explain imitation (Fan et al., 2010). Undoubtedly, imitation is more than mirroring, as imitation requires coordination in many areas: visual analysis, motor control, and selecting when and what to imitate. There is no direct evidence that the central function of the MNS is imitation. In fact, the MNS is more active during complementary actions than during imitative actions (Southgate & Hamilton, 2008).

Further studies exploring the mirror neuron system hypothesis of autism have shown mixed results on the true nature of the connection between the MNS, imitation, and ASD (Marsh & Hamilton, 2011; Press et al., 2010). Often, the imitation tasks children with ASD fail most do not require the hand-goal mirror system or goal understanding (Hamilton, et al., 2007). These findings are inconsistent with the mirror neuron hypothesis of autism.

As with action understanding, skeptics of the mirror neuron system hypothesis of autism argue that imitative deficits in people with ASD are due to impairments in other brain systems (Press et al., 2010). Although children with ASD often cannot complete imitation tasks without instruction, they usually can imitate when given instructions (Hamilton, 2007). In fact, in one study children with ASD exhibited an enhanced automatic imitation effect of human actions (Bird et al., 2007). Therefore, rather than causing generalized imitation deficits, ASD seems to cause deficits in the selective ability to know what or when to imitate. This inability to pick up on social cues and communication deficits is characteristic of people with ASD, and could be the cause of the imitative issues in ASD, unrelated to impaired mirror neurons (Southgate & Hamilton, 2008).

**Conclusion**

Despite the seemingly intuitive nature of the mirror neuron system hypothesis of autism, there are mixed results on its effectiveness. Though some studies show a link between mirror neurons and ASD, skeptics question whether these studies distinguish between the social deficits of ASD as an impairment of the mirror neuron system and that of other systems (Press et al., 2010). Both imitation and action understanding deficits in people with ASD are not directly related to a dysfunctional MNS. Other explanations, such as impaired visual processing of motion, dysfunctional attentional networks, or other compromised lower level systems, could account for the differences in mirror neuron activity in neurologically healthy people and in those with ASD. As mirror neurons are a part of a broader network in the brain, distorted input to the MNS could cause the deficits in imitation and action understanding seen in people with ASD (Oberman et al., 2005). To a certain extent, MNS functioning may be preserved in people with ASD. For instance, an EEG study showed that subjects with ASD and control subjects exhibit a suppression of mu rhythms in the sensorimotor cortices when observing object-directed actions (Fan et al., 2010). However, the mu suppression was lessened in individuals with worse symptoms. Perhaps the mixed evidence present in the literature is due to the heterogeneity of ASD. Could an impaired MNS contribute to some disorders of the autism spectrum but not others? More extensive research is required to make definitive claims that bridge the current gap in the literature.

The MNS may be a simplified mechanism that cannot fully account for the multi-layered and complex nature of social cognition and, thereby, ASD. However, impairments of a more basic mechanism, on which mirror neurons rely, could account for the social cognitive impairments associated with ASD. Future studies on the MNS may find other systems that interact with the MNS. This would allow us to better understand the
neurobiology of ASD. However, the intense focus on the effects of mirror neurons on social cognition and ASD has led to an underdeveloped, empirically unsound body of research. The gaps present in the literature must be addressed before the mirror neuron hypothesis of autism may be considered a valid theory. Research aiming to understand ASD may use neuroimaging, behavioral, or other diagnostic tools, along with better models of social cognition beyond the MNS.

References


