A systems level analysis of the mirror neuron hypothesis and imitation impairments in autism spectrum disorders

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1. Introduction

From children dressing up like their favorite TV characters to adults mirroring each other's body posture at a board meeting, imitation is a ubiquitous and fundamental aspect of human social behavior. Indeed, imitative behavior begins very early in life. As early as 42 h after birth, newborn infants have been found to mirror simple actions of others, such as tongue protrusion, lip smacking, and mouth opening (Meltzoff and Moore, 1983). These automatic imitation behaviors have been shown to decrease around two months of age (Abravanal and Sigafoos, 1984; Field et al., 1986; Fontaine, 1984) and reappear with increased complexity around one year of age (Meltzoff and Moore, 1992), suggesting that rudimentary imitation ability is present very early in life but may develop and change significantly over time.

Early imitation is thought to play a substantial role in the development of motor control, communication, and social abilities (Tomasello et al., 1993). For example, as children begin to speak,

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imitation of mouth movements is thought to teach them how to manipulate their own articulators (Jordan and Rumelhart, 2002). Imitation has also been implicated in the comprehension of others' behavior (Goldenberg and Karnath, 2006), with its earliest forms acting to provide a sense of connectedness between an infant and its world. In other words, imitation provides the child with information about the actions and intentions of the physical and the social world, which assists in the process of social learning (Rogers et al., 2003), and forms the foundation for future social development.

2. Mirror neuron system (MNS)

Because of the developmental significance of imitation, recent interest has centered on the neural substrates that mediate imitation. Specifically, the discovery of mirror neurons in nonhuman primates has offered promising clues to how we perform actions and perceive the actions of others. In nonhuman primates, neurons dedicated to the visual processing of the actions of others were identified mainly in area F5, and in area PF (Rizzolatti and Craighero, 2004). These neurons not only fire when a monkey performs an action but also when a monkey watches an action being performed (Gallese et al., 1996; Rizzolatti et al., 1996). While it may be difficult to directly study the existence of mirror neurons in humans, a substantial number of fMRI and EEG studies have found evidence that a homologue of the monkey mirror neurons exists in humans (Iacoboni and Dapretto, 2006; see Turella et al., 2009 for alternate perspective), where the monkey F5 is thought to loosely correspond to the human inferior frontal gyrus (IFG), and the monkey PF is thought to loosely correspond to the human inferior parietal lobule (IPL). An electrical stimulation study provided more direct evidence for the existence of mirror neurons in humans. Specifically, electrical stimulation of the sensorimotor cortex of a 36-month-old child undergoing epilepsy surgery resulted in sensorimotor hand reactions, and this same area was found to be activated by simply observing hand movement (Fecteau et al., 2004). Furthermore, an fMRI adaptation of it revealed that areas of the IPL in humans activated both during the observation and during the execution of actions (Chong et al., 2008).

In addition to simulation, the MNS may provide us with the tools for action understanding. Indeed, human beings are adept not only at interpreting the actions of oneself and that of others but also at reasoning about such actions with causal explanations. This may involve identifying the agent and object in an event and inferring the intention behind a certain action. Although the neural basis of action understanding is complex, the involvement of the mirror neuron circuitry in this process is relatively well established (Lestou et al., 2008). The role of MNS in action understanding has been explained by differing accounts. Gallese (2001, 2003) argues that what mirror neurons code is the relationship, in motor terms, between the agent and the object of action, whereas Knoblich and Jordan (2002) propose that mirror neurons code the perceived effect an action exerts on an object. While the former leans toward a maximalist role of mirror neurons, the latter tends to be minimalistic (see Pacherie and Dokić, 2006). Despite these differing views of the functions of mirror neurons in action understanding, the important role MNS play in action understanding is undeniable. Thus, the MNS does not simply respond to visual stimuli alone; it can reflect the understanding of intentions through the understanding of a motor act (Kohler et al., 2002). It has also been found that the MNS is sensitive to the timing of observed actions as well. The MNS therefore codes a goal-directed action and the separate movements which lead to the achievement of that goal (Rizzolatti and Craighero, 2004). These separate movements seem collectively stored in a sort of ‘action bank’ in the observers’ mind. When viewing an action being performed, one must look into their ‘action bank’ for a match to the action and then apply their understanding of the intention behind the action (Sinigaglia and Sparaci, 2010).

Even though the significance of the MNS is still debated (see Hickok, 2009), many researchers believe that this system directly influences imitation (Iacoboni et al., 1999; Koski et al., 2002, 2003; Heiser et al., 2003), and appropriate understanding of actions appears to be an important prerequisite for imitation where one has to represent the model and then plan and execute the imitative action. Therefore, it has been hypothesized that individuals with autism who have deficits in imitation (Williams et al., 2004) may also have a malfunctioning MNS (Oberman and Ramachandran, 2007; Williams et al., 2001). Indeed, the “mirror neuron dysfunction hypothesis of autism” has received widespread attention, with quite a few studies suggesting atypicalities of the MNS in persons with autism contributing to autism symptomatology (for recent reviews, see Williams, 2008 or Bernier and Dawson, 2009).

Recent evidence of impairments in action understanding in persons with autism may be associated with atypical functioning of the MNS in this population. Understanding an action may involve two important aspects: (a) comprehending the motor action (what), and (b) inferring the intention behind the action (why). It is argued that a mirror mechanism may be involved in both these aspects (Rizzolatti et al., 2009). In a recent study, Boria et al. (2009) examined the why and what of action understanding in autism and found that children with autism had difficulty in figuring out the intention behind an action by relying on motor information.

In addition to the perceptual and intentional elements of action understanding, several other factors may also contribute to action understanding which may be altered in people with ASD. For instance, Zalla et al. (2010) found individuals with autism exhibiting greater numbers of temporal inversions when presented with an action and asked to predict the outcome. The authors suggested that these temporal inversions disrupt the ability of individuals with autism to both understand and predict the actions of others. In addition, previous literature has pointed to the need of familiarity, not just with the action, but also with the actor for appropriate action understanding in individuals with autism (Le Bel et al., 2009).

Action understanding also involves appropriate visual attention, a topic of debate in individuals with autism (see Section 6.1). According to Vivanti et al. (2008), individuals with autism exhibited similar patterns of visual attention when observing a demonstrator perform an action but showed reduced attention to the demonstrator’s face. Such difficulties in attention may be linked to dysfunction in appropriate affective coordination in ASD.

It has also been suggested that the way we understand actions of others is through our own first person ability for action and emotion. Perhaps this allows us to share the emotional aspects of other’s gestures and actions; the lack of this appropriate mirroring (through the MNS) may impair appropriate action understanding as well as affective coordination in autism (Sinigaglia and Sparaci, 2010). If others' actions are truly understood by connecting to our own actions and emotions, then a breakdown in the MNS and/or empathy would hinder appropriate action understanding. This is of particular relevance to autism as several studies have indicated that a dysfunction in the MNS in autism may be at the core of their difficulty to empathize (Buccino and Amore, 2008). Deficits in MNS may also have an impact on theory of mind (ToM) in autism. For instance, Pineda and Hecht (2009) found that the mirroring system was primarily involved with two aspects of ToM, emotion and person–object interaction, both of which are suggested in previous literature to be necessary for appropriate action understanding. Thus, there is substantial evidence to suggest that people with ASD may have difficulty with action understanding and that this difficulty may be related to MNS impairments.
3. Criticisms of the mirror neuron system hypothesis

Although there may be considerable evidence for MNS dysfunction in persons with autism coming from EEG (e.g., Bernier et al., 2007; Martineau et al., 2008; Oberman et al., 2005), functional MRI (e.g., Dapretto et al., 2006; Williams et al., 2006), and structural MRI (e.g., Hadjikhani et al., 2007) studies, some researchers have critiqued a few aspects of the MNS hypothesis of autism. Two of these critiques allude to the possibility of different neurological processes occurring at resting state in individuals with autism triggering false detection of underactivation in MNS (Dinstein et al., 2008), and the possible existence of multiple mirroring systems and not a specific focal deficit in the MNS in autism (Hamilton et al., 2007; Southgate and Hamilton, 2008). The first criticism cites abnormal activation patterns in resting state in autism (Kennedy et al., 2006), and since most fMRI studies compare activation patterns to a rest baseline, the seeming activation difference in MNS in autism may result as a consequence of abnormal resting state activation instead of differences in activation during task performance. For instance, if it appears that a person with autism activates less during the task, this may actually result from their greater resting state activation (Dinstein et al., 2008). The second critique (Hamilton et al., 2007) suggests the existence of multiple mirroring systems. According to this view, a wide range of distinct processes need to function together in order for imitation to occur. In addition, different types of imitation are likely to require the active and coordinated functioning of varying neural systems. Due to the multiple functions involved in imitation, the imitation deficit seen in autism is likely too large to be explained by a specific deficit within the MNS and may involve multiple mirroring systems (Southgate and Hamilton, 2008). Even proponents of the MNS dysfunction hypothesis of autism admit that a deficit in the MNS, although able to explain some social-cognitive dysfunctions, as of yet does not explain other key symptoms of autism, such as repetitive behaviors (Oberman and Ramachandran, 2007).

Despite these critiques, the MNS hypothesis of autism is promising and seems to explain several symptoms of autism. The focus of this review will be on examining one such symptom, a deficit in imitation, and linking it with not only the connections within the MNS, but also how the MNS is connected to other brain regions. A key question we want to ask is whether the core deficit in imitation in autism is global, encompassing all aspects of imitation, or whether certain components of imitation skill show greater levels of impairment relative to others. Most previous studies have examined imitation as a unitary measure without much emphasis on the specific component processes involved. However, some available evidence suggests that the imitation deficit in autism may not be unitary. For example, Williams et al. (2004), in their comprehensive review of the imitation deficits in autism, concluded that individuals with autism exhibit greatest difficulties while imitating non-meaningful or novel actions. This specific deficit in a subset of imitation tasks suggests that at a neural level, persons with autism may not have a global MNS impairment, but they may have impairments within certain nodes of the MNS or in their anatomical connectivity and functional synchronization.

As an additional caveat to the MNS theory of autism, Southgate and Hamilton (2008) argue that the MNS explanation of autism is premature and does not have as much explanatory power as the cognitive models of imitation in autism, such as models that take into account executive function, attention, and theory-of-mind (ToM) difficulties. While the cognitive models of autism do assist in explaining many of the imitation impairments in autism, we think that a combination of the cognitive models and the MNS model may have more explanatory power than either of these models in isolation. This is especially true when we examine the overlap between the regions that are part of the MNS and the regions associated with attention, executive function, self-other understanding, and theory-of-mind (ToM).

Therefore, the primary aim of this paper is to characterize the nature of cognitive and neural processes that underlie the impairments in imitation and related social abnormalities in autism. Our approach will be from the perspective of the cortical underconnectivity theory of autism (Just et al., 2004; Kana et al., 2006). We believe that functional underconnectivity within the MNS (between core centers: IPL and IFG), and between the MNS and other associated regions, such as the STS and cerebellum in persons with ASD can best explain the uneven profile of imitation difficulties in this population. Furthermore, an underconnected MNS model may also help explain how cognitive impairments in this population may modulate the function of the MNS under conditions where (1) familiarity with the person-to-be-imitated, (2) the relevance and purpose of the action-to-be-imitated, and (3) the salience of the action-to-be-imitated play significant roles.

4. Components of the MNS

Recent research suggests that different regions of the MNS are thought to serve slightly different functions. Specifically, Hamilton and Grafton’s (2007) and Hamilton’s (2008) EP-M Model suggested two separate pathways within the MNS: an emulation pathway (for goal-oriented or explicit imitation) and a mimicry pathway (for automatic or reflex-like imitation). In the emulation pathway, while connections between the STS and IPL code for the goals of an action, connections between the STS and IFG code for action planning. In the mimicry pathway, the STS and IFG connect directly, bypassing the IPL, and Hamilton (2008) suggests that this pathway is the one most likely impaired in persons with autism. Although there is little anatomical evidence for a direct connection between the MTG and F5 (supposed homologues of the human STS and IFG, respectively) in the macaque monkey, diffusion tensor imaging (DTI) studies have provided evidence for anatomical connections (through arcuate fasciculus) between the STS and IFG (i.e., Catani and Ffytche, 2005; Friederici, 2009; Rilling et al., 2008) (Fig. 1).

The distinction between emulation and mimicry capabilities within the MNS, predicted by Hamilton’s (2008) EP-M model, coincides with the discovery of two different types of mirror neurons in monkeys: (1) broadly congruent mirror neurons that code for similar goals, and (2) strictly congruent mirror neurons that code for...
specific motor acts (Gallese et al., 1996). Broadly congruent mirror neurons appear to be twice as prevalent in the monkey area F5 compared to strictly congruent mirror neurons, but both types of neurons are thought to respond to any given action, providing flexibility in the interpretation and motor translation of that action (Keysers and Gazzola, 2009). In humans, enacting complementary actions has been found to generate activation in the IFG and IPL of the MNS more than enacting imitative actions (Newmann-Norlund et al., 2007), suggesting that similar to the monkey MNS, the human MNS may consist more of broadly congruent than strictly congruent mirror neurons. However, it seems unclear whether these different types of mirror neurons would align themselves along the pathways indicated by Hamilton’s (2008) EP-M model. It seems likely that mimicry would rely primarily on the strictly congruent mirror neurons, whereas emulation would rely primarily on the broadly congruent mirror neurons or a mixture of broadly and strictly congruent mirror neurons.

Additionally, the EP-M model fits nicely with empirical data suggesting the role of IPL in the interpretation of goals (e.g., Tunik et al., 2005; Buxbaum et al., 2005), and the role of the IFG in the interpretation of more specific action properties (e.g., Pobric and Hamilton, 2006; Urgesi et al., 2007). Models of action understanding also suggest the importance of the IPL as a site for integration of visual information from both dorsal and ventral visual streams with motor information (Fogassi and Luppino, 2005). As such, the IPL is thought to code motor acts in varying ways depending upon how they are performed.

An additional advantage of the EP-M model is that it incorporates not only feed forward connections between the core (IPL, IFG) and the contributing (STS) areas of the MNS (i.e., STS → IPL → IFG), but also bi-directional connections between these regions (i.e., STS ↔ IPL ↔ IFG). Kilner et al. (2007) argue that these bidirectional connections allow us to make online adaptation to our motor scripts when the goal of the motor act is unclear (For example, when a person across the street is waving her hand, it may be unclear whether she is waving hello to you or trying to stop a car). In the action understanding literature, a recent study (Lestou et al., 2008) also suggests bidirectional connections among the STS, IPL, and ventral premotor areas. Lestou and colleagues found that visual areas (including V1, MT and V5), parietal areas (SPL and IPL), the STS, and the ventral pre-motor regions were all involved in different aspects of the action-understanding process. However, the links between these circuits and the MNS suggest that action understanding may occur through an inverse model (where visual representations of observed actions in the STS are translated into motor plans in the parietal and pre-motor areas) and then a forward model (where motor plans in the ventral pre-motor areas are changed to predict actions in the STS through the parietal cortex).

Future research will need to further investigate how the inverse and forward neural pathways interact. However, it is striking that both Hamilton’s (2008) EP-M model of imitation and Lestou et al.’s forward and inverse models of action understanding include bi-directional pathways among MNS and MNS-related brain regions.

One key question surrounding Hamilton’s (2008) EP-M model is whether there are moderating factors that may affect the connectivity and direction of these bi-directional pathways. Hamilton suggested that difficulties associated with ToM in persons with autism may moderate the activation of the direct pathway between the STS and IFG that codes for mimicry. This is then thought to make mimicry appear abnormal in persons with autism, with it either appearing hyperactive (e.g., echolalia) or hypoactive (i.e., lack of social mirroring). Indeed, some studies have indicated that mimicry may be difficult for persons with autism. For example, Senju et al. (2007) found that when presented with videos of people yawning, children with autism did not exhibit as much automatic yawn contagion as typically developing children. Similarly, McIntosh et al. (2006) found that participants with autism were able to voluntarily mimic pictures of faces as well as typically developing controls, but they did not show automatic face mimicry (measured by electromyography [EMG]). While these results suggest that mimicry, or automatic imitation, may be reduced in persons with autism, there is also evidence of controlled imitation being impaired autism. One explanation for this comes from Oberman et al. (2009), who suggests that the IPL may serve as a social filter in the emulation pathway. In typically developing individuals, this filter would affect the emulation pathway by sending only socially relevant information to the IFG. However, this filter may malfunction in autism, over-filtering out socially relevant information, which then never allows this information to be processed at a higher level by the IFG.

Besides Hamilton’s (2008) EP-M model and Oberman et al.’s (2009) social filter model, it seems likely that underconnectivity (especially frontal-posterior underconnectivity) in persons with autism would compromise the information transfer within the MNS as well as between MNS and other brain areas in autism. Consequently, in the following section on a review of imitation impairments in autism, we include possible behavioral outcomes of disruptions in connectivity between the individual parts of the MNS and also behavioral outcomes of disruption in connectivity between the MNS and other brain regions.

5. Imitation impairments in autism

Children with autism have been found to have problems in both symbolic and non-symbolic imitative behaviors, in imitating the use of objects, imitating facial gestures, and in vocal imitation (Rogers, 1999). An imitation deficit in autism is a relatively firm finding that has been replicated in over 21 different studies (Williams et al., 2001), and such deficits are related to many of the social impairments in autism, such as face-processing (Hadjikhani et al., 2007), theory-of-mind (Williams et al., 2006), and joint attention (Villalobos et al., 2005). Despite these findings, the imitation deficit in autism may not be unitary with some types of imitation being more difficult than others. For example, Rogers et al. (1996) found improved performance in meaningful imitation compared to meaningless imitation in adolescents with autism. Similarly, Hamilton et al. (2007) found intact goal-state imitation and motor planning in children with autism. Indeed, because imitation ability may not be unitary, the types of imitation that are impaired may be explained by the MNS model we previously discussed. In using Hamilton’s (2008) EP-M model as the base, we can examine the MNS as a network consisting of the IPL and IFG. Then, we can contemplate what types of behavioral difficulties in imitation would be expected if there were a problem in communication between these two main areas of the system or between other brain regions that feed into these MNS regions. For example, if there were miscommunications between the STS and the IFG (in the mimicry pathway), then we might expect difficulties regulating automatic imitation. As previously mentioned, evidence from previous research does provide support for this hypothesis (e.g. Senju et al., 2007). If, however, the problem existed in the connections between the IPL and IFG, the difficulty will be manifested in the planning aspects of imitation, and in determining the intention without exhibiting difficulty in goal identification. Several studies support this idea. In addition to the studies previously discussed which conclude that children with autism are better at imitating actions with clear goals, a study by D’Entremont and Yazbek (2007) found that children with autism, unlike typically developing children and children with other developmental delays, were not more likely to imitate actions that were intentional over those that were accidental. This might be due to their limited ability to appreciate the intent of the person performing the action.
Bennetto (unpublished dissertation) examined imitation in autism focusing on its component skills: basic motor functioning, body schema, dynamic spatiotemporal representation, memory, and motor execution of non-meaningful hand and arm gestures. Although she found an overall impairment in imitation in autism, what was more interesting was the result pertaining to the component skills of imitation. Participants with autism performed poorly in the motor functioning and action planning aspects of imitation, but not on the spatiotemporal representation, body schema, and memory compared to typically developing individuals. The group differences between autism and control participants in motor functioning appeared to be due to difficulty with kinesthesia (i.e., the ability to reproduce given limb postures in a precise manner) and difficulty with apraxia (i.e., the ability to plan, execute, and perform skilled gestures). In fact, previous studies showed that persons with autism had difficulties with both kinesthesia (i.e., proprioception; Haswell et al., 2009) and dyspraxia (Dziuk et al., 2007). Visuo-kineesthetic integration is thought to occur between the left cerebellum and the right inferior parietal lobule (Hagura et al., 2009a, b). While IPL is part of the core MNS, cerebellum plays active role in visuomotor tasks that require online combination of visual and kinesthetic information (Liu et al., 1999; Imamizu et al., 2000). Additionally, difficulties with apraxia (i.e., dyspraxia or apraxia) have been associated with lesions of the IPL or disrupted connections between the IPL and the premotor cortex (Buxbaum et al., 2007; Sirigu et al., 1996; Tanaka et al., 1996). Subsequently, underconnectivity between the cerebellum and the IPL or between the IPL and other regions of the MNS in persons with autism could contribute to the specific difficulty in kinesthesia and dyspraxia during imitation. Thus, weaker connectivity may be seen not only within the MNS core, but also between the MNS and other contributing brain areas, such as the cerebellum.

A problem in communication between the STS and IPL in autism would entail difficulty in imitation tasks that involve goal-oriented actions. However, a detailed review of imitation difficulties in autism suggested that they often exhibit the greatest difficulty while imitating meaningless gestures rather than while imitating meaningful gestures or gestures that involve objects (Williams et al., 2004). Similarly, Hobson and Hobson (2008) found that children with autism had most difficulty when imitating the style of an action, especially when the style was not necessary for achieving the action’s goal. These results may suggest that persons with autism are better at imitation when the to-be-imitated action is goal-oriented with a clear end state (as meaningless gestures and the “style” of a gesture are determined more by the process of the imitative act rather than the end state or goal of the gesture). Furthermore, it is unlikely that the imitation of meaningful gestures being intact is due to better memory for meaningful compared to meaningless stimuli, as persons with autism do not show improved recall of meaning-encoded versus perceptually encoded words (Toichi and Kamio, 2002). This result suggests that persons with autism are not imitating meaningful gestures better just because they can more readily recall them. However, because actions in the real world are typically goal-oriented, the familiarity of these actions may make imitating them easier for persons with autism. Thus, these results suggest that imitation may be intact in autism when the goal state is salient, or, conversely, when the action is familiar.

When the goal-state of an action is evident, imitation may be performed more easily and accurately. For example, imitating an action after watching someone perform it involves knowing the degree to which the imitation should occur. Specifically, would successful imitation be defined as imitating everything, down to the automatic eye blinks of the other individual? Or would it be defined as merely achieving the end state through different means? Using contextual cues and background knowledge, one might choose the most appropriate degree of imitation. However, knowing which aspects of an action to attend to and then to emulate may pose difficulty for persons with autism who have been shown to demonstrate difficulties with certain abilities such as attention and ToM. In summary, behavioral research suggests that there may be decreased functional connectivity between certain regions of the MNS but that the connections between other regions may remain unaffected. The bulk of the imitation literature seems to point to difficulties arising within the connections between the STS and IFG (which makes up the mimicry pathway of Hamilton’s (2008) EP-M model) and between the IPL and IFG.

6. Cognitive processes related to imitation impairments

A pronounced underconnectivity between IFG and IPL within the MNS and that between other areas (such as STS, cerebellum) and the MNS may suggest not only difficulties in imitation but also difficulties in other cognitive processes that underlie similar regions and connections. Thus, the following sections describe two cognitive processes that may draw resources from the IFG, IPL, cerebellum, and STS and are thought to be impaired in persons with autism.

6.1. Attention

Effective imitation also requires a person to attend to and encode the relevant motor attributes that need to be emulated. For example, if you show me how to reel in a fish, I will be most successful at imitating you when I focus on your arm and hand movements, rather than on your face. Indeed, persons with autism have exhibited atypicalities in attention (for a review, see Travers et al., in press) that might interfere with their imitation ability and may affect the way the MNS codes this function. Reduced attention to social stimuli (Dawson et al., 1998, 2004; Osterling and Dawson, 1994), and increased attention to local features (e.g., “weak central coherence,” Frith, 1989, 2003; Frith and Happé, 1994; Happé and Frith, 2006; or “enhanced perceptual functioning,” Mottron et al., 2006) in autism can affect any cognitive or social function. Because of the attention to specific details at the expense of a gestalt, children with autism may not perceive an action to be imitated (modeled) in its global form, which might negatively impact their ability to imitate that action.

The brain areas associated with attentional reorienting overlap significantly with the brain areas commonly associated with the MNS. For instance, while both IFG (Corbetta and Shulman, 2002) and IPL (for a review see Singh-Curry and Husain, 2009) have been found to be involved in attentional reorienting, IFG has also been associated with inhibiting irrelevant information during imitation (Chong et al., 2008). Similarly, the posterior STS has been found to be involved in processing eye gaze (Pelphrey et al., 2005) and in inferring the direction of attention and the intentions of another person (Materna et al., 2008). Although cerebellum has traditionally been associated with motor control, of late, its role in drawing attentional resources has been emphasized by some studies (Townsend et al., 2000) with cerebellar abnormality contributing to attentional problems in autism (Allen and Courchesne, 2001). These results suggest that important attention networks heavily overlap with the cortical areas that are found to be associated with the MNS. Although it is unlikely that a disruption in the attentional network would be the sole factor causing imitation difficulties in persons with autism, this overlap theoretically could contribute to a particular pattern of imitation impairments seen in persons with autism. One possibility is that persons with autism may have the knowledge of what is most important to attend to in a familiar context or when the goal of the action is
salient. This knowledge, in turn, could improve imitation ability in these familiar contexts but hinder imitation ability in unfamiliar contexts. Alternatively, because attention is heavily intertwined with motor activity (including eye saccades and full body orientations), if the MNS is not functioning typically in persons with autism it would be likely that the attentional networks might also be affected. However, the purpose of this section is not to say that impaired attention is a core deficit in autism or that mirror neurons are crucially implicated in attention, but to suggest that the problems and peculiarities in attention seen in autism might be related to other skills, such as learning to imitate.

6.2. Self-other mapping

Imitation requires the ability to internally simulate the observed actions and internal states of others (Meltzoff and Moore, 1995). By creating an internal representation of the action, one can build a cognitive bridge between self and other necessary to develop an understanding of the other person’s thoughts and feelings (Gallese, 2003). Difficulty in such self-other mapping has been proposed as an important and most likely explanation of imitation deficits in autism (Rogers and Pennington, 1991; Williams et al., 2004). The most striking evidence derives from studies where people with autism produced “reversal errors” during imitation; e.g., they reproduced gestures with palm reversed or reversed direction of movement, seeming to indicate an inability to adopt the perspective of the person being imitated (Ohta, 1987; Brown, 1996; Hobson and Lee, 1999; White and Brown, 1999). In the context of the MNS hypothesis, one argument is that a dysfunctional MNS results in impairments in drawing self-other motor correspondences (Williams et al., 2001). Since self-other mapping involves perspective-taking, the MNS and its connections with other regions, such as the posterior STS may be critical in imitation. Therefore a deficit in perspective-taking may also moderate the connections between the posterior STS and IFG. In summary, attention and self-other mapping processes may involve the coordination of MNS and related regions, and the underconnectivity model of MNS impairments in persons with autism may be best understood by taking into account these cognitive impairments. The overlap among these cognitive and neural processes may help explain studies that have found intact MNS function in persons with autism under certain conditions. Once again, this is not to say that mirror neurons are crucially implicated in self-other mapping. However, self-other mapping and attention may be moderating or mediating factors that could partially account for the times when imitation and MNS function occur to be typically functioning in persons with autism.

7. Conditions of typical MNS function in persons with autism

Consistent with the idea of specific impairment in certain connections between areas of the MNS, but not others, several studies have found that persons with autism can exhibit typical MNS activation under certain conditions. For example, Oberman et al. (2008) examined how EEG changes during observed activation are modulated based on the degree to which the observer can identify with an active person on the screen. Since it has also been suggested that the MNS is involved in the ability of an individual to relate to others (as described earlier), a disturbed mirror neuron system (as has been hypothesized in autism) would be expected to result in an impaired ability to relate to people on the screen. Oberman et al. (2008) tested this idea by examining the level of mu suppression in scenes of actions involving familiar versus unfamiliar actors. In the unfamiliar condition, the actor was a stranger whereas in the familiar condition the actor was either the participant’s guardian or sibling. The only part of the actor shown was their hand. Even with only the actor’s hand visible, they found that the degree of mu suppression was related to the degree of familiarity the individual had with the actor. This suggests that the mu suppression occurs during observed actions within individuals with autism, but only when the individual is familiar with the person who is performing the action. This finding is important since, in addition to replicating previous findings, it suggests that the MNS may be sensitive to the level of familiarity with the actor and with the modeled action. This sensitivity to familiarity mirrors new findings that individuals with autism selectively activate fusiform gyrus when they view pictures of familiar faces (Pierce and Redcay, 2008). Past research has in fact indicated that the activation in the IFG and in the STS was highly correlated with the activation in other areas of the extended face processing network, suggesting that face processing performed by the fusiform may be modulated by the MNS. For instance, Hadjikhani et al. (2007) argue that the face recognition deficits in autism cannot be reduced to problems in the fusiform alone, and may themselves actually be the result of dysfunction within the MNS instead. The fact that increased familiarity with an actor can facilitate typical MNS activation further suggests that the MNS impairment in persons with autism may not be global. Familiarity has been found to be linked to increased connectivity among corresponding brain regions, possibly as a result of increased exposure and developing expertise (i.e., Bhattacharya and Petsche, 2005). Subsequently, familiarity effects in the MNS of persons with autism may reflect differing amounts of connectivity among the specific centers within the MNS, and among MNS and other regions that feed into it. Future research should examine MNS function and its connectivity in persons with autism in response to stimuli that continuously vary in their familiarity to the participant.

Both familiarity and self-other effects (described earlier) suggest a possible impairment in the coordinated functioning of the MNS system or in brain areas that feed into the MNS. However, more direct evidence of underconnectivity between areas associated with mirror neurons comes from a study by Villalobos et al. (2005). This study examined the dorsal stream connectivity between the superior parietal areas and the inferior frontal area 44 (IFG). The study (involving a visually initiated button press task) examined the functional connectivity between these areas by measuring the cross-correlation of the BOLD signal during visuomotor coordination. They found that while there was no significant deficit in connectivity between area 17 and the superior parietal area, there was underconnectivity between the primary visual area and the inferior frontal area 44. More evidence comes from an MEG (Magnetoencephalography) study by Nishitani et al. (2004) which found that activation within certain areas of the MNS in individuals with autism was delayed by an average of 45–60 ms compared to that of controls while imitating static pictures of lip forms. It has been suggested that one way in which the mirror neurons could be impaired in individuals with autism is in their ability to form action chains. In typically developing individuals, activation in response to viewing an action varies according to the goal of the action. This might be because “action-constrained” mirror neurons respond to the initial motor action by firing a specific action chain based on the initial motor movement, which allows the individual observing the action to have an internal copy of the action before it occurs allowing them to gain an understanding of others’ intentions. The ability to create action chains has been found (using electromyographic recordings) to be atypical in individuals with autism, such that children with ASD did not demonstrate the typical anticipatory muscle activation for the last step of the motor sequence either when observing the action chains of others or while producing the action chains themselves (Cattaneo et al., 2007). This suggests a difficulty in integrating individual motor acts into an action chain.
Thus, the pattern of strengths and weaknesses seen in individuals with autism when performing imitation or other cognitive tasks points to problems associated with the coordinated functioning of the system. Evidence for this comes from both behavioral and neuroimaging studies which further affirm that the difficulties in the MNS network may lie between the relatively longer distance communication between IPL and IFG, or between other regions, such as STS and IFG (but not between the STS and IPL).

8. Possible anatomical connections

The advent of diffusion tensor imaging (DTI), a technique to measure water diffusion in the brain, has provided a great avenue for examining the structural integrity of white matter more thoroughly (Engelbrecht et al., 2002; Neil et al., 2002). Of late, there have been several DTI studies in autism, examining mainly the fractional anisotropy (FA) which is an index of the degree of anisotropy of the diffusion of water in the brain. For instance, reduced FA values have been reported in: the ventromedial prefrontal cortex, ACC, TPJ, STS and amygdala in autism (Barnea-Goraly et al., 2004), in the corpus callosum (Alexander et al., 2007), in the genu and splenium of corpus callosum, and in the internal capsule (Keller et al., 2007), in the superior temporal gyrus (Lee et al., 2007), in the cerebellar feedback projections (Catani et al., 2008), in the right posterior limb of internal capsule (Cheng et al., 2010), and in the ACC (Thakkar et al., 2008). There were also reports of increased FA in autism, especially in the frontal lobe (Ben Bashat et al., 2007; Cheng et al., 2010), and an imbalance of FA in the frontal lobe as indicated by lower FA for short range fibers and higher FA for long range association fibers (Sundaram et al., 2008). Overall, these studies suggest atypical FA in several different areas of the brain in autism which might have an impact on brain function. Perhaps the closest match of white matter fibers connecting the MNS core may be the arcuate fasciculus (AF). Tractography studies suggest that the AF is a complex fiber bundle with a long direct segment connecting Wernicke’s area (posterior STS) with Broca’s area (LIFG), and an indirect segment that connects these two areas to the inferior parietal lobule (Catani and Ffytche, 2005). A recent diffusion tensor imaging (DTI) study found less lateralized mean diffusivity and fractional anisotropy in the AF in high-functioning adolescents with autism (Fletcher et al., 2010). Although the direct consequence of this may be on language, it might have an impact on the information transfer in the MNS. Another finding pertains to the altered cerebellar feedback projections in Asperger Syndrome (Catani et al., 2008) which may affect the cortex from receiving cerebellar feedback inputs. This may be relevant to the MNS since the communication between cerebellum and IPL may be critical in visuo-kineesthetic integration (Hagura et al., 2009a,b) which in turn is vital in imitation.

9. Conclusions and future directions

It is evident that the mirror neuron system is multidimensional in nature and hence the functions subserved by this system may also be multidimensional. In this review, we examined a complex cognitive and social function, imitation, and the neural circuitry assumed to underlie it, the MNS, in autism at a systems level using a compelling theoretical account, the cortical underconnectivity theory. An important take home message from this review is that the impairments in imitation in autism have to be examined carefully and in detail since the problems in imitation in autism may lie in the malfunctioning of its several subcomponents or in their lack of integrity. Evidence shows that several factors, such as attention, familiarity, relevance, self-other matching, and goal identification, play important roles in accomplishing an imitative act. At the neural level, the coordinated functioning of the anterior and the posterior nodes of the mirror neuron system may be critical in subserving these functions. A comprehensive analysis of imitation at this level may have significant clinical implications for people with autism. For example, if deficits in attention modulation are related to the MNS deficits seen in autism, then it follows that modulation of attention (which is often the basis of interventions such as ABA and TEACCH) may actually lead to improvement in MNS functioning, and hence pave the way for improved social skills. Future studies should focus on examining the functional as well as anatomical connections underlying MNS and other systems, and in linking DTI findings with fMRI evidence in autism.

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