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## **Imitation, mirror neurons and autism**

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### **ABSTRACT**

Various deficits in the cognitive functioning of people with autism have been documented in recent years but these provide only partial explanations for the condition. We focus instead on an imitative disturbance involving difficulties both in copying actions and in inhibiting more stereotyped mimicking, such as echolalia. A candidate for the neural basis of this disturbance may be found in a recently discovered class of neurons in frontal cortex, 'mirror neurons' (MNs). These neurons show activity in relation both to specific actions performed by self and matching actions performed by others, providing a potential bridge between minds. MN systems exist in primates without imitative and 'theory of mind' abilities and we suggest that in order for them to have become utilized to perform social cognitive functions, sophisticated cortical neuronal systems have evolved in which MNs function as key elements. Early developmental failures of MN systems are likely to result in a consequent cascade of developmental impairments characterised by the clinical syndrome of autism.

**Key words:** **imitation, mirror neurons, autism, 'theory of mind'**

### **Introduction: the basis of autism**

The autistic spectrum disorders are increasingly being recognised as an important cause of social disability<sup>1</sup> and have been the focus of a flurry of research in the last decade<sup>2-5</sup>. Here we suggest that juxtaposing some of these psychological findings with recent discoveries in neurobiology offers the prospect of a new and potentially powerful

model of both early social functioning and the disorders in it that are associated with autism.

The autistic spectrum disorders are characterised by impairments in social interaction, imaginative ability and repetitive and restricted patterns of behaviour. In those children with autism as opposed to Asperger's syndrome, the disorder has an onset before the age of 3 years and is associated with delayed and abnormal language development<sup>6-8</sup>. The condition is heterogeneous, both with respect to cause and clinical picture. It may be associated with abnormalities such as epilepsy, mental handicap and various brain pathologies. There is also evidence that autism is part of a broader phenotype<sup>9</sup> and sub-syndromal symptoms are often found in population surveys<sup>1</sup>. As such it may be best conceptualised as a dimensional rather than a categorical disorder<sup>10</sup>. The distinction between autism and Asperger's syndrome is also subject to diverse opinions. Happe<sup>11</sup> concludes that for most researchers "Asperger's syndrome is a label for high-functioning autistic individuals". This distinction was supported recently in a cluster analysis by Prior et al<sup>12</sup>. Perhaps due to this diverse and complex clinical picture, no common underlying mechanism has yet been identified. It is clear, however, that autism is a developmental disorder characterised by a cascade of specific impairments over the course of development.

Baron-Cohen et al<sup>13</sup> demonstrated that children with autism typically had special difficulties in understanding the beliefs of others and suggested that they lacked the 'theory of mind' ('ToM') necessary to pass such tests. This claim has since been supported by a wealth of experimental investigations and has led some to argue that at the

root of autism is a ToM deficit or delay<sup>14-16</sup>. However, a metarepresentational ToM deficit seems unsatisfactory as a primary explanation for autism. First, ToM as tested by Baron-Cohen et al<sup>13</sup> does not typically become at all robust in normal children until after the fourth year, yet autistic disorders are manifested earlier. This has led researchers attracted to ToM explanations of autism to a search for 'precursors' to ToM, which might be apparent in early autistic disorders. Candidates for such precursors include pretend play<sup>17</sup> and a capacity to engage in shared attention with another individual<sup>18</sup>. Second, clinicians have argued that early social deficits are often broader in scope than implied by the focus on ToM<sup>19</sup>; Hobson<sup>20</sup> for example, has argued that the primary deficit is more aptly described as socio-affective, characterised by a lack of empathic and emotional engagement with others. The third and final problem is that autism is often characterised by other social and non-social problems that appear ill-accommodated by a primary ToM deficit. These include repetitive and stereotyped behaviour (including copied behaviours), obsessive desire for sameness, delayed and deviant language development, (including echolalia) and difficulties in perceiving or planning at high-levels of organisation ('executive function'<sup>4</sup>). The challenge in understanding autism, then, is to identify dysfunction in underlying mechanisms that can account for a wider range of symptoms than the ToM or executive function theories alone, thus explaining clustering of symptoms in the autistic spectrum disorders. It does not necessarily include accounting for those characteristics which are not specific to the condition such as global developmental delay, aggression or sleep disturbance.

### **The role of early imitation**

The possibility that deficits in imitation might be particularly intimately connected with the earliest developmental stages of autism was first set out systematically by Rogers and Pennington<sup>21</sup>. According to these authors, imitation might fill at least two of the three gaps left by the ToM explanation noted above: first, imitation has characteristics suggesting that the mechanisms underlying it could be precursors (perhaps the first that can be identified in infancy) to full ToM; and second, imitation may also be fundamental to the other, broader kinds of social deficits seen in autism. The relationship between imitation and the third group of (largely non-social) deficits listed above is one we shall discuss once other parts of our model have been explained. Rogers and Pennington<sup>21</sup> collated existing empirical evidence of imitation deficits in autism, which we discuss in the following section. First, however, some key theoretical bases for a link between imitation mechanisms and later-developing ToM need to be recognised.

Imitation and the attribution of mental states bear some fundamental resemblances<sup>22-23</sup>. Both involve translating from the perspective of another individual to oneself. Thus in accurately reading the belief of another, one essentially copies the belief into one's own brain, creating a 'second-order' representation of the other's primary representation of the world (and, of course, not confusing it with one's own beliefs, at least in the normal case). Conversely in imitating, one must convert an action plan originating from the other's perspective into one's own. A more specific linkage between imitation and ToM is implied by the fact that one of the two principal models of how ToM operates is designated the 'simulation' theory<sup>24</sup>. Its rival is the 'theory theory',

which sees the child acting somewhat like a young scientist, observing patterns of behaviour in others, and developing theories about mental states to explain and predict them. The simulation theory instead proposes that children come to read minds by 'putting themselves in the other's shoes', and using their own minds to simulate the mental processes that are likely to be operating in the other. 'Acting as if you are the other' - simulation - is thus at the covert, mental level akin to what is involved at the overt level in imitation. Current views include the possibility that both 'simulation theory' and 'theory-theory' processes are at work in the human case<sup>25</sup>.

Meltzoff and Gopnik<sup>26</sup> reviewed evidence for imitation in the earliest phase of infancy and proposed that this could provide a key starting-state for the development of ToM. The nub of their hypothesis is that the new-born's capacity to translate between the seen behaviour of others and what it is like to perform that same behaviour offers a crucial basis for recognising the linkage between mental states and actions.

There are thus substantial theoretical reasons for considering imitation as a prime candidate for the building of a ToM. Rogers and Pennington's theory<sup>21</sup> was that at the root of autism is "impaired formation/co-ordination of specific self-other representations", manifest first in impaired imitation, followed by a cascade of impairments in emotion-sharing, joint attention and pretend play (thus including the broad range of social deficits), and ToM. What, then, is the evidence for imitation being affected in autism?

## Imitation in autism

Evidence for an imitative deficit in autism has been reviewed elsewhere<sup>21,27-29</sup>. None of these reviews is comprehensive but together they cite 21 experimental studies of the imitative competence of individuals with autism. The studies have been heterogeneous with respect to the mental ages tested, the types of control groups used and the imitation tests themselves, but only two studies did not find an imitative deficit in the autistic samples and then possibly because of the simplicity of the tasks, leading to ceiling effects. Smith and Bryson<sup>27</sup> conclude that the literature shows a 'consistent finding that people with autism do not readily imitate the actions of others'. Furthermore it is worth noting the magnitude of the imitative deficit. For instance, Rogers et al<sup>30</sup> detected group differences of approximately 1.5 standard deviations between the autistic and control group means. More recently Hobson and Lee<sup>31</sup> found that only 1 out of 16 (6%) subjects imitated the style of one of their tasks, compared to 12 out of 16 (75%) controls. A number of studies have detected significant group differences with just 10 subjects per group. The magnitude of this deficit then can be at least as great if not greater than the 'theory of mind' deficit. Rogers<sup>28</sup> additionally notes the difficulties faced by carers in intensively teaching imitation to young children with autism. Deficits in the imitation of 'symbolic' elements (such as pantomiming brushing one's teeth with a non-existent toothbrush) might be expected in view of the diagnostic criteria; thus of special interest are those concerning basic body movements or gestures. These were first demonstrated by DeMeyer et al<sup>32</sup> and have since been replicated in at least nine further studies<sup>27-29</sup>. Rogers<sup>28</sup> concludes that 'every methodologically rigorous study so far

published has found an autism-specific deficit in motor imitation'. The conclusion that the imitative deficit may be operating at such a fundamental level is important to our synthesis with neurobiological findings discussed further below.

The reason for difficulties in imitation associated with autism remains unclear but some clues may come from an examination of the type of imitative deficit present. Firstly, imitation of meaningless gestures would appear to be affected more than imitations of actions with objects<sup>30</sup>. Perhaps the use of objects in some tests may offer a 'prop', helping to shape a matching response; by contrast, difficulties in copying raw gestures underlines the more basic nature of the imitative deficit referred to earlier<sup>33</sup>. Secondly, when children with autism were asked to imitate an unconventional action with a common object (such as drinking from a teapot) they were more likely to make errors<sup>27</sup>. This again provides evidence for an imitative deficit more fundamental than that expected on the basis of other known impairments. Thirdly are reversal errors<sup>27,29</sup>; for example, in 'copying' the action of holding the hands up palm away, grasping the thumb of one hand with the other hand, autistic subjects tended to hold their palm towards themselves, re-creating the hand view they had seen (sometimes also failing to grasp the thumb) instead of translating the perspective the other had seen<sup>25</sup>. Finally there are greater group differences with respect to sequences of actions than when single actions alone are being imitated<sup>30</sup>. Together, these kinds of errors suggest that deficits may be occurring in the basic ability to map actions of others onto an imitative match by oneself<sup>29</sup> especially when such actions are complex.

Finally, there is a curious aspect of imitation-like phenomena in relation to autism, that concerns the well-known repetitive and stereotyped behaviours and speech that may occur. These may be copied from others, including words and phrases (echolalia) and sometimes actions, that are mimicked without regard to their normal goals and meanings. At first sight these phenomena seem contradictory to the notion of an imitative deficit, but they may instead offer clues to the underlying neural dysfunction. We will discuss this in a later section, in integration with the findings on neurobiology to which we now turn.

### **Neurobiology of imitation**

Patients with left frontal lobe lesions may show imitative dyspraxia<sup>33,34</sup>. These patients are unable to repeat actions performed by others, despite demonstrating adequate motor control of their limbs. Furthermore, they are unable to replicate such gestures on a manikin<sup>35</sup>. This is consistent with the idea that imitation may normally rely on representation of action at a 'supramodal' level<sup>36</sup>, which is unavailable to these patients; the same lesion site will accordingly disrupt the replication of a gesture whether on the self or on another body.

Work at the neuronal level in non-human primates has started to indicate the pathways by which representation of such actions may be built up. A number of different types of specialised neuron have been identified in the superior temporal sulcus (STS) of monkeys that are dedicated to visual processing of information about the actions of others. Particular populations of cells code the posture or the movements of the face, limbs or whole body<sup>37-41</sup>. Other classes of neurons appear to code movements as goal-

directed actions and are sensitive to hand and body movements relative to objects or goals of the movements (e.g. reaching for, manipulating or tearing an object)<sup>42-45</sup>

Of special relevance to our model is a subset of such action-coding neurons identified in the prefrontal cortex (area F5) in monkeys<sup>46,47</sup>. Such neurons will fire when the monkey performs a specific action, such as a precision grip, but also when an equivalent action (a precision grip, in this example) is performed by an individual the monkey is watching. These have been called 'mirror neurons' (MNs)<sup>47</sup>. Their potential relevance to imitation is signalled by another label: 'monkey see, monkey do' neurons<sup>48</sup>. F5 cell activity, however, does not automatically lead to motor responses and action performance, otherwise seeing actions performed would lead to obligatory copying (echopraxia). The execution of actions when F5 cells are activated by the sight of actions of others, may be inhibited by mechanisms operating elsewhere in the motor pathway<sup>49</sup> and perhaps involving orbitofrontal cortex<sup>50</sup>.

Although MNs cannot be studied directly in the same way in humans, the existence of a system with the properties of MNs is supported by ingenious alternative approaches<sup>47,51</sup> including the use of transcranial magnetic stimulation (TMS) of human motor cortex to produce electromyographic potentials in muscle groups<sup>52</sup>. Observing actions involving distal finger movements but not proximal whole arm movements selectively lowered the threshold for TMS to induce electromyographic activity in distal musculature. This demonstrates input from the sight of movements to the neural system involved in motor control of the same movements.

Several functional imaging studies have noted that the sight of hand actions produces activity in frontal regions (premotor cortex and Broca's area)<sup>53,54</sup>, which may be homologous to F5 in the monkey<sup>49</sup>. In a recent fMRI study, activation of the left Broca's area during observation of finger movements became more intense when that same action was executed simultaneously<sup>55</sup>. These imaging studies also reveal activity in parietal cortex. This area, along with possibly the superior temporal sulcus, also shows some evidence of mirror neuron activity<sup>56</sup> & M.Iacoboni (pers com).

### **The functional significance of mirror neurons**

MNs appear to have the capacity to embody a 'supramodal representation' of action, functioning as a bridge between higher visual processing areas and motor cortex (between seeing and doing). As yet, MNs have been investigated with respect to hand actions, but it seems likely that others are concerned with different actions, such as facial expression and speech, and perhaps eye movements and the higher-level abstractions<sup>41, 42</sup>. However, MNs have only recently been discovered. Their precise significance is not yet known, but some specific suggestions are particularly relevant to our discussion.

#### **1. Speech**

Rizzolatti and Arbib<sup>49</sup> have suggested that the part of the monkey brain which contains MNs dealing with hand actions has evolved to subserve speech in humans, with language building on top of a 'prelinguistic grammar of actions' already existing in the primate brain. By acting as a bridge between perceived and performed action and speech, the MN system is thus suggested to have provided the foundations for the evolution of dialogue. Furthermore if MNs do process auditory representations as they do visual ones,

they may be important in representing the relationships between words and their speaker like the personal pronouns. If this is true, the MN system may also provide crucial foundations ontogenetically, particularly with respect to the development of the pragmatic aspects of speech, and thence more complex aspects of language. However, not only the pragmatics of speech may depend on a functional mirror neuron system. Lack of invariance in the physical structure of phonemes gave rise to the motor theory of speech perception, which suggests that we hear sounds according to how we produce them<sup>57,58</sup>. If MNs are an important link between the production and perception of speech - or between sender and receiver<sup>49</sup> - then an intact MN system may be important for other stages of language development as well.

#### **2. Theory of Mind.**

Gallese and Goldman<sup>59</sup> have suggested that it may be possible to predict and also 'retrodict' an observed person's mental state by constructing the appropriate mental correlates of an act once it is 'reconstituted' in the observer's own MN system. They suggest that MN activation can permit the generation of an executive plan to perform an action like the one being watched, thereby getting the observer 'into the mental shoes' of the observed (but see also Gallese<sup>60</sup>). They also note this is a process that requires an ability for controlled inhibition to prevent concomitant execution of an observed action. They argue that such a mechanism is in keeping with the 'simulation' model of ToM, which also requires that observed action sequences are represented in the observer 'off-line' to prevent automatic copying, as well as to facilitate further processing of this high-level social information.

### 3. More basic intersubjective phenomena: emotional contagion and shared attention.

Before moving on to consider the possible role of mirror neurons in autism, it is important to note that there seems no reason in principle why MNs should not address a wide range of actions and the mental states they connote. For example, since emotional states are closely linked to certain facial expressions, observation of a facial expression might result in mirrored (but mainly inhibited) pre-motor activation in the observer and a corresponding 'retrodicted' emotional state. Such a process might help to explain the phenomenon of emotional contagion, in which people automatically mirror the postures and moods of others<sup>61</sup>. This seems particularly likely in view of the close connections between STS neurons, the mirror neuron circuits and the amygdala<sup>43</sup>. Indeed, there is direct electromyographic evidence that observers adopt facial muscle activity congruent with expressions witnessed even when this process is not at an overt level<sup>62</sup>.

Like emotion reading<sup>20</sup>, a capacity for shared attention has been proposed as an important precursor to full theory of mind, partly on the basis of evidence that deficits in this capacity are apparent early in the life of individuals with autism, their occurrence thus being explored as an early warning sign<sup>16,63,64</sup>. Here we note simply that being able to identify the focus of attention of another, or to be able to consider drawing their attention to the focus of one's own attention, is another case of being able to 'stand in the other's shoes'. In shared attention, each individual's attentional focus mirrors the other, raising the prospect that MNs could play a role in this achievement.

### 4. Imitation

In discussing the possible role of MNs in each of the above capacities, some references to imitative-like phenomena ('standing in the others shoes') have been made. It might be thought that the obvious functional role of MNs would indeed lie in imitation (in which case MN outputs would *not* be inhibited). However, noting that there is little evidence of imitation in monkeys<sup>65,66</sup> Gallese and Goldman<sup>59</sup> suggested that in the monkeys in which they have been identified, MNs are functioning to facilitate social understanding of others (to the extent the monkey 'stands in the same 'mental shoes' as the other, as Gallese and Goldman put it). This is not argued to amount to ToM (for which there is also little evidence in monkeys<sup>22,23</sup>), but it may nevertheless represent the kind of foundation which permitted the evolution of ToM in humans<sup>59</sup>.

However, we note there is better evidence for imitation in apes than in monkeys, and of course imitation is both evident and functionally important in our own species<sup>66,67</sup>. We suggest that the evolution of imitation in humans is likely to have utilised an existing MN system, even if its prior uses lay in more generalised kinds of social understanding. As mentioned earlier, fMRI with human subjects during a simple imitation task did indeed find activation in area 44 as well as in parietal cortex, suggesting that the MN system is involved in imitation in humans.

If Gallese and Goldman are right about the function of MNs in monkeys, certain additional capacities had to evolve before MNs could support either imitative or more advanced ToM functions. We may guess that these additional factors reflect the increased cortical volumes of great apes and humans and the representational capacities associated with them; their precise nature is a question for future research. For now, the critical

hypothesis is that MNs provide a key foundation for the building of imitative and mindreading competencies. Accordingly, if Rogers and Pennington were right about the linkage between imitation and ToM, we should thus expect that MNs play important roles in the whole ontogenetic cascade from early imitation to elaborated ToM. This would clearly be consistent also with Gallese and Goldmann's<sup>59</sup> hypothesis that MNs and ToM are linked.

### **Mirror neurons and autism**

These ideas lead directly to our hypothesis that some dysfunction in the MN system might be implicated in the generation of the constellation of clinical features which constitute the autistic syndrome. The most basic hypothesis would be that there is a failure or distortion in the development of the mirror neuron system. This could be due to genetic or other endogenous causes, to external conditions adverse to MN functioning, or some interaction between these. Such factors might affect all MN groups or be confined to just certain groups such as those in the parietal cortex. Complete failure is not necessarily implied, for there might be merely a degree of delay or incomplete development.

Considering the factors discussed in previous sections, such dysfunction could prevent or interfere with imitation, or perhaps more fundamentally, lead to the “impaired formation/co-ordination of specific self-other representations” proposed to lie at the root of the cascade of autistic problems<sup>21</sup>. This in turn could explain the failure to develop reciprocal social abilities including shared/joint attention, gestural recognition and language (particularly the social/pragmatic aspects that Rogers and Pennington<sup>21</sup> note

are the most affected), as well as breakdowns in the development of empathy and a full ToM.

Such a simple ‘MN-dysfunction, imitation-dysfunction’ model is unlikely to provide the whole story, however, insofar as we also need to explain features of repetitive, inflexible and stereotyped behaviour and language that appears to incorporate some copying from others, in some patients with autism. We would suggest that in fact these latter features are testimony to the perception-action linkage problems that occur in autism; they are consistent with the hypothesis that in autism, the mirror neuron system is as a whole malfunctioning. In these cases the system might be evidencing poor modulation. Recall that it has been suggested that a controlled inhibitory system is essential for allowing MN's to operate 'off-line' for simulation ToM to function and develop. If damage extends to such inhibitory components, then certain forms of mimicry might occur, yet be oddly performed.

### **Autism, executive functions and mirror neurons**

In recent years it has been shown that autistic individuals experience difficulties in executive functions like planning<sup>68-72</sup>. It tends to be assumed that executive functions such as planning ability and attentional shifting are the product of developmental processes largely restricted to the individual. But it is also possible that the child learns something of these functions from others, perhaps initially in relatively concrete contexts, such as playing with building bricks in infancy, and then at higher levels of abstraction and over longer time frames, such as planning meals. The initial stages in such a process might correspond to some kind of ‘program-level’ imitation<sup>73</sup>. There is evidence for this

in three-year-old children who are able to acquire, by imitation, alternative hierarchical plans for running off a sequence of actions to complete a functional task<sup>74</sup>. Insofar as MNs code for actions on objects, directed towards a goal, they could be key elements in such a process<sup>75</sup>, helping to translate perceived executive functions into praxis and then generalising them to similar situations. With poor MN development, the key building blocks permitting planning functions to be acquired from the external culture might be unavailable.

If mirror neurons play a part in the development of executive function as well as ToM, one would expect to see a correlation between performance on tests of each of the two abilities. This has recently been demonstrated<sup>76</sup>. The same principles may apply to the acquisition of other executive functions, such as approaches to problem solving and attentional shifting, which can be a problem for autistic children<sup>68,69</sup>. Evidence in favour of this proposition comes from Griffiths et al<sup>70</sup>. They found that apart from tests requiring rule reversal, there was no deficit of executive function in children under 4 years of age with autism. This suggests that the executive deficits are not primary but arise later on in a disrupted pattern of development. Some executive functions, including inhibition and possibly visual working memory appear to be spared in autism<sup>4,67,78,79</sup>. These might be functions much less easily learnt by imitation.

Autistic children show not only characteristic ToM and planning deficits, but also impairment in reconstructing the personal past<sup>80</sup>. Suddendorf<sup>81-83</sup> has proposed that the executive capacity to disengage or dissociate from one's actual current state (putting it offline, as it were) in order to simulate alternative states underlies both 'theory of mind'

and mental 'time-travel' – the ability to mentally construct possible (e.g. planned) events in the future and reconstruct personal events from the past. Thus, in this account mirror neurons may be implied through simulation and executive functions.

### **Neuroimaging mirror neurons and 'theory of mind'**

If ToM and related social deficits in autism are the result of a poorly functioning system of mirror neurons, this might be manifest in recent neuroimaging studies with relevant tasks. The mirror neuron region has been implicated in reading facial emotion in a normal population<sup>84</sup>. Similarly, a task that involved reading emotional expressions from looking at images of eyes, found that individuals with autism showed less involvement of areas normally activated during emotional interpretation, namely the left putative mirror-neuron region (BA 44/45), the superior temporal gyrus (BA 22) bilaterally, the right insula and the left amygdala<sup>85</sup>. A recent review<sup>86</sup> of studies of both typical individuals and those with autism, seeking to identify sites active in ToM functions found that a well demarcated area of the paracingulate gyrus has been consistently implicated, as have areas of the anterior cingulate cortex but not the mirror neuron regions. The paracingulate gyrus and the anterior cingulate cortex are closely linked and receive dense serotonergic innervation, consistent with them performing a modulatory function and this could explain their involvement. One possible reason for the failure of these tasks to activate MN regions may be related to the control tasks that have been used. As these have been predominantly action-based such as following an action-based story, they would be expected to activate the MN regions as much as the ToM task, so discounting their apparent relevance.

### **Testing the hypothesis**

From our hypothesis, several testable predictions flow. First, imitative deficits should be apparent in autism especially where studies take place in the earliest years such as in the study by Charman et al<sup>87</sup>. Particular aspects of imitation expected to be most susceptible are those where imitation involves a co-ordinated activity between different modes of sensory input, different groups of action-coding neurons and self-other visual transformations.

Secondly, we suggest that the McGurk effect<sup>88</sup> whereby the perceived sound is altered by perceiving lip movements making a different sound, may be the result of MN functioning. In this case we predict that on testing groups of children with autism, non-standard McGurk effects will be apparent.

A third prediction can be related to the work of Baron-Cohen et al<sup>64</sup> using the CHAT screening test for autism. These authors found that joint attention at 18 months was a predictive screening item for autism (focussing on siblings of individuals with autism). Our hypothesis predicts that even earlier, appropriately-sensitive screening for an imitative deficit would be predictive in this way.

Fourth, we would predict that imaging studies will indicate altered activation of putative MN regions in the brain during imitation tasks attempted by subjects with autism. Similarly, electrophysiologic studies will show altered muscle activity during the observation of actions, whether facial, vocal or with the hands.

One recent study has attempted to examine mirror neurone activity in Asperger's syndrome<sup>89</sup> Magnetoencephalography was used to detect a decrease in the 20Hz activity

that occurred in the MN region during median nerve stimulation whilst subjects viewed an action. The study did not find a significant difference between the 5 Aspergers' participants and a control group. Our analysis predicts that more extensive testing of people with autism will reveal such a difference. With the small sample size and small expected effect size (the hypothesis was tested in older individuals with the milder form of the disorder) this first study had minimal power and there was a high risk of a type 2 error. It is therefore important that further work is extended to larger groups with other characteristics.

### **Concluding Discussion**

The discovery of mirror neurons offers a potential neural mechanism for the imitation of actions as well as other aspects of understanding social others. Evolution of this system may have been critical in the emergence of proto-culture and Machiavellian manoeuvring in the most encephalized non-human primates, followed by elaborate ToM and language in humans<sup>90</sup>. In the development of the human child, mirror neurons may be key elements facilitating the early imitation of actions, the development of language, executive function and the many components of ToM. A failure to develop an intact, sensitively regulated, mirror neuron system may therefore impair the development of these important human capabilities.

Our exploration of this hypothesis highlights numerous aspects of our ignorance. Unanswered questions include:

1. What other cognitive and neural capacities work in conjunction with MNs to support imitation and ToM functions?
2. How do MNs relate to other social information processing neurons in performing social cognitive functions?
3. How physically extensive are MN functions which relate to autism? Do they just exist in Broca's area or are there such groups in locations such as parietal cortex, paracingulate gyrus and superior temporal sulcus?
4. Do MNs have functions in non-visual modalities as preliminary reports suggest (C. Keyzers, pers.comm; Baker and Perrett, unpublished studies) ? For example, is the sound of an action (or vocal utterance) mirrored by the same neurons as those which mirror its sight? What is the range of actions addressed by MNs?

Despite the various candidates suggested in the literature, a 'prime mover' source of the complex cascade of impairments that characterise autism has so far proved elusive. We are suggesting that developmental delay or distortion of a mirroring system with an early age of onset could be such a 'prime mover'. The heterogeneity of the autistic condition may argue against a single cause, yet the commonalities of the clinical syndrome nevertheless permit the possibility of a core dysfunctional mechanism. If this mechanism is normally a precursor to a cascade of effects on other variable systems, then its dysfunction is likely to result in a quite variable clinical picture. Our proposal offers such a mechanism, together with some preliminary evidence for its existence and empirically testable hypotheses. If it gains further empirical support, this may suggest

important new avenues for both psychological and pharmacological remediative strategies.

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