Emulation and mimicry for social interaction: A theoretical approach to imitation in autism

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The 'broken mirror' theory of autism argues that dysfunction of the 'mirror neuron system' is a root cause of social disability in autism. The present paper aims to scrutinize this theory, and when it breaks down, to provide an alternative. Current evidence suggests that children with autism are able to understand and emulate goal directed actions, but may have specific impairments in automatic mimicry of actions without goals. These data are not compatible with the broken mirror theory, but can be accounted for by a new model called EP-M. The EP-M model segments the mirror neuron system into an indirect, parietal route for goal emulation and planning (EP) and a direct occipital-frontal route for mimicry (M). This fractionation is consistent with neuroimaging and behavioural studies of the mirror neuron system in typical children and adults. I suggest that top-down modulation of the direct M route may be dysfunctional in individuals with autism, leading to abnormal behaviours on mimicry tasks as well as other social disabilities.

There exists a small population of children with dramatic impairments in social and communication abilities, in conjunction with normal or exceptional performance on a limited set of non-social tasks. These children were defined as autistic by Kanner and Asperger over 60 years ago (Frith, 2003). Extensive behavioural testing has now characterised more precisely the autistic profile in terms of poor performance on tasks requiring an understanding of mental states, abnormal imitation and emotion processing, and good performance on tasks requiring the perception of detail or local form.

A number of theoretical models have been proposed to account for some or all of these behaviours. Of particular importance are the ideas that a delay in the development

of theory of mind may account for many of the social and communication disabilities seen in children with autism (Baron-Cohen, Leslie, & Frith, 1985; Frith, Morton, & Leslie, 1991; Happe, 1995), while differences in information processing style, in particular weak central coherence, may account for many of the non-social features of autism (Frith & Happe, 1994). Other theories focusing on emotion (Baron-Cohen et al., 2000), perceptual processing (Behrmann, Thomas, & Humphreys, 2006), gender differences (Baron-Cohen, 2002) and self-other processing (Hobson, 1995) have been proposed.

However, in the last five years, a bold new attempt to understand autism at both the cognitive and neural levels has attracted widespread attention. The provocatively named 'broken mirror' theory of autism (Iacoboni & Dapretto, 2006; Ramachandran & Oberman, 2006; Williams, Whiten, Suddendorf, & Perrett, 2001) has been hailed as a unifying explanation for the various social disabilities seen in autistic spectrum disorders. The aim of the current paper is to examine the broken mirror hypothesis, in particular in relation to studies of the imitation and understanding of other people's actions. The data reviewed provide clear evidence against a simple 'broken mirror' account, and demonstrate that a more sophisticated model of different types of imitation behaviour is required. I present a new candidate model, called EP-M because it proposes an indirect EP route for emulating actions and a direct M route for mimicking. This model details how different regions of the human mirror neuron system contribute to different types of imitation behaviour, and may be differentially impaired in autism. Using EP-M, is it possible to account for data from wide range of developmental, psychophysical and neuroimaging experiments, and to make new predictions for future studies.

The 'broken mirror' hypothesis

At its simplest, the broken mirror hypothesis claims that children with autism have a dysfunction of the mirror neuron system, and that this is the primary cause of their social disability (Dapretto et al., 2006; Iacoboni & Dapretto, 2006; Ramachandran & Oberman, 2006; Williams et al., 2001). Thus, to understand and test the hypothesis, we must first examine the mirror neuron system (MNS). The core of the human mirror neuron system is the inferior parietal lobule (IPL) and inferior frontal gyrus (IFG) (Rizzolatti & Craighero, 2004). Both of these regions respond robustly when hand

actions are performed (Grafton, Mazziotta, Woods, & Phelps, 1992), imagined (Grafton, Arbib, Fadiga, & Rizzolatti, 1996), observed (Buccino et al., 2001), planned (Johnson et al., 2002) and imitated (Aziz-Zadeh, Koski, Zaidel, Mazziotta, & Iacoboni, 2006; Buccino et al., 2004; Iacoboni et al., 1999), and are widely assumed to contain 'mirror neurons' similar to those studied in equivalent regions of the macaque brain (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996). Closely associated with the MNS is a region stretching from lateral occipital sulcus through middle temporal gyrus to the superior temporal sulcus (abbreviated to MTG for simplicity), which is robustly engaged in action observation tasks (Grossman & Blake, 2002) and is also modulated by motor performance (Astafiev, Stanley, Shulman, & Corbetta, 2004). Some definitions of the MNS also include more diverse regions showing 'mirror' responses to pain (Singer et al., 2004), disgust (Wicker et al., 2003) and touch (Keysers et al., 2004), but the present paper will focus only on the core MNS in IFG, IPL and MTG which encodes human hand actions.

The mirror neuron regions of the human brain have several important functions. First, it is often forgotten that the MNS is part of the motor system, with an essential role in controlling our own actions (Grafton et al., 1992). The MNS is necessary for performing flexible, visually-guided goal-directed hand actions, such as using a fork to eat or using a hammer to hit a nail. Damage to the MNS leads to apraxia, characterised by an inability to perform tool-use hand actions in daily life or to verbal or visual command (Buxbaum, Kyle, & Menon, 2005; Halsband et al., 2001; Heilman, Rothi, & Valenstein, 1982). Second, the MNS has attracted much attention in recent years because as well as its motor role, it has an important social function. In particular, the MNS is robustly activated by imitation tasks (Aziz-Zadeh et al., 2006; Buccino et al., 2004; Iacoboni et al., 1999) and action observation tasks (Buccino et al., 2001). These activations are likely to reflect the role of the MNS in understanding the goal or meaning of an observed action (Buccino, Binkofski, & Riggio, 2004; Hamilton & Grafton, 2006) and in predicting the future actions of another person (Kilner, Vargas, Duval, Blakemore, & Sirigu, 2004; Wilson & Knoblich, 2005). Based on these findings, we can make explicit the first prediction of the 'broken mirror' theory of autism. If children with autism have a dysfunctional mirror neuron system, we would expect them to be impaired

in all the cognitive tasks which depend on the MNS, that is, performing goal directed actions, imitating the actions of others, and understanding the goals of others. Evidence for and against the integrity of these cognitive abilities in autism will be reviewed below.

However, the 'broken mirror' theory of autism claims to explain much more than just imitation behaviour. Researchers investigating the MNS have attempted to link this system to a wide variety of social functions, including language (Rizzolatti & Arbib, 1998), theory of mind (Gallese & Goldman, 1998) and empathy (Gallese, 2003). Despite the sparse evidence for some of these links, the 'broken mirror' theory makes the same claims, arguing that the lack of a functioning mirror neuron system could be the underlying cause of the disabilities in theory of mind and in emotion processing which have been observed in autism. The robustness of these more speculative claims will be considered in the final section of the paper.

Testing the broken mirror hypothesis

The first prediction of the broken mirror hypothesis is that children with autism should perform poorly on tasks requiring the mirror neuron system, in particular tests of visuomotor control, imitation and action understanding. Numerous studies have examined imitation abilities in children with autism (Hobson & Lee, 1999; Ohta, 1987; Rogers, Bennetto, McEvoy, & Pennington, 1996; Rogers, Hepburn, Stackhouse, & Wehner, 2003; Smith & Byrson, 1998), and a recent review concluded that children with autism are delayed in imitation skills relative to control children (Williams, Whiten, & Singh, 2004). This imitation data would seem to provide straightforward evidence in favour of the broken mirror hypothesis.

However, some recent results cast doubt on this conclusion. I recently carried out a behavioural study of MNS function in autism, in collaboration with Rachel Brindley and Uta Frith (Hamilton, Brindley, & Frith, in press). In order to assess goal emulation and goal understanding abilities in autism, we tested 25 autistic children with a verbal mental age around 4 years 6 months as well as 31 VMA matched controls. All the autistic children were substantially impaired on a battery of theory of mind tasks, as expected (Happe, 1995). However, when the children were tested on a goal-directed imitation task (Bekkering, Wohlschlager, & Gattis, 2000; Gattis, Bekkering, &

Wohlschlaeger, 2002) (Figure 1A), autistic children performed just the same as typically developing children. Both groups imitated the demonstrator's goal but failed to use the correct hand on contralateral trials, with good performance on all other trials (Figure 1C). In typical children, this systematic pattern of error has been taken as evidence that the children encode action goals rather than the means by which the goal is accomplished (Wohlschlager, Gattis, & Bekkering, 2003). Thus, the presence of hand errors on contralateral trials in the autistic group indicates that these children also understand the demonstrator's goal and emulate it.

FIGURE 1 ABOUT HERE

We also examined motor planning and gesture understanding abilities in the same children. On a test of motor planning abilities (Rosenbaum et al., 1990), we found that both controls and the autistic group were better able to plan an action when the experimenter demonstrated the correct action first (Hamilton et al., in press/ experiment 3). Finally, we tested children on gesture understanding, using stimuli developed for assessing patients with apraxia (Mozaz, Rothi, Anderson, Crucian, & Heilman, 2002) (Figure 1B&D), and found that children with autism performed better than controls at this task (Hamilton et al., in press/ experiment 4). That is, children with autism were better able to judge if a pictured gesture matched a cartoon, compared to their VMA matched controls. Overall, we concluded that autistic children have no difficulties understanding the meaning of an action or imitating the goal of an action.

These results are not compatible with the 'broken mirror' theory of autism, but are congruent with several other studies. Two independent groups have used an incomplete intentions task (Meltzoff, 1995) with autistic children. In this task, the child sees an adult try and fail to perform a task and then has the chance to do the same task; both groups report that children with autism emulate the adult's goal, just like typical children (Aldridge, Stone, Sweeney, & Bower, 2000; Carpenter, Pennington, & Rogers, 2001). Young children with autism are also able to imitate object-directed actions in order to receive non-social feedback in the form of lights and sounds (Ingersoll, Schreibman, & Tran, 2003). Other researchers have reported good imitation of object-use actions by children with autism (Stone, Ousley, & Littleford, 1997), good imitation of meaningful actions (Rogers et al., 1996), and good performance on an explicit imitation task (Beadle-

Brown & Whiten, 2004). Children with autism also have an intact ability to monitor their own actions and intentions (Russell & Hill, 2001). Two studies using neuroimaging methods in adults provide similar results. Adults with autism represent the goal of another person's action (Sebanz, Knoblich, Stumpf, & Prinz, 2005) and show normal brain responses during the observation of goal directed actions (Avikainen, Kulomaki, & Hari, 1999).

To summarise, on a range of studies testing explicit, goal directed imitation, both behaviour and neural activity was normal in the autistic population. These data are not compatible with a straightforward 'broken mirror' theory of autism in which the whole of the mirror neuron system is dysfunctional in these individuals.

Emulation and mimicry in autism

The data reviewed thus far present a challenge. If children with autism are passing a variety of tasks testing MNS function, what are we to make of the multitude of reports claiming profound imitation deficits in autism? For example, children with autism failed to mimic meaningless actions (Rogers et al., 1996; Stone et al., 1997) or gestures (Smith & Byrson, 1998). They make perspective errors on some meaningless imitation tasks (Ohta, 1987; Smith & Byrson, 1998) and have difficulties with common batteries of a mixture of imitation tasks (Charman et al., 1997; DeMeyer et al., 1972; Rogers et al., 2003). Work with adults suggests that mixed lists of meaningful and meaningless actions may all be treated as if they are meaningless (Tessari & Rumiati, 2004). Abnormal brain activity has also been reported in children with autism during tasks involving imitation of emotional facial expressions (Dapretto et al., 2006). Similarly, autistic children fail to show mu rhythm suppression (Oberman et al., 2005) or motor cortical facilitation (Theoret et al., 2005) during observation of meaningless actions.

Looking over these lists of tasks, we can see some similarities between the ones which the children with autism pass, and the ones which they fail. Autistic children show normal performance and normal brain activity on imitation tasks which involve a goal or object. I classify these tasks as *emulation* tasks, using the word in the sense of goal emulation (Byrne & Russon, 1998). An individual who emulates an observed action

must first obtain a teleological understanding of the goal or meaning of an action (Csibra, in press), and may then, if she chooses, plan or reconstruct the action by her own means. Thus, emulation describes the process of goal-directed imitation (Bekkering et al., 2000; Wohlschlager et al., 2003) which I suggest is intact in children with autism (Hamilton et al., in press).

In contrast, the tasks which children with autism fail can be broadly classified as *mimicry* tasks. That is, these tasks require the child to spontaneously copy the low level, kinematic features of an action. The tested actions do not normally involve an object, but are either meaningless hand gestures, or facial actions including emotional expressions. Typically developing children spontaneously mimic each other as a form of communication (Nadel, 2002) and typical adults unconsciously mimic each others meaningless actions in order to facilitate social interaction (Chartrand & Bargh, 1999; Lakin & Chartrand, 2003). However, this spontaneous mimicry of meaningless actions appears to be lacking in children with autism. Some differences between emulation and mimicry are summarised in Table 1.

TABLE 1 ABOUT HERE

Further evidence for a distinction between emulation and mimicry in autism can be found in two studies which have examined both types of imitation. McIntosh and colleagues used electromyography to record from facial muscles and obtain an accurate, implicit measure of mimicry. They found that adults with autism did not show automatic mimicry of emotional facial expressions, but typical adults did. However, both groups showed the same responses when explicitly instructed to copy the expression they saw in the stimulus picture (McIntosh, Reichmann-Decker, Winkielman, & Wilbarger, 2006). Thus, the adults with autism failed to spontaneously mimic but could emulate when the desired facial expression was explicitly set as a goal. A second behavioural study of children examined the imitation of both the style and goal of a novel action, for example, rattling two objects to make a sound either loudly or softly (Hobson & Lee, 1999). Hobson & Lee found that children with autism tended to imitate the goal of the action, they were able to hold the objects and make a sound, but did not spontaneously copy the style or manner in which the action was performed. That is, the children emulated the demonstrator, but did not mimic the precise style of the action.

Overall, these data demonstrate that the conclusion that children with autism show a global deficit in imitation is premature. Children with autism clearly have major difficulties with tasks requiring mimicry of style or of meaningless action. However, on a different subset of imitation tasks, those which involve using an object and those which require understanding the goal of an action, the autistic children are not impaired. These results mean that a global breakdown of the mirror neuron system is not found in autism, and therefore the broken mirror hypothesis cannot be sustained. I now consider how our understanding of the mirror neuron system can be refined to accommodate these data.

Breaking up the mirror neuron system

If we are to understand the origins of the different types of imitation behaviour - emulation and mimicry - in typical individuals as well as those with autism, we must take a closer look at the mirror neuron system itself. Many previous neuroimaging studies of the human MNS (for example Buccino et al., 2001; Buccino et al., 2004) report activation in all three MNS nodes, that is, the inferior frontal gyrus (IFG) and inferior parietal lobule (IPL) and the middle temporal gyrus (MTG). Similarly, many theoretical approaches to the function of the MNS treat the system as a whole (Gallese, Keysers, & Rizzolatti, 2004; Keysers & Perrett, 2004; Rizzolatti & Craighero, 2004) and do not systematically distinguish between the three different nodes. However, new work suggests that this idea of a unitary MNS is too simplistic. By examining the functions of the different components of the MNS, it is possible to obtain a new model of the cognitive and neural systems underlying human emulation and mimicry behaviour.

The first clue to the breakdown of the MNS comes from a neuroimaging study I conducted to examine the representation of the goals of other people's actions in the brain. Several previous studies have attempted to localise goals or intentions in the human brain, reporting activity in IFG (Iacoboni et al., 2005) and in the superior temporal sulcus (Pelphrey, Morris, & McCarthy, 2004). However, both these studies used subtraction methods which are not ideal for dealing with goals, because an action with the goal object or the goal context removed becomes a mime or a complex action, not a goal-less action. I have been able to bypass this problem by using a repetition suppression approach. Compared to observation of novel goals, observation of repeated

goals results in suppression of activity in the anterior intraparietal sulcus (Hamilton & Grafton, 2006), part of the parietal node of the MNS. Because repetition suppression arises due to the population coding within a brain region (Grill-Spector, Henson, & Martin, 2006), this means that the anterior intraparietal sulcus contains populations of neurons which encode the object-goal of an observed action, for example, the goal of taking a cookie (Figure 2A).

FIGURE 2 ABOUT HERE

A second study replicates and extends this finding. I used repetition suppression to differentiate between neural representations of the identity of a grasped object (wine-bottle or dumbbell) and the type of grasp used to take the object (a fingertip grasp on the neck of the wine bottle or a whole hand grasp on the middle of the bottle) (Hamilton & Grafton, in press). As before, repetition suppression for the object-goal was found in the anterior intraparietal sulcus. In contrast, repetition suppression for the type of grasp was found in the other two MNS regions - IFG and MTG (Figure 2B).

These results are consistent with several other studies which implicate the IPL in a more abstract, goal-oriented action representation than the IFG or MTG. Transcranial magnetic stimulation over the anterior intraparietal sulcus disrupts a person's ability to perform goal directed hand actions (Tunik, Frey, & Grafton, 2005), and stroke damage to the IPL disrupts the ability to understand and imitate goal directed actions (Buxbaum et al., 2005). Recordings from mirror neurons in the macaque parietal lobule reveals that these cells encode complex, goal directed action sequences for both self and other (Fogassi et al., 2005). In contrast, studies of IFG suggest that this region is required for interpreting low level action properties (Pobric & Hamilton, 2006; Urgesi, Candidi, Ionta, & Aglioti, 2007), and for performing grasping actions (Ehrsson et al., 2000; Rizzolatti et al., 1996).

Taking all these results together, it is now possible to build a cognitive model of visual-motor control and action understanding in the different components of the mirror neuron system (Figure 3). This model is described in detail elsewhere (Hamilton & Grafton, in press), so only a brief summary is provided here. The key feature is that the three nodes of the MNS do not all perform the same function. Rather, the MTG node provides a visual representation of the low level, kinematic parameters observed actions,

the IPL node provides a more abstract representation of the goal of the observed action and the IFG node provides a motor representation of the observed kinematic parameters, in preparation for imitating the action. These three nodes enable humans to plan and perform complex visually guided hand actions, to imitate another person's action and to understand the meaning of that action.

Emulation and mimicry in the brain

Figure 3 illustrates the proposed localisation of different visuo-motor representations of action in the brain. However, to understand imitation behaviours, it is not enough to simply localise action representations; we must also understand how information flows between the different nodes. I now present a new model of cognitive information processing within the mirror neuron system, which provides an explanation for both emulation and mimicry behaviours, and for the differential impairment of these behaviours in autism.

FIGURE 3 ABOUT HERE

The primary route of information processing through the MNS is the EP route illustrated in Figure 3. There is clear anatomical evidence for this pathway in both humans (Catani, Jones, & ffytche, 2005) and macaques (Rozzi et al., 2006), and it is central to most models of MNS function (Keysers & Perrett, 2004). The important feature of the EP route is that imitation occurs in two stages. First, the visual representation of the observed action (in MTG) is used to infer the goal or meaning of the action (in IPL). The process of inferring a goal, which is a key component of emulation behaviours, requires the E route. Once a goal representation is obtained, the P route can then be used to plan an action based on that goal, which may or may not be similar to the observed action. The planned action is represented in IFG, in terms of its motor parameters. To give a concrete example, consider an adult showing a child how to hammer a toy peg into a board. The adult takes the toy hammer and with an exaggerated gesture and sound, gently taps the top of one peg. The child watches carefully, and understands that the aim of the toy is to sink the peg (E route). She then grabs the toy, and with her thumbs forces the peg through the board (P route). The child has succeeded in emulating the adult's action, using her own means to achieve the desired goal. In other

circumstances, the E step might occur alone, resulting in action understanding without copying. Or the child might form her own goal and use the P route to accomplish it, again without copying. But when the two steps occur together, the observed behaviour can be described as goal-emulation. Previous theories have proposed that action understanding and skill learning depend on emulation abilities (Csibra, in press; Wohlschlager et al., 2003), and the indirect EP route provides a route for emulation to occur.

However, increasing evidence suggests that the EP route is not the only way that information about actions is processed in the brain. Humans have a tendency to mimic the low level kinematic features of observed actions (Chartrand & Bargh, 1999), and a series of studies demonstrate direct effects of a perceived action on the performance of a similar or different action (Brass, Bekkering, & Prinz, 2001; Heyes, 2001). Furthermore, observation of action interferes with performance of an incompatible action (Kilner, Paulignan, & Blakemore, 2003) and performance interferes with observation (Hamilton, Wolpert, & Frith, 2004). All of these effects are best explained by a direct M route (Figure 3), which allows immediate associations between visual representations of kinematic features and motor representations of the same kinematic features. For example, as two people watch each other dance across a nightclub, one might unconsciously copy the style and rhythm of the other, by representing the kinematic patterns of the observed dance in MTG and translating them by means of the M route directly to a motor representation in IFG. This behaviour might increase the social bond between the dancers, without either having an explicit goal in each dance move.

Though the behavioural evidence for the direct M route is powerful, there is an important reason to hesitate before allowing this route in the model. The majority of our data on neuro-anatomical connectivity comes from the macaque, but there is little anatomical evidence for a direct connection between middle temporal regions and inferior frontal gyrus in macaques. Thus, most reviews of the MNS in the macaque focus on the role of the EP route alone (Keysers & Perrett, 2004; Rizzolatti & Craighero, 2004). It is interesting to note here that the MNS regions in the macaque respond only to goal directed actions (Fogassi et al., 2005; Gallese et al., 1996; Umilta et al., 2001), consistent with the idea that the goal-mediated EP route is dominant in the monkey.

However, using anatomical studies of the macaque to draw conclusions about the function and structure of the human brain can be misleading. New studies using diffusion tensor imaging to track neural pathways suggest that a direct route between MTG and IFG may exist in humans (Catani et al., 2005; Rilling et al., 2006). These same studies also demonstrate dense interconnections between MTG and IPL (E route), and between IPL and IFG (P route). Overall, current anatomical data support the present proposal that imitation and action understanding can use both an indirect EP route via the IPL and an M route directly linking MTG to IFG. As a caveat, it is important to note that the human anatomical studies were conducted in the context of examining language processing, not the MNS. Further research will be needed to determine the exact termination of each neural path and their role in action processing.

Finally, the EP-M model presented in Figure 3 is a dual route model of action processing. It is similar to previous dual route schemes, which have been proposed for the imitation of actions (Rothi, Ochipa, & Heilman, 1991; Tessari & Rumiati, 2004) and for language processing (Lichtheim, 1885). Drawing on this heritage, the EP-M model now maps the dual route architecture to the brain in the context of the MNS. The EP route from MTG to IPL to IFG requires an understanding of the goal of an observed action in the IPL, which can then be planned and performed. The M route directly associates visual and motor kinematics, allowing automatic mimicry of actions without any abstract interpretation. In typical individuals, both routes may often be used together, but in neuropsychological populations, in individuals with autism or by using neuroimaging, it is now possible to distinguish between the two routes. Thus, the EP-M model makes new predictions for action representation in developmental and neuroimaging studies.

Emulation and mimicry in autism

Of particular interest here is how the EP-M model can help us understand the pattern of performance in imitation tasks which we observed in children with autism. As summarised above, autistic children succeed on tasks requiring emulation of the goal of an action, but show abnormal performance on tasks requiring automatic mimicry. This behavioural distinction maps cleanly onto the EP-M model. In particular, I suggest the indirect EP route is intact in children with autism, allowing the children to emulate an

observed action if they understand the goal of the action. In contrast, the functioning of the direct M route may be compromised in autism, reducing the child's ability to spontaneously imitate a meaningless gesture or facial expression.

The EP and M pathways both start from the same visual representation of action and both involve self-other processing and , which means that purely perceptual theories (Behrmann et al., 2006) or self-other theories (Hobson, 1995) cannot account for the difference between the pathways. Furthermore, both the EP and M pathways involve the brain regions which make up the human MNS. Thus, the distinction between an intact EP route and a damaged M route in autism makes a radical reassessment of the 'broken mirror' hypothesis necessary. This hypothesis argued that the whole of the MNS is dysfunctional in autistic children (Ramachandran & Oberman, 2006; Williams et al., 2001). In contrast, the present data demonstrate that the MNS can be fractionated into two different pathways, the EP and M routes, while behavioural testing of children with autism indicates that the EP route is intact but the M route is compromised. Thus, we must reject the idea that a global MNS dysfunction – a single 'broken mirror' – is responsible for the disabilities in social interaction seen in autism. However, it is still possible that the MNS has some role in autism. In particular, the M route, which provides for direct associations between visual and motor kinematic representations, may be compromised in children with autism. Could a deficit in the M route alone be sufficient to cause the social impairments observed in autism, and thus 'rescue' part of the broken mirror hypothesis?

There are several reasons to believe that mimicry behaviour has an important social function. Typical individuals do not mimic one another all the time, but must select who to mimic and when. Adults mimic when they want to enhance a social affiliation with another individual (Lakin & Chartrand, 2003) and being mimicked enhanced pro-social behaviour (van Baaren, Holland, Kawakami, & van Knippenberg, 2004). However, too much mimicry is liable to be consciously detected and result in ridicule rather than friendship. Thus, mimicry behaviours must be carefully controlled to achieve a social bond. One possibility is that children with autism, with an impairment in the direct M route, fail to mimic and thus fail to gain the social advantages of mimicry. It is likely that the proponents of the 'broken mirror' theory would support this idea.

However, an alternative hypothesis is possible. Individuals with autism may have an intact M route, but might have difficulties modulating the route and deciding who and when to mimic. Thus, in some situations, children with autism might show excess mimicry (for example, echolalia or echopraxia), while in other situations they would fail to mimic at all. An intriguing recent experiment provides some support for this 'top-down modulation hypothesis'. When typical adults are required to inhibit their natural tendency to mimic observed actions, neural activation is found in the medial prefrontal cortex, precuneus and bilateral temporal-parietal junction (Brass, Derrfuss, & von Cramon, 2005). This network of brain regions is commonly activated in tasks requiring inferences about other people's mental states, i.e. theory of mind tasks (Frith & Frith, 2003). Impairments of theory of mind (Baron-Cohen et al., 1985; Happe, 1995) and abnormal activity in the theory of mind network (Castelli, Frith, Happe, & Frith, 2002; Happe et al., 1996) are characteristic of autism. The results of Brass and colleagues suggest that the theory of mind network may be responsible for controlling mimicry behaviour in typical adults. Thus, dysfunction of the 'theory of mind network' could be a cause (not a consequence) of abnormalities of the direct M route and abnormal mimicry behaviour in autism.

Under this hypothesis, the mirror neuron system is not broken in autism. Rather, top-down modulation of the direct M route is abnormal and this lack of modulation causes abnormal social behaviour. Such abnormalities in top-down modulation would have a devastating effect on mimicry but could also have a wider impact on other social and even non-social behaviours. For example, differences in top-down modulation of face processing (Bird, Catmur, Silani, Frith, & Frith, 2006) in autism might be able to account for some of the perceptual differences in autism (Behrmann et al., 2006). The top-down modulation hypothesis has much in common with the idea that weak central coherence is a defining characteristic of autism (Frith & Happe, 1994). In both cases, there is no single, low-level cognitive process which is impaired in autism; rather an imbalance in higher level processing can lead to a broad and varied array of impairments, in particular in social skills, as well as strengths in some non-social skills. Thus, we return to an appreciation of the complex mixture of cognitive abilities and disabilities seen in children with autism. And as so often in the past, Uta Frith's work gives an

important insight into the cognitive systems underlying intact emulation behaviour and impaired mimicry behaviour in autism.

These last ideas are of course speculative and only hint at some ways in which we may be able to understand imitation behaviours in relation to autism. New experiments will be needed to determine if a broken mirror neuron system, a broken 'M-route', a dysfunction of top-down modulation, or some other cause, can account for the varied profile of cognitive strengths and weaknesses seen in autism. The EP-M model provides one way in which to make sense of the varied performance of autistic children on imitation tasks, and makes new predictions for future experiments in neuroimaging and developmental social neuroscience.

Conclusions

This paper aimed to examine and dissect the 'broken mirror' theory of autism, which argued that a dysfunction of the mirror neuron system is responsible for poor social skills in autism. I presented data demonstrating that children with autism are able to understand and emulate the goals of other people's actions, but may have difficulties with mimicry. This pattern of behaviour can be understood in terms of the EP-M model, in which there are two possible routes for imitation behaviour. The indirect EP route allows emulation and planning of goal directed actions, while the direct M route associates low level visual and motor representations. Current data suggests that the M route may be abnormal in autism, but the EP route intact. The EP-M model is compatible with the idea that individuals with autism have particular difficulties with top-down modulation of the M route, but further research will be needed to test this hypothesis.

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Figure 1. Emulation in autism

A. Goal directed imitation task. Three possible trial types are illustrated, with the child as the upper figure and the adult demonstrator as the lower figure. On each trial, the child is required to copy the adult's action. The typical hand error is illustrated on the contralateral trial.

B. Gesture understanding task. One stimulus card is illustrated. The child was shown cartoon and the pictures and was asked 'which hands fill the gap'? Each child performed 8 trials with object use gestures and 8 with symbolic gestures.

C. Performance on the goal directed imitation task. Both control children and those with autism made substantially more errors on the contralateral trials when dots were present on the tabletop.

D. Performance on the gesture understanding task. Autistic children gave significantly more correct responses than control children.

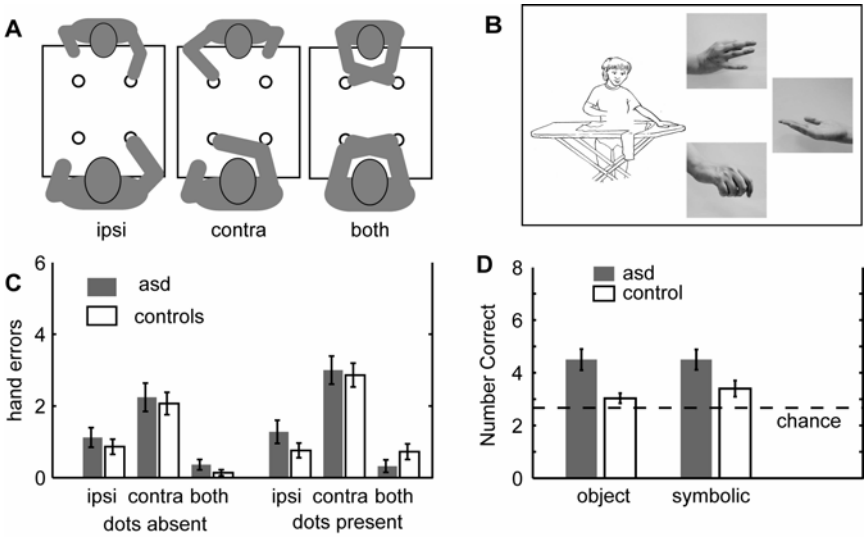

Figure 2. Neural representations of goals and kinematics

A. The neural representation of goals, for example, 'take a cookie' (top row), is found in the left anterior intraparietal sulcus (bottom row)

B. The neural representation of grasps, for example, grasping a wine bottle with the whole hand compared to grasping it with the fingertips (top row) is found in the inferior frontal gyrus and lateral occipital cortex (bottom row).

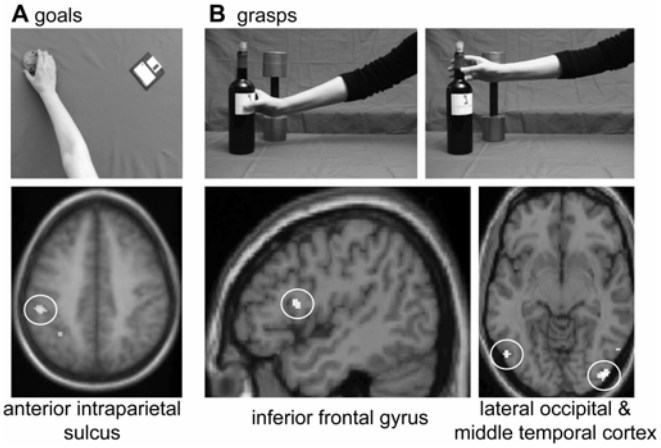

Figure 3. The EP-M model

The EP-M model distinguishes three nodes for social-motor information processing within the mirror neuron system. The MTG (middle temporal gyrus) node provides a visual representation of kinematic features of observed actions; the IPL (inferior parietal lobule) node represents the goal of the action; and the IFG (inferior frontal gyrus) node contains a motor representation of the kinematic features of the action. There are three routes by which information can flow between these nodes. The E-route from MTG to IPL allows for emulation and understanding of the goal of an action and the P-route from IPL to IFG allows for action planning. Together, these two form the indirect EP route which supports goal-emulation behaviour. In contrast, the M-route from MTG to IFG allows the formation of direct associations from visual kinematic to motor kinematic representations, and supports mimicry behaviours.

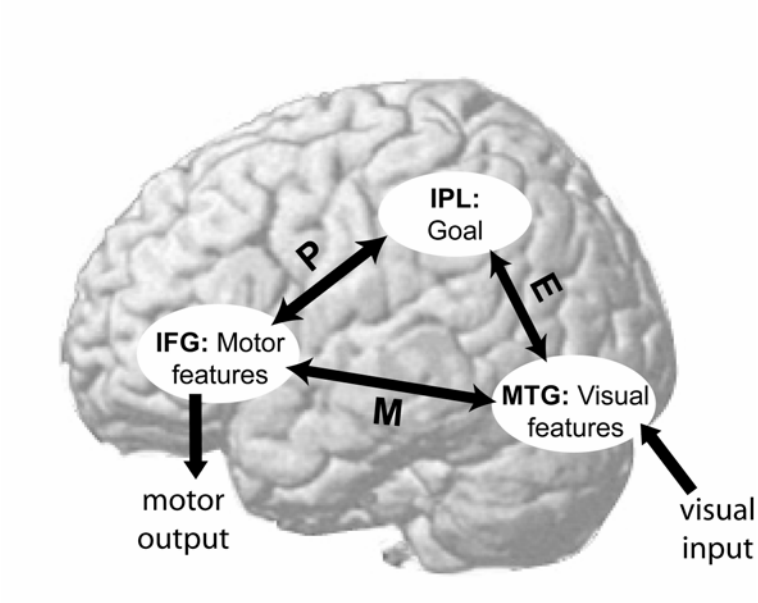

Table 1. Characteristics of emulation and mimicry

Emulation	**Mimicry**
Requires goal oriented action representations	Uses low level representations of kinematic features
Occurs in two stages: the E route involves understanding of the action goal while the P route involves planning a new action to achieve the goal	Occurs in a single stage, in the M route which directly links visual and motor representations of kinematic features.
Relies particularly on inferior parietal lobule / anterior intraparietal sulcus	Relies on middle temporal gyrus / lateral occipital cortex and inferior frontal gyrus
Often explicit and controlled	Normally implicit and automatic
Intact in autism	Impaired in autism
Useful in practical situations such as gaining food or using tools	Useful in social situations such as building social affiliations