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Does the mirror neuron system and its impairment explain human imitation and autism?

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Abstract

The proposal that the understanding and imitation of observed actions is made possible through the 'mirror neuron system' (Rizzolatti, Fogassi, & Gallese, 2001) has led to much speculation that a dysfunctional mirror system may be at the root of the social deficits characteristic of autism (e.g., Ramachandran & Oberman, 2006). This chapter will critically examine the hypothesis that those with ASD may be in possession of a 'broken' mirror neuron system. We propose that the deficits seen in imitation in individuals with ASD reflect not a dysfunctional MNS, but a lack of sensitivity to those cues that would help them identify *what* to imitate. In doing this, we will also argue that imitation in typically developing children cannot be explained by appealing to a direct-matching mechanism, and that the process by which young children imitate involves a far more complex yet effortless analysis of the communication of those who they learn from.

Keywords: Imitation; Emulation; Mirror neuron system; Autism

1. The 'dysfunctional mirror neuron system' hypothesis of autism

Action understanding and imitation are both proposed to be subserved by a direct-matching mechanism of the mirror neuron system (MNS, Rizzolatti & Craighero, 2004), and so any dysfunction of this system should be expected to manifest in impairments in either or both of these capacities. There is, in fact, a long history of reports of deficient imitative abilities in individuals with autism (for reviews see Rogers & Pennington, 1991; Smith & Bryson, 1994; Williams, Whiten, & Singh, 2004). Over a large number of studies, children with autism have consistently been reported to imitate less than typically developing children on a variety of imitation tasks. These difficulties include imitation of object-directed actions (e.g., Whiten & Brown, 1999), pantomimed actions (Rogers et al., 1996) and gestures (Roeyers et al., 1998).

The hypothesized link between the MNS and action imitation has led a number of authors to propose that the deficient imitative abilities of individuals with autism spectrum disorder (ASD) may result from a dysfunctional MNS (Oberman et al., 2005; Rizzolatti & Fabbri Destro, 2007; Williams et al., 2006). In recent years, several papers have appeared reporting evidence supporting this hypothesis. Two of these studies explored the activation of the autistic brain during action observation without requiring any imitative response from participants. Oberman and colleagues (2005) measured the suppression of the mu rhythm, a component of the electroencephalogram (EEG) proposed to reflect MNS activation (Pineda, 2005) because it is suppressed both when we execute actions ourselves, and when we observe others executing actions. They found that, although individuals with autism exhibited normal mu-suppression over sensorimotor cortex during the execution of a hand movement, there was no corresponding suppression of the mu-wave when they observed someone else performing a hand movement. In another study, Théoret et al. (2006) used transcranial magnetic stimulation (TMS) to induce motor-evoked potentials (MEPs) while participants watched videos of finger movements, either from their own perspective (the

hand appeared in an orientation that was consistent with the subject's own orientation), or from the perspective of another individual so that it appeared upside down to the observer. The authors reported that, for control subjects, MEPs were facilitated during observation of both self- and other-presented views of the hand, but for individuals with autism the facilitation was only apparent during observation of the 'other' hand orientation.

Further studies explored the activity in the MNS during observation for imitation, and during imitation itself, by functional magnetic resonance imaging (fMRI) (Dapretto et al., 2006; Williams et al., 2006) or by magnetoencephalography (MEG) (Nishitani et al., 2004). The fMRI studies reported activation differences during imitation between individuals with ASD and matched controls in areas comprising the mirror neuron system. Dapretto and colleagues reported a difference in activation of the *pars opercularis* of the inferior frontal gyrus (IFG) during imitation of facial expressions in children with autism and those without autism, with higher activation in those without autism. Williams et al. (2006) found activation differences in the posterior parietal area (PPA), but no effects in the IFG. (In fact, unlike the study on which their study was based [Iacoboni et al., 1999], they reported no activation of the IFG even in control subjects during imitation.) Crucially, however, neither of these studies reported any significant difference between the behavioural imitative capacity of the participants with autism and those without, suggesting that whatever the role of the IFG or the PPA, it is not crucial to the ability to be able to accurately imitate another person. A similar dissociation between behavioural and neural responses was found by Nishitani et al. (2004), who reported that the delayed activation of the IFG in Asperger syndrome was not reflected in their overt imitative response of facial expressions, which were executed with the same latency as by control subjects.

Together, these findings support the view that the functioning of the MNS, just like the performance in some imitation tasks, is atypical in autism. However, the 'dysfunctional

mirror system' hypothesis puts forward the more specific claim that a dysfunctional MNS is one of the *causal factors* that lead to imitation deficits and possibly further symptoms of ASD. If proper functioning of the mirror neuron system is necessary for imitation, and the ability to imitate is essential for the normal development of social cognition, a dysfunctional mirror neuron system would explain many aspects of autism spectrum disorder.

2. Arguments against the 'broken mirror' hypothesis

However, we believe that there are good theoretical and empirical reasons to question the dysfunctional MNS hypothesis of autism. In this section, we argue that the nature of the links between the MNS, imitation and autism does not indicate a simple causal relationship among them. We show first that the primary purported function of the mirror neuron system, action understanding in terms of goals (Rizzolatti & Craighero, 2004), appears to be intact in autism. Second, we review evidence demonstrating that, although imitation is atypical in autism, the imitative *abilities* of the individuals with the disorder do not seem to be affected. Third, we challenge the idea that imitation in general is supported by the human mirror system, and that variation in imitative performance would map onto variation in motor mirroring processes.

2.1. Action understanding is not impaired in autism

Although, compared with imitation, the domain of action interpretation and understanding in ASD has received comparatively little attention, what evidence does exist suggests that those with ASD are not impaired at action understanding.

In a classic study of infants understanding of the goals and intentions behind actions, Meltzoff (1995) presented typically developing 18-month-old infants with demonstrations of actions which failed to achieve their goals. In a subsequent imitation phase, infants did not

imitate the actions that the adult actually did (the failed-attempt), but instead performed the action that the adult had been trying to achieve. The same paradigm has recently been employed for use in children with autism (Aldridge, Stone, Sweeney, & Bower, 2000; Carpenter, Pennington, & Rogers, 2001). These studies found that autistic children, like typically developing infants, performed the action that the demonstrator had intended to perform, suggesting that they understood what the demonstrator had intended (but failed) to achieve, rather than interpreting her actions in terms of their actual observable outcomes.

Results from studies that asked observers to describe the patterns of actions of animated shapes further suggest that individuals with ASD are not impaired at action understanding. When asked to provide a verbal description of a short animation in which two triangles and a circle move around each other, adult participants readily attribute an elaborated plot to the scene, for example, describing one triangle as an aggressor who wants to stop the other shapes from getting into his house (Heider & Simmel, 1944). Although individuals with ASD rarely provide mentalistic descriptions of the triangles' behaviour (e.g., that 'one triangle is *tricking* the other triangle') (Abell, Happe, & Frith, 2000; Klin, 2000), they nonetheless provide descriptions in terms of goal-directed actions (e.g., that 'one triangle is *chasing* the other triangle') (Castelli, Frith, Happe, & Frith, 2002), and are also able to distinguish mechanical launching events from intentional reactions (Bowler & Thommen, 2000). Young children with autism also perform as well as typically developing children on an 'unfulfilled intentions' version of this study, in which a circle is depicted rolling up and down a hill, getting closer and closer to a target object. Both typically developing children and those with ASD provide descriptions of the scene in terms of the intentions of the circle to reach the target (Castelli, 2005).

A recent study by Sebanz and colleagues (2005) investigated the hypothesis that individuals with autism are impaired at representing the actions of others. When instructed to press either the left or right key depending on whether the stimulus is green or red respectively (the relevant dimension), typical adults are slower to respond when the colour is presented on an incompatible background (a finger pointing left when the colour is red) than when it is compatible (a finger pointing right when the colour presented is red). If this task is carried out as a go-nogo task, in which subjects have to press a button only if the colour is red, the interference of the incompatible stimulus disappears. However, if the task is distributed between two individuals such that one person's task is to respond if the colour is red and the other's is to respond if the colour is green, the effect of the irrelevant stimulus returns. Now, if the participant's 'colour' appears in the context of a finger pointing towards the other participant, his response is slower than if he were doing the task alone. Because the task at the individual level is identical between the joint and individual conditions, the slower reaction time in the joint condition likely arises because the participants are representing the other person's requested response (Sebanz, Knoblich, & Prinz, 2003). Individuals with ASD show the same effect when performing the task with another individual, suggesting that they also represent the actions of others (Sebanz, Knoblich, Stumpf, & Prinz, 2005).

2.2. Imitative abilities are not impaired in ASD

Many studies have explored the imitative performance of individuals with autism, but their findings are somewhat contradictory. Early research consistently reported deficits in imitation. For example, Rogers et al. (2003) employed a battery of imitation tasks including manual (e.g., clapping hands), object-directed (e.g., using elbow to touch box) and oral (stick out tongue) actions, and found that in all three domains toddlers with ASD imitated less

than age-matched controls. However, the majority of tasks reporting imitation deficits in autistic children appear to involve non-object directed actions (Hamilton, in press). For example, Rogers et al. (1996) report significantly less imitation in older children with autism than in control children on a series of pantomime and meaningless actions.

Nevertheless, recent investigations into the question of imitation in ASD suggest that there may not be such a deficit in the imitative *abilities* of individuals with autism. In a well-known test of 'goal-directed' imitation, Bekkering and colleagues showed 4 to 6-year-old typically developing children actions in which an adult moved her hand to touch dots on the table in front of her. The hand movements were either ipsilateral or contralateral to the adult's body. In a control condition, no dots were present on the table, but the demonstrator performed the same actions to touch the table. When children were asked to copy the demonstrator, they tended to ignore the type of action that was performed (i.e., whether it was ipsi- or contralateral) and simply performed the touching of the dots with whichever hand was closest. However, when the dots were not present, children tended to imitate the type of action demonstrated. The failure to use the correct hand when the dots are present has been interpreted as the child giving priority to the adult's goal (i.e., touching the correct dots), whereas when the dots are absent, the way in which the action is carried out is itself interpreted as the goal (Bekkering, Wohlschlaeger, & Gattis, 2000). Recently, Hamilton, Brindly, & Frith (2007) reported that children with autism performed in the same way as controls, on this task. This suggests that (1) they had no imitation impairment (their level of imitation was comparable to that of the control children), and (2) they interpret others' actions in terms of goals, and this induces the same kinds of imitative errors as it does in typically developing children.

A number of further studies support this view. For example, the neuroimaging studies on imitation in autism that found abnormal activation of the mirror neuron system

(described in section 1), reported no differences between the abilities of the ASD individuals and the control participants in imitating either facial expressions (Dapretto et al., 2006; Nishitani et al., 2004) or meaningless finger movements (Williams et al., 2006). In fact, one study even suggests an enhanced tendency to imitate in individuals with autism. Bird and colleagues have recently employed an 'automatic' imitation paradigm in subjects with ASD (Bird et al., in press). The term 'automatic' imitation is used to describe the phenomenon that people's motor movements are facilitated by observing the same movement in someone else and impaired when they observe a different movement of the same body part (Brass, Bekkering, & Prinz, 2001; Heyes, Bird, Johnson, & Haggard, 2005). This effect appears to be weaker or absent when the observed action is performed by a non-human actor, like a robot (e.g., Press, Bird, Flach, & Heyes, 2005). In the study by Bird and colleagues, young adults with ASD were instructed to open or close their hand whenever the hand they were watching on the screen began to move. Sometimes the observed hand would open and sometimes it would close. When the participants were instructed to open their hand whenever they saw movement, their responses, just like the ones of individuals without ASD, were slower when the observed hand was closing, and faster when it was opening. This demonstrates that people with ASD are also subject to automatic imitation tendencies. Even more interestingly, individuals with ASD displayed higher specificity of automatic imitation to a human actor compared to a robot, than the control subjects. Since, like the mirror neurons of monkeys, the human mirror system is considered to be tuned to biological actions only (Tai et al., 2004), the especially attenuated effect of the robot hand action compared with the human hand action in individuals with autism would be unlikely if they possessed a dysfunctional mirror neuron system.

Many authors make a distinction between imitation as an automatic process and imitation as a cognitively mediated mechanism for social learning (e.g., Byrne, 2005), or

between emulation and mimicry (Hamilton, in press). Automatic imitation or mimicry is evident in the involuntary and unconscious matching of posture, gestures and prosody between individuals (Chartrand & Bargh, 1999), and probably serves to facilitate social functions (Decety & Chaminade, 2003) by generating empathy or mutual identification (Byrne, 2005). Automatic imitation also manifests as response facilitation on a number of experimental tasks (Brass et al., 2000). On the other hand, imitation that serves learning seems to involve more complex processes, like identifying which elements are relevant to the accomplishment of the observed skill and which are incidental or idiosyncratic to the demonstrator. Some authors have suggested that it is only the former, more automatic type of imitation that is likely to be subserved by a process of direct matching (Byrne, 2005). Perhaps then, individuals with ASD might be expected to display a deficit of automatic imitation, but not necessarily of voluntary imitation.

In fact, a recent paper by McIntosh et al. (2006) did find a difference between automatic and voluntary imitation in autism. Participants viewed pictures of faces with happy or angry expressions while the activation of their facial muscles was measured. The authors found that, unlike in control subjects, individuals with ASD showed no automatic activation of the muscles associated with performing the expression they observed. Nevertheless, all participants performed well on the task of voluntary imitation, in which they were asked to copy the expression that they saw. However, the idea of a specific deficit in automatic imitation in autism seems to be incompatible with other findings. For example, the imitation impairment observed in the automatic imitation paradigm is higher in those with autism than in controls (Bird et al., submitted). Furthermore, the high instance of echolalia and echopraxia, the excessive vocal and motor imitation of what you hear or see (Fay & Hatch, 1965; Lord, Rutter, & LeCouteur, 1994), could be seen as incompatible with an automatic imitation deficit (Griffin, 2006). Children with autism are also able to recognize when they

themselves are being imitated (Tiegerman & Primavera, 1994; Field et al., 2001), which, according to Byrne (2005), is precisely the kind of ability that would be expected to be subserved by the MNS.

In sum, many studies, including some by the proponents of the 'dysfunctional MNS' hypothesis (Dapretto et al., 2006; Williams et al., 2006), suggest that individuals with autism are capable of voluntary imitation. As for automatic imitation, the picture is less clear. Some symptoms of autism, as well as some experimental studies, suggest that automatic imitation is not absent, but even enhanced in ASD. Other findings show a lack of automatic imitation of facial expressions in autism. We find the fact that the conflicting reports on automatic imitation in autism come from studies investigating the imitation of different kinds of actions (i.e., hand actions vs. facial expressions) informative. We will return to this potentially interesting distinction later in the chapter.

2.3. The mirror neuron system and human imitation

Having reviewed literature showing that neither the mirror neuron system nor imitation is specifically dysfunctional in autism, we now turn to the question of whether it is plausible to assume that human imitation is based on a direct-matching mechanism implemented in the mirror neuron system. Imitation, as is evident from a perusal of the many studies on infant imitation, appears to go beyond direct motor matching. Infants are not blind imitators, as evidenced by the selective nature of their action reproduction. By examining what human infants imitate, we can evaluate whether or not the predictions made by the direct-matching hypothesis are actually borne out.

The hypothesis that the MNS implements the basic neural mechanism that enables the direct transformation of perceptual information into motor commands that lead to imitation is an attractive proposal because it offers a plausible solution to the problem of

how an appropriate mapping is created between the body of the demonstrator and the imitator (Nehavniv & Dautenhahn, 2002). According to Rizzolatti and Craighero (2004), two types of newly acquired behaviours are based on imitation learning, and therefore subserved by the mirror neuron system. The first type of learning is 'substitution', in which a pre-existing motor pattern is substituted for a newly observed motor pattern that is better suited to the task. The second type of learning is the acquisition of a new motor sequence, which involves the decomposition of an observed motor pattern into elementary motor acts already in the observer's repertoire. These 'elementary' motor acts have been proposed to activate the corresponding motor representations in mirror areas.

These proposed functions of the mirror neuron system make several predictions concerning the imitative performance of naïve observers. First, Rizzolatti and Craighero (2004) suggest that 'substitution', as a form of imitation learning, occurs when the new action is recognized as being better suited to fulfil the goal of the action. This predicts that a less efficient means of achieving an outcome should never be substituted for a more efficient means. Second, the decomposition-recomposition model proposed by Rizzolatti and Craighero (2004) predicts that imitation should result in actions that bear a high degree of motor resemblance to that of the demonstrator. Finally, the direct-matching hypothesis makes no explicit claims concerning how an observer decides *what* to imitate. However, a number of researchers have highlighted the 'goal-directed' nature of imitation (e.g., Bekkering et al., 2000), which has been proposed to be driven by the MNS (Wohlschlager & Bekkering, 2002). Below we present evidence from studies of imitation in infancy that are difficult to reconcile with these predictions.

2.3.1. Action substitution and efficiency

Rizzolatti and Craighero (2004) proposed that imitation learning may take the form of substitution of an old motor pattern for a new behaviour (that already exists in the motor repertoire of the individual), should the new motor behaviour provide a more adequate means to the goal. This proposal presupposes that the observer has some way to evaluate whether the observed action is a better way of fulfilling the task than the action that the observer was previously using.

In a classic studies of infant imitation, Meltzoff (1988) asked whether infants of 14 months would imitate a novel act one week after seeing it. The novel act that the infant watched was an experimenter using his forehead to illuminate a lightbox. Meltzoff found that the infants who had witnessed the head-touch action also used their own heads to illuminate the box, while infants in a control group who had not witnessed the head-touch action never performed this new action. Clearly, using one's head to press a box is a less 'appropriate' or efficient means than simply using one's hands. However, in a replication of this study, Gergely and colleagues (2002) found that most infants used their hands to press the box *before* they imitated the head-touch, demonstrating that they were aware of the availability and efficiency of this action (Gergely & Csibra, 2005; 2006). The fact that they went on and imitated the less efficient means is at odds with the idea that imitative learning is used for substituting less efficient actions with more efficient ones. Further evidence confirmed that 14-month-old infants do know that the inefficient head action is not the most appropriate action for the task. In the Gergely et al. version of the task, infants either saw the head action carried out by someone whose hands were visibly free to perform the task, or someone whose hands were covered by a blanket and were not available (Gergely et al., 2002). In this study, the 14-month-olds who were in the condition where the demonstrator's hands were occupied did not perform the head action themselves, whereas

those infants, who saw the demonstrator with their hands free, did imitate the head action. This suggests that infants were aware that the hands were more appropriate effectors to fulfil the task.

Other studies confirm that young children imitate unnecessary or inefficient actions, when they are demonstrated by an adult. For example, Nagell et al. (1993) found that, while chimpanzees would not imitate a less efficient goal-directed action, young children would copy whatever action was demonstrated to them, irrespective of its physical efficiency. In another study by Horner and Whiten (2006), 3-year-old children imitated an action that they could clearly see was causally unrelated to the goal, and appeared to prefer to model the demonstrator at the expense of efficiency. Children imitate inefficient and unnecessary actions even when their attention is specifically drawn to the fact that some observed actions may be unnecessary (Lyons et al., 2007) and this is especially true when the causally unnecessary action is socially cued by the experimenter (Brugger et al., 2007). The fact that children imitate these inefficient actions even in the absence of the experimenter (Gergely et al., in preparation; Horner & Whiten, 2005) suggests that they are not performing them in order to fulfil any social function or simply to please the experimenter.

2.3.2. Fidelity of imitation

Actions are hierarchically organized (Jeannerod, 2006), and there are different levels on which one can construe and imitate an observed action (Byrne & Russon, 1998; Csibra, 2007). It is not clear how a direct-matching mechanism alone would enable the interpretation and re-enactment of observed actions at different levels, since the level on which one interprets an action appears to depend on a number of factors, such as the presence or absence of an object (Wohlschlager & Bekkering, 2002). Indeed, it is debatable whether it is ever possible for an observer to imitate someone else's actions completely

faithfully since her body will never have the capacity to reconstruct exactly what has been done by the demonstrator (Csibra, 2007).

According to Rizzolatti and Craighero (2004), the mechanism by which new motor acts are incorporated into the motor repertoire for reproduction involves a process of decomposing the observed action into known motor acts, and then recomposing these broken-down components into a new behaviour that the individual can then perform. Since this process does not allow for action interpretation at different levels, it should result in a high fidelity action reproduction. The observers' ability to reconstruct the action faithfully will obviously depend on their motor capacity, and it would be expected that faithful motor reproduction may not be entirely possible in infancy. However, major deviations from the demonstration would not be compatible with MNS-driven imitation learning. It is therefore difficult to explain why, in the various versions of the Meltzoff (1988) study, infants rarely copy exactly what the demonstrator has done (a head-touch using the forehead), but illuminate the lightbox using their mouth, cheek, chin, or ears (G. Gergely's observations). This kind of imitation fits with an interpretation of the demonstrator's action at a higher level of the action hierarchy ('use the head to contact lightbox'), but it is difficult to see how direct matching would allow for such an interpretation of the action (Csibra, 2007).

2.3.3. Goal-directed imitation

According to Iacoboni (2005), the identification of the goal of an observed action by mirror neurons in the premotor cortex dictates what is imitated by the observer. Bekkering and colleagues argue that the object-directed (used synonymously with 'goal-directed') nature of human imitation provides support for the view that imitation is subserved by the MNS. This conclusion was based on the analogy between the finding that mirror neurons in monkeys do not fire unless the observed action is object-directed, and the fact that the presence of

an object facilitates imitation in humans. This analogy led Wohlschlager and Bekkering (2002) to suggest that mirror neuron activity and imitation are mediated by the same system of direct matching. The presence of an explicit goal during imitation indeed does result in higher levels of activity in mirror neuron areas in humans (Koski et al., 2002).

Notwithstanding the difficulty (arising from the proposal that direct-matching is the mechanism by which actions are understood) of how it would be possible to identify the goal of an action that is not yet in one's motor repertoire, there is evidence that imitation in young children and infants is also 'goal-directed'. Bekkering, Wohlschlager, and Gattis (2000) showed that 3 to 5-year-old children tended to ignore the particular hand used by the demonstrator when her action was directed towards a target object, but imitated using the correct hand when no 'goal' object was present. Recently, Carpenter, Call, and Tomasello (2005) have reported a similar finding in 12- and 18-month-old infants. Infants in one condition were shown a toy mouse either hopping or sliding towards a toy house, whereas in another condition infants just saw the demonstrator performing the particular action (hopping or sliding) but with no house present. In the first group, both 12- and 18-month-olds tended to emulate the action of putting the mouse in the house, but neglected to imitate the particular manner in which this was done. However, when no house was present, infants tended to imitate the particular action (hopping or sliding) significantly more.

In a recent study however, we found evidence that infant imitation is not always goal-directed, but is dependent on a sophisticated interpretation of the communicative intent of the demonstrator. Gergely and Csibra (2006) have proposed that imitation in human children is facilitated and modulated by the presence of ostensive communication cues (e.g., eye contact), which trigger in the recipient the assumption that the demonstrator is going to demonstrate some *new and relevant* information for them. On this basis, we

hypothesised that infants would select action elements for imitation on the basis of their communicative novelty rather than relying exclusively on a hierarchical analysis of goals. We presented 18-month-old infants with a variation of the Carpenter et al. (2005) paradigm. Half of the infants received the exact same demonstration as infants in the house-present version of that study, while the other half were first told and shown that the toy animal lived in the house (thus rendering the house aspect of the demonstration 'old') before they were shown the same demonstration as the other group (the animal either hopping or sliding into the house). Infants in this second group imitated the details of the action (hopping or sliding) significantly more than infants in the group where all the information was new, and interestingly, in a non-negligible number of trials, infants even neglected to put the animal in the house at all, in favour of imitating the style of the action modelled by the demonstrator (Southgate, Chevallier, & Csibra, under review). This finding suggests that imitation is more than identifying and re-enacting the goal of the demonstrator.

3. An alternative hypothesis for the connection between ASD and imitation

The above study highlights one of the shortfalls of the direct-matching theory of imitation and imitative learning. Although direct matching may offer a plausible solution to the correspondence problem (Heyes and Bird, in press), it cannot tell the observer *what* to imitate. Since much evidence shows that human imitation is a selective process, additional mechanisms are required to account for imitation. In this section, we propose that *communication* plays a key role in human imitation, and this also provides an explanation for the apparent imitation deficit, as well as atypical patterns of MNS activation, in autism.

3.1. Imitation and communication

The term 'imitation' refers to the phenomenon of the reproduction of some behaviour of an individual by another individual. However, some theorists think about imitation not only as a phenomenon but also as a special mechanism that underlies the reproduction of observed behaviours. Others, like Heyes and Bird (in press), proposed that there is no special mechanism of imitation but it is achieved by simple associative mechanisms. While we agree that imitation is not accomplished by specifically dedicated mechanisms and that action reproduction is sometimes based on associative mechanisms, we think that most instances of imitation are actually achieved by the process of *emulation* (Csibra, 2007).

The term 'emulation' in this context refers to a mechanism that reproduces an action from its description. An action can be described at many levels of precision and resolution, and action descriptions created for the purpose of reproduction usually involve a reference to the outcome of the action. For example, the action to be reproduced in the task that demonstrated 'goal-directed imitation' in children (Bekkering et al., 2000) can be described as "touch the point", or as "touch the point with your right hand", or as "touch the point with your right finger", or as "touch the point with the palmar surface of your right finger", each successive description specifying the end of the action with more and more precision. The temporal or sequential aspect of an action can also be specified at various levels of precision, describing details of the subgoals (e.g., path of the hand movement) through which the end goal should be achieved. The process of emulation generates the action simply by feeding these goal specifications into the observer's own motor system, which will achieve them in its own way that will not necessarily match the details of the observed behaviour. Thus, the fidelity of emulative action reproduction depends on what level of precision is chosen to interpret and reconstruct the observed action.

The crucial question that an imitator is confronted with when observing a model is *what* to imitate, i.e., what level of action interpretation and what precision of emulative action reproduction she is expected to perform. Studies on 'goal-directed imitation' demonstrate that children, including children with autism, tend to interpret and emulate the to-be-imitated actions at a higher level of goal descriptions when these are supported by factors like the availability of target objects. Such a behaviour is adaptive in many social learning situations, because the minute motor details of action execution are normally irrelevant with respect to goal achievement. However, human cultural practices and norms tend to be *opaque* in the sense that often neither their purpose nor the causal relations between the performed actions and their useful outcome are evident for a naïve observer (Gergely & Csibra, 2006). For example, humans engage in tool making for which, to an observer, there may appear no immediate goal at the time of construction, and perform rituals that do not reveal how they are supposed to work. If much of human culture consists of such cognitively opaque practices, it would make little sense for imitation to be driven solely by the identification of goals, as in many cases there will be no obvious goal, but there may nevertheless be important actions worthy of imitation and learning. To cope with the problem of cognitive opacity, Gergely & Csibra (2005, 2006) have recently proposed that humans evolved a suite of adaptations to ensure that cultural knowledge is efficiently transferred across generations.

The key element of this proposal is that communication from the model towards the observer can help to identify what is the relevant aspect of the modelled behaviour to be learnt, hence what level of emulation the imitator is expected to perform. The necessary adaptations for such a communication system involve many elements, of which the most important in this context is that the observer has to be sensitive to the cues that signal the model's intention to communicate. Human infants show an early, or even innate, sensitivity

to such *ostensive cues*, which trigger an expectation of relevant content to be communicated by the source of these cues (Csibra & Gergely, 2006). In the context of action demonstration, these cues will tell children that the model's behaviour will reveal some relevant information for them to be learnt and reproduced.

The implication of this theory is that an observer needs to be sensitive to, and to correctly interpret, the ostensive communication cues that accompany demonstrations in order to benefit from the model's pedagogical efforts. Without this one would not be able to select the relevant aspects of another person's behaviour that are important to attend to and reproduce. We propose that individuals with autism lack this sensitivity to ostensive cues and the expectation of relevant information to be manifested, which could account for the reported impairments in imitation and a host of other phenomena.

3.2. Understanding communicative intent is impaired in autism

From their earliest utterances, human infants appear to abide by Gricean maxims (Grice, 1975) and tailor what they say to their partner's knowledge state (Greenfield & Smith, 1976). However, even high-functioning adults with autism do not find this an easy task, often neglecting to include details that meet the intended recipient's communicative needs (Bruner & Feldman, 1993). Unlike typically developing children, who begin to use pointing around 9 to 12 months of age, children with autism rarely point (Baron-Cohen, 1989). The sophisticated communicative understanding underlying the pointing behaviour of 12-month-old infants, discussed by Tomasello and colleagues (Tomasello, Carpenter, & Liszkowski, 2007), suggests that the absence of pointing in young children with autism may arise from a lack of understanding of communication.

On the comprehension side, children with autism also seem to be unresponsive to the communicative cues produced by others. For example, in response to others' points,

children with autism often look at the hand that is pointing rather than the object being pointed at, suggesting that they lack the understanding that pointing is a communicative, referential act. Similarly, children with autism fail to use gaze to locate a hidden object (Leekam, Lopez, & Moore, 2000), a task that is trivially easy for typically developing children (Behne, Carpenter, & Tomasello, 2005). Furthermore, children with autism do not show the typical reactions to a number of ostensive cues that would enable them to learn from others. Typically developing newborns show a strong preference for faces (Johnson et al., 1991) and, within the face, for direct gaze over averted gaze (Farroni et al., 2002). Human infants' attention automatically shifts to the direction of others' perceived gaze shifts (the gaze-cueing effect that is well-known in adults, see Driver et al., 1999), but only if the movement of the eyes is preceded by direct gaze (Farroni, Mansfield, Lai, & Johnson, 2003). Autistic children on the other hand, do not preferentially attend to faces (Osterling & Dawson, 1994) or the eyes (Klin et al., 2002), and the gaze cueing effect appears to be absent, or at least divergent (Johnson et al., 2005; Senju, Tojo, Dairoku, & Hasegawa, 2004). While direct gaze cues attention better than a non-social cue in typically developing children, the same differential effect is not present in children with autism (Senju et al., 2004), and it does not facilitate face detection (Senju et al., 2003). Typically developing neonates show a preference for infant-directed speech (Cooper & Aslin, 1990), a pattern of exaggerated prosody that may serve to elicit infants' attention (Fernald & Simon, 1984), and is proposed to serve as an auditory ostensive stimulus for infants (Csibra & Gergely, 2006). Human neonates also prefer to listen to speech rather than a non-speech analogue (Vouloumanos & Werker, 2007). Children with autism, however, do not prefer infant-directed speech (Kuhl et al., 2005) and do not even show a preference for their mothers voice over the noise of a busy canteen (Klin, 1991). Finally, a failure to respond to the sound

of their own name appears to be one of the earliest observable indications of autism (Nadig et al., 2007; Werner, Dawson, Osterling, & Nuhad, 2000).

If it is the case that imitation is driven by a sensitivity to the communicative intent of the demonstrator, and an impairment in understanding communicative intent inflicts individuals with autism (Sabbagh, 1999), then imitation should be an expected impairment in those with ASD. This impairment may result in inappropriate imitation, but may manifest as either too little imitation (as is often reported) or reproducing unnecessary aspects of another person's behaviour. For example, in studies where there is no instruction to imitate, researchers find that individuals with ASD either fail to imitate, or imitate less than typically developing children (e.g., Brown & Whiten, 1999). Since parents rarely explicitly instruct their children to 'do as I do', this may explain the lack of spontaneous imitation in children with autism in their every day lives. However, as it is evident from recent studies, when instructed to imitate, children with autism imitate as well as non-affected children (Dapretto et al., 2006; Hamilton et al., 2007). Nevertheless, even instruction to imitate would not always result in the same level of imitative competence in individuals with ASD as in typical individuals. This is because telling someone to 'do as I do' does not specify on what level he is supposed to emulate the demonstration (Bird et al., in press). As we have argued above, a sensitivity to the subtle cues in the communication of the other individual is necessary to extract from the demonstration the relevant level of action reproduction.

Gergely and colleagues' study on infants' selective imitation (discussed above) could illustrate how communication cues and expectation of relevance drive children's reproduction of observed actions (Gergely et al., 2002). If infants expect relevant information, they should be sensitive to the relative amount of information inherent in various elements of the demonstration. The amount of information is dependent on the conditional probability of an event (the less probable it is, the more information it carries).

Touching the lightbox with the forehead is an unlikely event when the model's hands are free, and thus will be judged as the most informative part, and the most likely content, of the demonstration. In contrast, touching the lightbox with the head is not as unlikely when someone's hands are occupied. In this case, the information that the box can be lit up will be a more informative element of the demonstration than the mode of pressing the box. Thus, 14-month-old infants interpreted the action to be reproduced at the level of the overall goal (lighting up the box) in the hands-occupied condition, but went down to the level of effector (lighting up the box by head-touch) in the hands-free condition.

A further version of this study confirmed that the selective imitation effect in this task was dependent on the ostensive communication cues that could induce the expectation of high relevance in infants. In this version, infants observed the same demonstration, but now the model performed the head-touch actions without ever looking at the infant or emitting any communicative cues. In this situation, infants were less likely to reproduce the head-touch action and, more importantly, the selectivity of imitation disappeared: they touched the box with their head in the hands-occupied condition as often as in the hands-free condition (Kiraly et al., 2004). Recently, a version of this study was also run on a sample of children with autism (Somogyi et al., 2006). Although they received as many ostensive communicative cues as children in the original version of this study (Gergely et al., 2002), they behaved in a similar way to typically developing infants in the non-ostensive condition. Like in a number of studies reported here, the children with autism imitated the head-touch action at a level comparable with typically developing children (around 70%) in the hands-free condition, but crucially, the level of imitation was equivalent in both the hands-free and hands-occupied conditions in autistic children. This suggests that children with autism are unable to modulate the interpretation of the demonstration on the basis of communicative cues and do not expect that the model's manifestation will reveal relevant information for

them. It is thus not the ability to copy an action, but the ability to extract the relevant level of emulation from a communicative demonstration that may be impaired in autism.

This conclusion is consistent with other symptoms of the disorder. If one of the core deficits of autism is the lack of sensitivity to communicative intent, any aspect of cultural knowledge that is transmitted via communication could be expected to be impaired in autism. For example, knowledge about social norms and conventions is learnt exclusively from other individuals of our species, and so may be impaired in autism. In fact, there are reports that this is indeed the case (e.g., Loth, 2007).

3.3. The mirror neuron system in autism

We have argued that, while the ability to imitate may not be especially impaired in autism, the atypical pattern of their imitative behaviour can be derived from their insensitivity to communicative cues, and communicative intent. In this sense, imitation impairment may not be a core deficit, but rather a symptom of autism; not a cause but a consequence of the disorder. This proposal leaves open the question of how to explain the findings of differential functioning of the mirror neuron system in autism, which we reviewed at the beginning of this chapter.

First, when considering mirror neuron system activation in response to the observation of hand actions, the findings are not easy to interpret. Oberman and colleagues (2005) did not find mu rhythm suppression in individuals with autism in response to the observation of hand actions. What is surprising, however, is that the MNS of their control subjects responded to the observation of non-transitive, non-object-directed hand actions. Other studies on mu rhythm suppression (Muthukumaraswamy, Johnson, & McNair, 2004) and neuromagnetic activation of the mirror neuron system (Nishitani & Hari, 2000) reported hardly any MNS activation modulation unless the hand approached or manipulated

an object. The findings of Théoret et al. (2006) are also difficult to interpret. They recorded no motor activation when their subjects with ASD saw a hand from their own point of view (as if it was their own hand), but found normal activation when they saw a hand facing the subject (as if it was someone else's hand). This is an unexpected finding and would more likely suggest an impairment in self-body image than in social mirroring. Hobson and Meyer (2005) have, in fact, reported that children with autism, unlike typically developing controls, did not use their own body to indicate to an experimenter where to place a sticker, instead pointing to the experimenter's body, which suggests an impairment of self-body image. Finally, the study by Williams et al. (2006) failed to find inferior frontal activation in adolescent individuals with autism during the imitation or observation of finger movements. However, they did not find such activation in their control subjects either. The authors reported that some parietal regions, considered to be a part of the MNS, are less activated in ASD than in the control subjects during imitation. However, this was also true during action execution, while there was no difference found during action observation. It is thus not clear why this activation difference should be considered to reflect the mirror neuron system. This lack of activation during action execution was also found in a recent study by Cattaneo and colleagues. They used electromyography to record the activity of the mouth-opening mylohyoid muscle when subjects had to either grasp an object for eating, or for placing in a container. They found that while the mylohyoid muscle in typically-developing children showed activity from the point where the subject began to reach for the object, the same muscle in a group of children with autism was only activated later as they brought the object to their mouth. Typically developing children also showed this anticipatory muscle activity when watching someone else reach for an object they were going to eat, but children with autism did not (Cattaneo et al., 2007). Rather than pointing to a specific impairment of the MNS, this result suggests that children with autism have impairments in

their ability to sequence their own actions. In fact, difficulties in motor planning in children with autism are well documented (e.g., Hughes, 1996). As mirroring is, by definition, dependent on one's own action capabilities, any impairment in the production of actions would be expected to lead to impairments in mirroring, but not because there is anything impaired in the mirroring mechanism.

The evidence for atypical MNS activation in autism during observation or imitation of facial actions is also ambiguous. Dapretto and colleagues (2006), using a task of emotional expression imitation or observation, reported that individuals with autism, unlike typically developing children, showed no activation of the inferior frontal gyrus (IFG). Others, however, have failed to replicate these results. For example, Ashwin et al. (2007) found no activation difference in the IFG during an emotional face perception task between a group of individuals with ASD and control subjects. Even if the reduced MNS activation to facial expressions in autism were reliable, it does not necessarily indicate a causal role of motor mirroring in understanding emotional expressions. An alternative explanation for this effect could be that it is the result, rather than the basis, of emotion understanding. Facial expressions are inherently communicative (Fridlund, 1994), and so it is not surprising that autism, whose primary symptoms include disordered communication, affects the comprehension of emotional expressions as well. It is well known that individuals with autism do not spend as much time looking at faces, and especially looking at the eye regions, as non-affected people (Gliga & Csibra, 2007), and their relative inexperience with faces could also contribute to the failure, or slower speed, of recognizing facial expressions. Such an impairment would also make it less likely that individuals with ASD would generate the appropriate facial expression in response to the perception of an emotional signal, which could explain the reduced activity of, for example, appropriate facial muscles during

observation of emotional expressions (McIntosh et al., 2006). However, this may be the result, rather than a cause, of impaired understanding of emotions.

Translating this account into neurological terms, it is possible that the lack of IFG activation observed in children with autism results from dysfunction of the amygdala, which has reciprocal connections with IFG. An abnormal amygdala has been given a central role in autism (Schultz, Romanski, & Tsatsanis, 2000), and has been proposed to underlie difficulties in emotion recognition (Baron-Cohen et al., 2000). Indeed, numerous recent studies have reported anatomical abnormalities in the amygdalae of individuals with autism (e.g., Aylward et al., 1999; Munson et al., 2006). Functional studies also found reduced amygdala activations in autism. When asked to judge the emotion conveyed by the eyes, non-affected individuals activated their amygdala whereas the ASD subjects did not, and performed significantly worse on the task than the controls (Baron-Cohen et al., 1999). Ashwin et al. (2007), who did not find activation difference in the MNS, also reported reduced amygdala activation in people with ASD in a facial expression perception task. Even Williams et al. (2006), who studied observation and imitation of hand movements, found significantly reduced amygdala activation in their ASD subjects.

One possibility then is that the additional IFG activation evident in control subjects (and lacking in ASD subjects) in the Dapretto et al. (2006) study stems from an automatic process of emotion identification. Hypoactivation of the amygdala, known to be involved in emotion recognition, could lead to differences in the activation of the mirror neuron system by virtue of the connections between the two, but this would not necessarily imply that the mirror neuron system itself was dysfunctional. Whether or not this was a plausible explanation for this result is difficult to judge because Dapretto et al. (2006) reported in their results that there was no difference in amygdala activation between the groups, but

their data reveals significantly lower amygdala activation in the ASD group compared with the typically developing children (Supplementary Table 3).

The amygdala is not the only brain region that is affected in autism. Consistently with the proposal that autism is a primarily communicative disorder, ASD individuals also show differences in activation of the medial frontal cortex, an area proposed to play an important role in recognizing the communicative intent of others (Amodio & Frith, 2006; Kampe, Frith, & Frith, 2003). For example, Wang and colleagues found that whereas typically developing children showed increased activation of the medial prefrontal cortex when interpreting the intended meaning of an ironic scenario, children with autism did not, and were less accurate than controls in detecting the communicative intent behind the remark (Wang, Lee, Sigman, & Dapretto, 2006). Facial emotional expressions also fail to activate medial prefrontal areas in autism (Ashwin et al., 2007).

While we propose that, on the cognitive level, the imitation deficit in autism could be satisfactorily explained by the communicative impairment that is undoubtedly implicated in the disorder, we are not committed to any particular theory about the neural bases of this impairment. Nevertheless, we think that the available evidence on the MNS dysfunction in autism is too ambiguous to support the 'broken mirror' hypothesis, and alternative proposals, like the ones that emphasize the role of the amygdala or the medial prefrontal cortices, or even impairments in the individual's own motor capabilities, are not less compatible with recent findings in neuroimaging.

4. Conclusions

We have argued that the numerous studies reporting intact imitative abilities in individuals with autism, both voluntary (e.g., Dapretto et al., 2006; Hamilton et al., 2007; Somogyi et al., 2006) and automatic (Bird et al., in press), do not fit with the view that their mirror neuron

system is impaired. We have presented evidence from infants and children which suggests that imitation goes far beyond a process of direct matching, entailing a sophisticated analysis of the communicative intent of the demonstrator. While it is an open question whether a direct matching mechanism enables or facilitates imitation, it cannot account for the selective nature of imitation reported here. Since individuals with autism can imitate (even if they need to be told to do so), and their imitation impairments appear to arise at the level of selecting when and what to imitate, it seems unlikely that a dysfunctional mirror neuron system underlies the social difficulties they face.

Nevertheless, atypical patterns of activation in the MNS of individuals with autism are to be expected as the result of other deficits (like impaired communication or motor sequencing) that would have consequences for normal MNS functioning, and could explain the controversial findings reported in the literature. Individuals with ASD are less interested and less engaged in social interactions, are less willing to cooperate or communicate with others, and have less experience with dealing with social stimuli. It is thus not surprising that their mirror neuron system, whose main function probably involves making social interactions smooth (Csibra, 2007), does not show the same patterns of activation as the mirror neuron system of unaffected individuals.

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