Opinion

Cell PRESS

Unbroken mirrors: challenging a theory of Autism

Victoria Southgate[1] and Antonia F. de C. Hamilton[2]

[1] Centre for Brain and Cognitive Development, School of Psychology, Birkbeck College, London, WC1E 7HX, UK
[2] School of Psychology, University of Nottingham, Nottingham, NG7 2RD, UK

The 'broken mirror' theory of autism has received considerable attention far beyond the scientific community. This theory proposes that the varied social–cognitive difficulties characteristic of autism could be explained by dysfunction of the mirror neuron system, thought to play a role in imitation. We examine this theory and argue that explaining typical imitation behavior, and the failure to imitate in autism, requires much more than the mirror neuron system. Furthermore, evidence for the role of the mirror neuron system in autism is weak. We suggest the broken mirror theory of autism is premature and that better cognitive models of social behavior within and beyond the mirror neuron system are required to understand the causes of poor social interaction in autism.

Introduction

Individuals with autism have great difficulty with many aspects of social interaction, but the cause of this disability remains unknown. In recent years the discovery of a mirror neuron system (MNS) in the human brain, made up of regions that respond to the actions of self and other (see Figure 1), has led to an increased interest in the brain systems that underlie basic social processes. The primary function of the MNS is proposed to be related to action interpretation [1], but it also has been implicated in other social–cognitive processes, including imitation [2], theory of mind [3], language [4] and empathy [5]. The broad range of social–cognitive functions attributed to the MNS overlaps to some extent with the various social–cognitive difficulties seen in autism. Thus, there is an intuitive appeal in linking these phenomena and in developing a unified neurocognitive theory of autism. Such a 'broken mirror' model has been suggested in several guises in recent years [6–8]. The present paper will examine the data and theories supporting the broken mirror hypothesis of autism and finds several reasons to be cautious of it.

The broken mirror theory

The broken mirror theory of autism has its origins in studies of imitation behavior [6]. Several influential studies suggest a role for mirror neuron regions in hand-action imitation [2,9], and there is also evidence for some degree of imitation deficit in children with autism [10]. On this basis it is argued that dysfunction of the MNS could be the cause of such impaired imitation [6,8]. This model, thus, implies a three-way relationship between a brain system (the MNS), a behavior (imitation) and a disorder (autism) (Figure 2). Some versions of the broken mirror theory take a broader scope than imitation [7] and, drawing on speculations about MNS contributions to empathy [11], theory of mind [3] and language [4], propose that damage to the MNS could cause problems in all these areas [7]. However, in this opinion paper we focus primarily on the evidence for imitation as a key link between the MNS and autism because it is the only social–cognitive ability for which there is both evidence of MNS involvement [2] and some documented deficits in autism [10]

We will first examine the theory and evidence linking mirroring regions of the brain to imitation (Figure 2, arrow a), and imitation to autism (Figure 2, arrow b). We then consider whether these and other studies support a link between mirror neurons and autism (Figure 2, arrow c) and the idea that damage to the MNS should cause specific deficits in social abilities.

Imitation and the MNS

The discovery of neurons in monkeys that respond to both performed and observed actions [1] has led to the identification of a MNS in the human brain (Figure 1), which appears to play a role in imitation. Observation of actions for imitation elicits greater activation in MNS regions than observation without instruction to imitate [2,9,12]. Furthermore, both temporary [13] and permanent lesions [14] to MNS areas cause difficulty with imitation of some actions. Nonetheless, despite the implied involvement of the MNS in imitation, there are at least two reasons for being cautious of the proposal that impaired imitation in autism must stem from a dysfunctional MNS. First, successful imitation goes beyond simply matching actions across bodies, and second, a dysfunctional MNS should be expected to manifest in problems other than imitation.

Imitation is more than mirroring

Some recent models of imitation describe a process limited to the direct transformation of visual information into motor output [2,9,15]. In postulating a dysfunctional MNS as the cause of impaired imitation, broken mirror theorists implicitly endorse this direct mapping. However, successful imitation is not only the matching of corresponding actions from one body to another but also requires several different cognitive processes. These include visual analysis, representation of action goals, selection of what and when to imitate and motor control (Figure 3). Different

Corresponding author: Southgate, V. (v.southgate@bbk.ac.uk).

1364-6613/$ – see front matter doi:10.1016/j.tics.2008.03.005 Available online xxxxxx

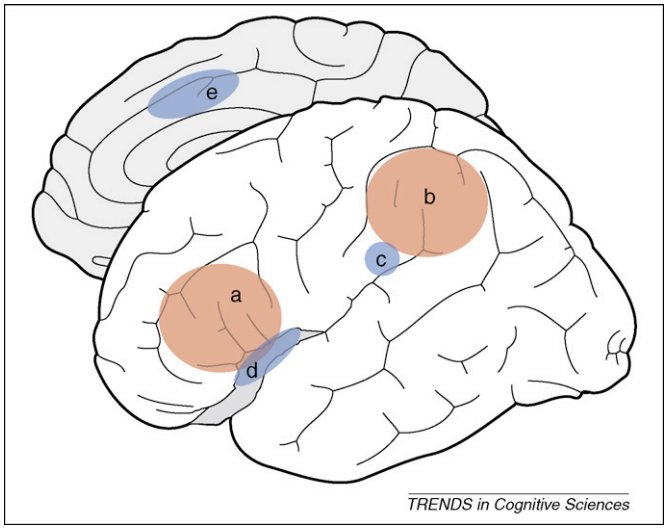

Figure 1. Mirror regions of the human brain. The core of the mirror neuron system is located in the inferior frontal **(a)** and inferior parietal **(b)** regions of the human brain. These areas are the human equivalent of regions F5 and inferior parietal lobule in the macaque brain, where mirror neurons have been reported [1], and several fMRI studies have demonstrated that these areas respond during performance, observation and imitation of hand actions [1]. In the current paper discussion of the MNS refers to this core system. Some recent data suggest that our concept of a mirror system should be extended to encompass brain regions that respond to a wider range of stimuli for both self and other (blue regions). For example, the secondary somatosensory cortex **(c)** responds to performed and observed touch, whereas the anterior insula **(d)** and anterior cingulate **(e)** cortex respond when a person experiences, observes or imitates emotional facial expressions (reviewed in [49]). It is possible that future work will identify more brain regions with mirroring properties. However, it is unlikely that all these different brain regions function as a single mirror neuron system. Rather, there might be several mirroring systems that could be differentially involved in different social processes and differentially impaired or intact in autism.

imitation tasks might require many types of action representation [16] and varying degrees of action selection [17,18] (Box 1). For example, as described in Box 1, young children evaluate the rationality of an action in selecting whether or not it should be imitated, but this evaluation does not involve the MNS [19].

Furthermore, current data do not support proposals about how the MNS might enable imitation learning [20]. One proposed type of imitation learning involves substituting a new and more efficient means for an old motor act [1]. However, young children sometimes choose to replace an efficient action with an inefficient action [17] or imitate actions that they know to be causally unrelated to the desired outcome [18,21]. Such behaviors are difficult

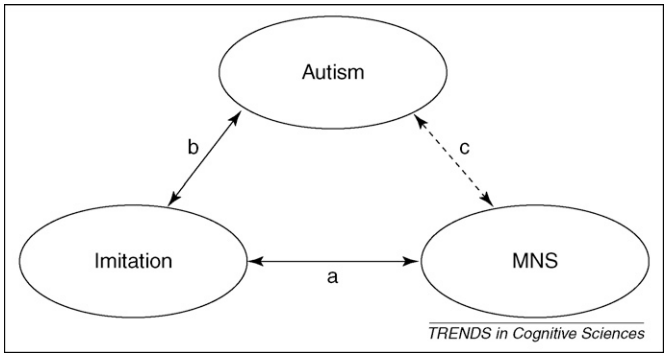

Figure 2. Conceptual links inherent in the broken mirror theory. The theory requires links between a behavior (imitation), a brain system (the MNS) and a disorder (autism). In the current paper we examine the evidence for each link in turn.

Box 1. The selective nature of human imitation

Several studies show that human imitation is modulated by an evaluation of the efficiency of the observed action and the presence of communicative cues. 14-month-old infants who watch a model using her head to illuminate a lightbox use their own heads to illuminate the box significantly more often when the model had her hands free when demonstrating the action, than when she had her hands occupied [17] (Figure I). When the model's hands were occupied with a blanket, infants used their hands to illuminate the box, but not their heads. This suggests that in deciding what to imitate, infants evaluated the necessity of the model's action to bring about the outcome. If the model had her hands occupied with the blanket, infants were able to reason that the strange head action was necessitated by her constraints and, because the infants' own hands were free, the head action was not necessary for them to copy. If the model had her hands free, the unjustified head action was inferred to offer some unobservable advantage and, so, copied. Crucially, infants only used their heads to turn on the lightbox if the model ostensively demonstrated the action to the infant. If the model did not communicate with the infant, infants in both the hands-occupied and hands-free conditions imitated the head action to the same degree [33]. Problems either in evaluating the efficiency of actions or in recognizing and interpreting communicative cues could contribute to atypical imitation. A recent study (E.Somogyi *et al.*, unpublished) has found that children with autism do not differentiate the hands-free and hands-occupied conditions and imitate the head action to a high level in both conditions. This suggests that the ability to match actions is intact, but the ability to select actions for imitation might be impaired.

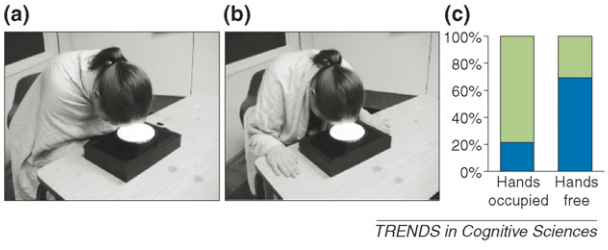

Figure I. Model performing head action with hands occupied (left) and hands free (right). Blue indicates imitation of head action, green indicates use of hands. From Gergely *et al.*, 2002.

to explain by a process of direct matching but can be accounted for if one considers other factors outside the MNS that are likely to influence imitation (Figure 3). If imitation relies on systems beyond the MNS, it is not sensible to assume a straightforward mapping between imitation behavior and the MNS.

The MNS is not for imitation alone

Even those involved in the discovery of mirror neurons and the human MNS agree that the basic function served by the MNS is unlikely to be imitation [1]. For example, macaques have mirror neurons but rarely imitate one another [22], and the human MNS is more active for complementary actions than for imitative actions [23]. Plausible theories suggest the MNS serves the basic function of facilitating action prediction [24,25] or the understanding of action goals [15]. Dysfunction of the MNS should, therefore, impair these abilities, as well as cause poor imitation. In line with this, neuropsychological patients with lesions to MNS regions show poor action interpretation as well as poor imitation [14]. However, the available evidence suggests that individuals with autism do not have difficulties with understanding actions

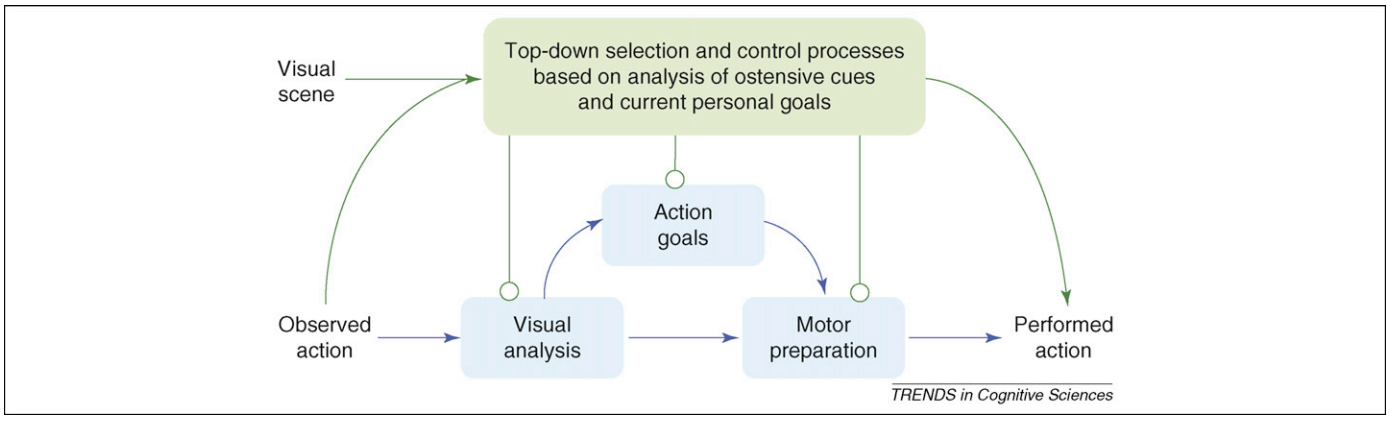

Figure 3. A possible cognitive model of imitation behavior. This figure illustrates some of the cognitive components that might underlie imitation behavior. Previous work [16,26] suggests that successful imitation involves at least three distinct types of representation: visual analysis of the observed action, extraction of a more abstract goal or semantic content and a motor plan for performing the action (blue boxes and lines). These all could be embedded in the core MNS. However, there is increasing evidence that imitation is subject to selection and top-down control processes, for example, based on the ostensive cues given by the observed actor (green boxes and lines) or based on the individual's current motivation. This selection could act at any stage of the imitation process to reduce or enhance imitation behavior. Thus, normal imitation depends on normal processing of communicative and ostensive cues and normal top-down selection and control systems. One or both of these processes might be abnormal in autism [20,26].

[20,26–28]. For example, like typically developing children, children with autism are able to infer the intended goal of a failed action [28]. If the MNS serves the basic function of action interpretation, results such as these suggest that it is not dysfunctional in autism.

Imitation in autism

A second component of the broken mirror hypothesis is the claim that children with autism have a specific deficit in imitating actions. Indeed, a recent meta-analysis concludes that imitation difficulties are a core feature of autism [10]. However, this view has recently been challenged [29]. More importantly, if there is no single cognitive process or brain system underlying imitation behavior, then interpreting the reasons for poor imitation performance in children with autism is not straightforward.

Imitation success and failure in autism

Children with autism often fail imitation tasks [10], in particular, those requiring imitation of meaningless actions or facial expressions, and for which no explicit instructions to imitate are given. This failure is key to the broken mirror hypothesis, but there are several recent findings suggesting intact imitation in autism, which are not easily accommodated. For example, individuals with autism show an enhanced automatic imitation effect [30] and normal interference effects when observing an incompatible action [31]. Furthermore, children with autism can perform a variety of imitation tasks correctly when they are explicitly instructed to imitate [29,32]. These results are not compatible with the broken mirror proposal that children with autism have a fundamental difficulty in matching the actions of self and other.

The fact that children with autism can imitate but tend not to do so without instruction suggests that their difficulties might arise from problems with knowing when and what to imitate [20,26]. Knowing when and what to imitate probably depends on the ability to exploit the social and communicative cues of others [33]. Typically developing children use the presence of social cues to modulate their imitation [18], but there is some evidence that children with autism do not (Box 1). A reduced sensitivity to social cues, including a lack of preference for looking at the eyes, and infant-directed speech [34,35] is well-documented in autism and could quite plausibly lead to atypical imitation behavior [20] (Figure 3).

The MNS and autism

The evidence cited above demonstrates that imitation does not depend solely on the MNS and that the proposed MNS contribution to imitation (matching the actions of self and other) is unlikely to be damaged in autism. Consequently, we suggest that studies of imitation behavior and its relationship to the MNS do not support the broken mirror theory of autism (Figure 2, arrows a and b). However, some data have emerged recently suggesting that neural responses in the MNS of individuals with autism differ from those of control participants. Although these experiments do provide a more direct test of the broken mirror theory (Figure 2, arrow c), unfortunately the available data do not paint a clear picture of MNS activity in autism, being either difficult to interpret or contradictory.

Some of these studies have used indirect measures of neuronal activation in the MNS, including muscle activation [36], excitability of motor cortex [37] or suppression of resting-state rhythms over motor cortex [38]. It is, however, difficult to pinpoint the origin of the atypical activation that these authors attribute to the MNS. For example, reduced resting-state (mu) suppression over sensorimotor cortex in individuals with autism compared with controls could be due to differences in MNS processing [38] but could equally well be due to differences in earlier visual processing. Reduced attention to social stimuli [34], reduced processing of biological motion [39] and differences in more general understanding of complex visual information [40] have all been documented in autism. Because visual systems, in particular those processing biological motion, are a necessary input to the MNS, it is quite plausible that abnormal visual processing in autism could cause abnormal responses within the MNS. Therefore, atypical MNS activity in response to viewing biological

actions should not be automatically attributed to a dysfunctional MNS.

Several studies have also produced contradictory results. One study reports that individuals with autism lack modulation of primary motor–cortical excitability for viewing hand actions from a 'self' perspective (but not 'other' perspective) [37]. However, the conclusion that this might reflect a dysfunctional MNS is incompatible with reports that mirroring phenomena are apparent only for viewing actions attributed to another person [41]. Reported fMRI results are also inconsistent, with one study failing to find activation in the inferior frontal gyrus even in control participants [42], contrary to the study on which it was modeled [2].

Studies that have measured both imitation performance and brain activation in MNS regions should provide the clearest evidence for the broken mirror theory [32,42,43]. However, closer examination suggests these results actually constitute evidence against a specific relationship between MNS activation and imitation in autism. Specifically, these studies report atypical activation of MNS areas in individuals with autism during imitation tasks despite the fact that the imitation behavior of these subjects did not differ from control participants [32,42,43]. This strongly suggests that the atypical activation of MNS areas is not related to imitation ability.

To summarize, the evidence for a direct, causal relationship between the MNS regions of the human brain and the social difficulties seen in autism is, at best, weak. Others have also proposed alternative ways of interpreting this data without appealing to a dysfunctional MNS [44]. We also note that even if a perfect experiment revealed a clear relationship between the activation of MNS regions of the autistic brain and imitation behavior, this would not prove that the MNS activity causes poor social cognition in autism, but would leave open the possibility that a lack of attention to social or communicative cues (originating elsewhere in the brain) could cause abnormal responses in the MNS.

Beyond imitation

The evidence reviewed above describes why studies of imitation do not make a convincing case for a core MNS dysfunction in autism. Nevertheless, the conceptual link between a brain system for basic social information processing and the social problems seen in autism might remain appealing. Several theories now hint at a broader 'principle of mirroring', sometimes expressed as a 'shared manifold' or the 'like-me' hypothesis [11,45]. The neural substrates of this principle of mirroring would presumably include the traditional mirror neuron regions in frontal and parietal cortex but could extend to any brain regions showing overlapping activation for self and other, including those involved in somatosensory or emotional processes (see Figure 1).

It has been argued that the ability to match any representations relating to self and other, instantiated in the extended mirror neuron regions, is absent in autism and is the primary cause of deficits in social cognition [7]. As yet there is no experimental evidence in support of these broader claims, but there are several reasons to be cautious of such an ambitious but underspecified theory. In particular the critique above highlights the danger of attempting to link brain regions directly to behaviors, and the breakdown of behaviors, without considering the different cognitive processes involved. As we have highlighted, the core MNS regions of the brain are likely to support several cognitive processes, and a breakdown in imitation behavior could arise from the failure of several different cognitive systems either within or outside the MNS (see Figure 3 for a possible model). This same principle continues to apply when an extended MNS or a process of 'self–other' mapping is considered. We suggest that the development of a principled and testable model of autism needs to be grounded in an understanding of cognition and how it breaks down [46].

Box 2. Questions for future research

- Can we develop a full cognitive model of imitation behaviors? Such a model needs to take into account data demonstrating the importance of top-down control of imitation and the factors that influence this. Can we delineate these factors and determine how they interact? Is it possible to map these cognitive processes to different brain regions within and beyond the mirror neuron system?
- How does action processing in the MNS relate to other types of social-information processing in other brain structures? It is likely that the MNS interacts with other brain regions involved in social interactions, for example the amygdala, and atypical functioning of any of these structures might have an impact on the MNS. Further research into the cognitive systems underlying social interactions and their instantiation in the brain is needed to understand how the MNS contributes to social cognition.
- What factors help children with autism imitate? Studies suggest that children with autism imitate better with explicit instruction, but can other manipulations also improve performance and would this help the child learn other skills?
- Can we pinpoint a single cognitive or neural cause of autism? The broken mirror theory is the latest of many attempts to isolate a single cause for the heterogeneous differences in social and non-social skills seen in individuals with autism. Does it make sense to pursue this path or is a different approach needed [50]?

Concluding remarks

We have reviewed evidence for links between imitation behavior, the MNS and autism, and find all three sources of potential support for the broken mirror theory are lacking. In particular, it is not yet clear which cognitive components of imitation are supported by a MNS and at which level (e.g. execution, selection) the imitative problems of individuals with autism originate. Other, as yet unanswered questions, have also been raised (Box 2). As such, it is premature to speculate on the involvement of the MNS in autism and, we believe, unwise to promote such a theory in the popular press [47] or to make claims concerning intervention techniques on the basis of such a theory [48].

Acknowledgements

A.F. de C.H is supported by the Economic and Social Research Council grant 061–25–0318 and by a pilot grant from Autism Speaks. V.S. is supported by a European Commission Pathfinder Cooperative Research Grant (CALACEI). We thank Coralie Chevallier, Gergely Csibra, Uta Frith and Dana Samson for helpful comments on the manuscript.

References

1 Rizzolatti, G. and Craighero, L. (2004) The Mirror-neuron system. *Annu. Rev. Neurosci.* 27, 169–192
2 Iacoboni, M. *et al.* (1999) Cortical mechanisms of human imitation. *Science* 286, 2526–2528
3 Gallese, V. and Goldman, A. (1998) Mirror neurons and the simulation theory of mind-reading. *Trends Cogn. Sci.* 2, 493–501
4 Rizzolatti, G. and Arbib, M.A. (1998) Language within our grasp. *Trends Neurosci.* 21, 188–194
5 Iacoboni, M. (2005) Understanding others: imitation, language and empathy. In *Perspectives on Imitation* (Hurley, S. and Chater, N., eds), pp. 77–100, MIT Press
6 Williams, J.H. *et al.* (2001) Imitation, mirror neurons and autism. *Neurosci. Biobehav. Rev.* 25, 287–295
7 Oberman, L.M. and Ramachandran, V.S. (2007) The simulating social mind: the role of the mirror neuron system and simulation in the social and communicative deficits of autism spectrum disorders. *Psychol. Bull.* 133, 310–327
8 Iacoboni, M. and Dapretto, M. (2006) The mirror neuron system and the consequences of its dysfunction. *Nat. Rev. Neurosci.* 7, 942–951
9 Decety, J. *et al.* (1997) Brain activity during observation of actions. Influence of action content and subject's strategy. *Brain* 120, 1763–1777
10 Williams, J.H. *et al.* (2004) A systematic review of action imitation in autistic spectrum disorder. *J. Autism Dev. Disord.* 34, 285–299
11 Gallese, V. (2003) The manifold nature of interpersonal relations: the quest for a common mechanism. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* 358, 517–528
12 Buccino, G. *et al.* (2004) Neural circuits underlying imitation learning of hand actions: an event-related fMRI study. *Neuron* 42, 323–334
13 Heiser, M. *et al.* (2003) The essential role of Broca's area in imitation. *Eur. J. Neurosci.* 17, 1123–1128
14 Buxbaum, L.J. *et al.* (2005) On beyond mirror neurons: internal representations subserving imitation and recognition of skilled object-related actions in humans. *Brain Res. Cogn. Brain Res.* 25, 226–239
15 Rizzolatti, G. *et al.* (2001) Neurophysiological mechanisms underlying the understanding and imitation of action. *Nat. Rev. Neurosci.* 2, 661–670
16 Tessari, A. and Rumiati, R.I. (2004) The strategic control of multiple routes in imitation of actions. *J. Exp. Psychol. Hum. Percept. Perform.* 30, 1107–1116
17 Gergely, G. *et al.* (2002) Rational imitation in preverbal infants. *Nature* 415, 755
18 Brugger, A. *et al.* (2007) Doing the right thing: infants' selection of actions to imitate from observed event sequences. *Child Dev.* 78, 806–824
19 Brass, M. *et al.* (2007) Investigating action understanding: inferential processes versus action simulation. *Curr. Biol.* 17, 2117–2121
20 Southgate, V. *et al.* (in press) Does the mirror neuron system and its impairment explain human imitation and autism? In *The Role of Mirroring Processes in Social Cognition* (Pineda, J.A., ed.), Humana Press
21 Horner, V. and Whiten, A. (2005) Causal knowledge and imitation/emulation switching in chimpanzees (Pan troglodytes) and children (Homo sapiens). *Anim. Cogn.* 8, 164–181
22 Visalberghi, E. and Fragaszy, D.M. (1990) Do monkeys ape? In *"Language" and Intelligence in Monkeys and Apes: Comparative Developmental Perspectives* (Parker, S.T. and Gibson, K.R., eds), Cambridge University Press
23 Newman-Norlund, R.D. *et al.* (2007) The mirror neuron system is more active during complementary compared with imitative action. *Nat. Neurosci.* 10, 817–818
24 Wilson, M. and Knoblich, G. (2005) The case for motor involvement in perceiving conspecifics. *Psychol. Bull.* 131, 460–473
25 Csibra, G. (2007) Action mirroring and action understanding: an alternative account. In *Sensorimotor Foundations of Higher Cognition: Attention and Performance, XXII* (Haggard, P. *et al.*, eds), Oxford University Press
26 de, C. and Hamilton, A.F. (2008) Emulation and mimicry for social interaction: a theoretical approach to imitation in autism. *Q J Exp. Psychol. (Colchester)* 61, 101–115
27 Sebanz, N. *et al.* (2005) Far from action blind: action representation in individuals with autism. *Cogn. Neuropsychol.* 22, 433–454
28 Carpenter, M. *et al.* (2001) Understanding of others' intentions in children with autism. *J. Autism Dev. Disord.* 31, 589–599
29 Hamilton, A.F. *et al.* (2007) Imitation and action understanding in autistic spectrum disorders: how valid is the hypothesis of a deficit in the mirror neuron system? *Neuropsychologia* 45, 1859–1868
30 Bird, G. *et al.* (2007) Intact automatic imitation of human and robot actions in autism spectrum disorders. *Proc. Biol. Sci.* 274, 3027–3031
31 Gowen, E. *et al.* (2008) Movement interference in autism-spectrum disorder. *Neuropsychologia* 46, 1060–1068
32 Dapretto, M. *et al.* (2006) Understanding emotions in others: mirror neuron dysfunction in children with autism spectrum disorders. *Nat. Neurosci.* 9, 28–30
33 Gergely, G. and Csibra, G. (2006) Sylvia's recipe: the role of imitation and pedagogy in the transmission of human culture. In *Roots of Human Sociality: Culture, Cognition and Human Interaction* (Enfield, N.J. and Levinson, S.C., eds), Berg Publishers
34 Klin, A. *et al.* (2002) Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Arch. Gen. Psychiatry* 59, 809–816
35 Kuhl, P.K. *et al.* (2005) Links between social and linguistic processing of speech in preschool children with autism: behavioral and electrophysiological measures. *Dev. Sci.* 8, F1–F12
36 Cattaneo, L. *et al.* (2007) Impairment of actions chains in autism and its possible role in intention understanding. *Proc. Natl. Acad. Sci. U. S. A.* 104, 17825–17830
37 Theoret, H. *et al.* (2005) Impaired motor facilitation during action observation in individuals with autism spectrum disorder. *Curr. Biol.* 15, R84–R85
38 Oberman, L.M. *et al.* (2005) EEG evidence for mirror neuron dysfunction in autism spectrum disorders. *Brain Res. Cogn. Brain Res.* 24, 190–198
39 Blake, R. *et al.* (2003) Visual recognition of biological motion is impaired in children with autism. *Psychol. Sci.* 14, 151–157
40 Behrmann, M. *et al.* (2006) Seeing it differently: visual processing in autism. *Trends Cogn. Sci.* 10, 258–264
41 Schutz-Bosbach, S. *et al.* (2006) Self and other in the human motor system. *Curr. Biol.* 16, 1830–1834
42 Williams, J.H. *et al.* (2006) Neural mechanisms of imitation and 'mirror neuron' functioning in autistic spectrum disorder. *Neuropsychologia* 44, 610–621
43 Nishitani, N. *et al.* (2004) Abnormal imitation-related cortical activation sequences in Asperger's syndrome. *Ann. Neurol.* 55, 558–562
44 Dinstein, I. *et al.* (2008) A mirror up to nature. *Curr. Biol.* 18, R13–R18
45 Meltzoff, A.N. (2005) Imitation and other minds: the "like me" hypothesis. In *Perspectives on Imitation* (Hurley, S. and Chater, N., eds), pp. 55–78, MIT Press
46 Morton, J.M. (2004) *Understanding Developmental Disorders: a Causal Modelling Approach,* Blackwell
47 Ramachandran, V.S. and Oberman, L.M. (2006) Broken mirrors: a theory of autism. *Sci. Am.* 295, 62–69
48 Altschuler, E.L. (2008) Play with online virtual pets as a method to improve mirror neuron and real world functioning in autistic children. *Med. Hypotheses* 70, 748–749
49 Gallese, V. *et al.* (2004) A unifying view of the basis of social cognition. *Trends Cogn. Sci.* 8, 396–403
50 Happe, F. *et al.* (2006) Time to give up on a single explanation for autism. *Nat. Neurosci.* 9, 1218–1220