Mirror neuron system dysfunction may underlie a self–other matching impairment, which has previously been suggested to account for autism. Embodied Cognition Theory, which proposes that action provides a foundation for cognition has lent further credence to these ideas. The hypotheses of a self–other matching deficit and impaired mirror neuron function in autism have now been well supported by studies employing a range of methodologies. However, underlying mechanisms require further exploration to explain how mirror neurons may be involved in attentional and mentalizing processes. Impairments in self–other matching and mirror neuron function are not necessarily inextricably linked and it seems possible that different sub-populations of mirror neurons, located in several regions, contribute differentially to social cognitive functions. It is hypothesized that mirror neuron coding for action–direction may be required for developing attentional sensitivity to self-directed actions, and consequently for person-oriented, stimulus-driven attention. Mirror neuron networks may vary for different types of social learning such as “automatic” imitation and imitation learning. Imitation learning may be more reliant on self–other comparison processes (based on mirror neurons) that identify differences as well as similarities between actions. Differential connectivity with the amygdala–orbitofrontal system may also be important. This could have implications for developing “theory of mind,” with intentional self–other comparison being relevant to meta-representational abilities, and “automatic” imitation being more relevant to empathy. While it seems clear that autism is associated with impaired development of embodied aspects of cognition, the ways that mirror neurons contribute to these brain–behavior links are likely to be complex.

Keywords: autism; autistic disorder; Asperger’s syndrome; imitation; gaze; attention; motor control; perception; action; mirror neurons; fMRI; parietal cortex; Broca’s area; pSTS; ‘theory of mind’ (TOM)

Introduction

Experimental neuroscience has burgeoned in the last decade as a result of technologies that permit even more detailed and direct measurement of brain function. Hypotheses that explain psychopathology in terms of brain function can now be tested directly. The “mirror neuron hypothesis of autism” is one example [Williams, Whiten, Suddendorf, & Perrett, 2001, discussed in detail below], advanced following the discovery of mirror neurons in the F5 area [Gallese, Fadiga, Fogassi, & Rizzolatti, 1996], and later in the inferior parietal area [Fogassi et al., 2005], of the macaque monkey brain. These neurons fire not only when an action toward an object is executed but also when the same action is observed. The role of these neurons in the evolution of human cognition and social behavior has been subject to widespread speculation [Rizzolatti & Craighero, 2004]. The last 7 years have seen a great expansion in our knowledge both of mirror neurons themselves, and the functions of the brain areas in which they are located. The mirror neuron hypothesis of autism has also received considerable attention [Iacoboni & Dapretto, 2006; Oberman & Ramachandran, 2007].

Consequent upon this, many of the issues raised by Williams et al. [2001] have been clarified and a review of the hypothesis is timely. Before doing so, thought needs to be given to an assertion made by Williams et al. [2001] that mirror neuron dysfunction could be the single primary deficit that explains the behavioral picture that characterizes autism in all of its many presentations. Arguing for this position (as to argue for any single neurological primary deficit) risks being unproductive in the longer term. Complex cognitive functions, such as those that serve social interaction, depend upon networks of many component parts. To try and ascribe a more central role to the mirror neuron system, than to, for example, the amygdala, forces the debate to focus on the question of whether one aspect of brain anatomy is more important than another in the causation of autism. If both are important and autism arises from deficits in either one or other component or the connections between them, such a contention would be unhelpful.

It may therefore be more fruitful to ask what contribution (if any) a specific neurobiological process might make to the development of those cognitive functions that are impaired.
in autism. This requires the combination and interaction of top-down and bottom-up approaches. From the top-down position we can look at the behavioral picture of autism and try to understand the cognitive functions that are affected. Then, from the bottom-up position, we can ask how a specific neurobiological process (in this case the mirror neuron system) contributes to those cognitive functions, and whether the specific neurobiological process is disrupted in autism.

Taking this approach, the mirror neuron/self–other matching theory of autism generates many testable hypotheses that can be grouped into two separate levels: the cognitive (or general) level and the neurobiological (or specific) level. The form at the cognitive level was largely set out by Rogers and Pennington [1991], based on a self–other matching deficit. Although they suggest that a deficit in praxis may underlie the self–other matching deficit, validation of their theory is not dependent upon the involvement of a specific neural substrate. The hypothesis at the neurobiological level [Williams et al., 2001] requires both that mirror neuron function in itself is disrupted in autism, and also that this results in impairment of self–other matching. These two levels of hypotheses are linked but not inextricably so. It is possible that the mirror neuron function can be impaired without it affecting self–other matching, or that self–other matching can be impaired without the mirror neuron function being disrupted. As the evidence accumulates and is re-examined, it is likely to be necessary to revise our understanding of the systems involved, namely the cognitive function served by self–other matching, what we understand to be the contribution of the mirror neuron system to that cognitive function and/or what we understand to be the contribution of the mirror neuron system to other cognitive functions that are impaired in autism. With constant re-examination of the evidence, a program of research can evolve that may contribute more fruitfully to our understanding of both autism and social cognitive development more generally.

This article reviews the current evidence and is presented in the following manner: Part I considers the background to the cognitive and neurobiological hypotheses; Part II examines tests of both cognitive and neurobiological hypotheses; Part III is an evaluation of the evidence for mirror neuron function in humans, particularly with reference to their role in the cognitive functions impaired in autism; Part IV discusses the consequent cognitive hypothesis; finally Part V presents a summary and revised perspective of how mirror neurons and self–other matching may contribute to the deficits observed in autism.

Part I: Background

What are Mirror Neurons?

Mirror neurons are defined by their function: firing when an action is observed but also when that same action is executed. On its own, the notion that self–other matching is served by mirror neurons carries a degree of tautology; it carries a risk of trying to explain a function in terms of itself. The “action” aspect of the definition is therefore a crucial component. “Mirror neurons” are those neurons in premotor and parietal association cortex (intraparietal sulcus—IPS), which converging evidence points to as having mirror properties for the observation and execution of action. This is the “core” mirror neuron system [Oztop & Arbib, 2002]. The posterior superior temporal sulcus provides visual input into the mirror neurons via the temporo-parietal junction (TPJ) and so these areas form an important part of the “mirror neuron system” as a whole.

The Neurobiological (Mirror Neuron) Hypothesis of Autism

The discovery of mirror neurons led Williams et al. [2001] to propose a revision of Rogers and Pennington’s [1991] self–other translation model (discussed below). Their argument at that time consisted of several points, which are summarized here:

1 Autism is characterized by developmental impairments in imitation, joint attention and mental state representation. It is associated with developmental coordination disorder and language delay.

2 The “theory of mind” (ToM) deficit in autism cannot be explained as resulting from a deficit in false-belief understanding because it develops before false-belief understanding develops. An earlier developmental process that underpins development of false-belief understanding is more likely to be important.

3 A simulation “ToM” (representing another’s mental state by imagining one’s self “in another’s shoes”) may develop on the basis of an intact self–other matching function. Understanding another’s intentions in the context of memory allows for “retrodiction” of associated thoughts and memories.

4 Mirror neurons can serve a self–other matching function for action coding. They also code for the relationship between a goal and an object, allowing for formation of intentional representations.

5 Dysfunction of mirror neuron modulation (perhaps disinhibition) could explain both overactive copying in the case of echolalia and also failure to generate covert or “offline” imitation. This latter function may be necessary to develop “ToM.”
6 Autism does not necessarily involve language impairment, suggesting that impairment of “mirroring” function in one domain (e.g., social reciprocity) does not necessarily require impairment in another (e.g., language). Separate “mirror neuron” networks may serve gaze and language.

7 There may also be separate mirror neurons for other functions, most particularly joint attention. There might also be neurons with “mirror” properties in the region of the superior temporal sulcus.

The Self–Other Matching (Cognitive) Hypothesis

The origins of the self-other matching hypothesis of autism may be traced back to the first paper published on the imitation impairment [Ritvo & Provence, 1953], just 10 years after Kanner [1943] had published his seminal paper on autism. Over the following decades this was followed by many now well-reviewed studies showing that children with autism did not imitate [Rogers, 1999; Rogers & Williams, 2006; Smith & Bryson, 1994; Williams, Whiten, & Singh, 2004]. Rogers and Pennington [1991] conducted an influential review that found strong evidence for the existence of a deficit affecting imitation of simple body movements as well as symbolic imitation. Using Stern’s [1985] model of interpersonal development, Rogers and Pennington suggested that a biological impairment in autism restricted the capacity of the infant to “form and co-ordinate social representations of self and other at increasingly complex levels via amodal or cross-modal representational processes.” They hypothesized that a primary deficit in such a capacity would lead to a cascade of effects including impaired imitation, social, communicative and affective skills and that a deficit of praxis involving the prefrontal–limbic neural system could form the basis for this impairment.

Smith and Bryson’s [1994] review of 15 studies hypothesized that the imitative deficit associated with autism could be due in part to impairment in the perceptual organization of movements, manifesting in an abnormal representation of actions. Increasingly sophisticated studies over the following decade demonstrated imitation impairment in very young children with autism [Charman et al., 1997] and those with milder degrees of autism [Rogers, Bennetto, McEvoy, & Pennington, 1996]. The impairment appeared to affect gesture particularly [Roeyers, Van Oost, & Bothuyn, 1998] and interestingly, also, imitation of action “style” [Hobson & Lee, 1999], though differences in controlling for task difficulty between gesture and action-on-object make it difficult to judge whether specific aspects of imitation are affected. The most recent studies support the hypothesis that the imitation deficit concerns representational aspects of action [Perra et al., 2008; Smith & Bryson, 2007], although not all agree [Hamilton, Brindley, & Frith, 2007; Vanvuchelen, Roeyers, & De Weerdt, 2007].

Embodied Cognition

The hypothesis that autism stems from a deficit in a mechanism that serves to match codings for action between motor and perception domains is based on a more general principle that certain aspects of cognition are founded upon action, encapsulated by theories of “embodied cognition.” Embodied theories of cognition [Niedenthal, Barsalou, Winkielman, Krauth-Gruber, & Ric, 2005] view the processes of knowledge use and acquisition as fundamentally grounded in their physical context and the brain’s modality-specific systems. This means that those cognitive operations that are heavily influenced by both action plans and emotional states come to function within the biological environment of the brain that serves action plans and emotional states. An example of embodied cognition would be found in a model of empathy. When we observe emotions being expressed by others we may not only feel them ourselves but also express them in our actions by making subtle facial movements that can be detected using electromyography [Hietanen, Surakka, & Linnankoski, 1998; Surakka & Hietanen, 1998]. Empathy cannot be separated from the action-based systems that serve the experience and expression of emotion.

Klin, Jones, Schultz, and Volkmar [2003] have advanced an embodied cognitive model of autism, termed the “enactive mind” (EM) approach. This construes the mind as an active entity that shapes itself as a result of solving problems experienced in the environment. The developing child’s actions are guided by perceptions, which in turn are driven by attentional demands related to social needs. The emphasis of their EM approach is on perception and visual attention. In contrast, self-other matching theory considers cognitive development to be grounded in systems that serve action. However, there is clearly cross-over, as both theories consider the development of attentional control to be grounded in visuo-motor systems that require cross-modal influences. A further essential component of the “self-other matching” model of autism is its focus on detection of similarities and differences between self and other [Pennington, Rogers, & Williams, 2006].

Part II: Tests of the Mirror Neuron Hypothesis of Autism

Tests of the Neurobiological Hypothesis

Several studies using different methodologies have now tested the mirror neuron hypothesis of autism. Nishitani, Avikainen, and Hari [2004] studied a mixed sex group of
adults with Asperger’s syndrome. Subjects imitated still images of orofacial gestures that were projected onto a screen, while brain activity and lip movements were recorded using magnetoencephalography and electromyograms (EMG). There was no difference in the delay before which mouth movement started in each group, but curiously EMGs lasted almost twice as long for the autism spectrum disorder (ASD) group. In the control group imitation was associated with activation in occipital, inferior parietal, superior temporal and Broca’s areas bilaterally. In the ASD group, several individuals failed to activate Broca’s area on one or both sides. The results of this study are consistent with those of Dapretto et al. [2006]). They used functional magnetic resonance imaging (fMRI) to examine differences in brain activity between 10 children with autism and 10 controls (aged about 12 yrs, normal IQ) imitating facial expressions while in the scanner. They found no difference in imitation abilities outside of the scanner but reduced activity in Broca’s area inside the scanner. Most striking, activity in Broca’s area correlated with the severity of autism as measured on the social subscales of the ADI-R [Lord, Rutter, & Lecouteur, 1994] and ADOS-G [Lord et al., 2000].

The next question, for both studies, is whether group differences were mediated by specific problems with self–other matching (imitation). A possibility not yet excluded is that group-wise effects reflect other differences between conditions, such as those that relate to the attention or the direction of action. Unfortunately, neither study benefited from having control tasks, which might have shown that the group difference was confined to the copying element of the task.

An effect of direction is suggested in a further study of mirror neuron function in autism by Theoret et al. [2005]. They also found impaired Broca’s area function during action observation but suggest that the effect is specific to the directional context in which the action is performed. Theoret et al. drew on the methodology originally used by Fadiga et al. [1999] that provided some of the most compelling evidence for “mirror neurons” in humans. Transcranial magnetic resonance stimulation evokes activity within premotor cortex, which can then be detected through its effects on myoelectrical activity in the hand. Fadiga et al. showed that observation of an action supplemented this activity but, importantly, activity was only supplemented in the muscles involved in the action being observed. Theoret et al. repeated the technique in individuals with autism and controls. They found expected patterns in controls; action observation increased the motor-evoked potentials (MEP). In the 10 adult participants with Asperger’s syndrome, some action observation supplemented the MEP but whether it did so depended upon the direction of the action. Only actions performed toward the observer were supplemented.

One other fMRI study has examined imitation in autism. Williams et al. [2006] used the protocol published by Iacoboni et al. [1999] to examine neural correlates of imitation in a group of normal IQ adolescent males with autism. They found differences in the parietal lobe but not in Broca’s area. The group differences were less dramatic than those described by Dapretto et al. [2006], perhaps because of the very simple and less social nature of the imitation task. Engagement of parietal rather than ventral premotor cortex may have occurred because the task involved hand rather than facial imitation. This study also highlighted other group differences that could shed light on the imitation impairment in autism, including differential activity in the amygdala across several conditions, and also in a location within the posterior aspect of the superior temporal sulcus (pSTS) region, in the imitation vs. observation contrast. In controls, the pSTS region was clearly active during imitation but hardly active during observation. The reverse was the case for the autism group. As pSTS provides visual input into the mirror neuron system, this study suggested that imitation in autism is associated with greater dysfunction of other structures supporting the core mirror neuron system, than of the core components themselves.

**Mu-wave suppression.** Electroencephalography is another useful tool in the investigation of the “mirror neuron” impairment in autism. The mu rhythm is detected over the premotor cortex and decreases during preparation of motor actions. It is thought to reflect down-stream modulation of motor neuron cells by the premotor cortex [Pineda, 2005]. A link between the mu rhythm and “mirror neurons” was first suggested by Altschuler, Vankov, Wang, Ramachandran, and Pineda, [1997]. Oberman et al. [2005] demonstrated reduced mu-wave suppression during autism when actions are observed, but not during action following instruction. Again, or whether the effect was specific to self–other matching was not demonstrated. However, this finding has been replicated by Bernier, Dawson, Webb, and Muriel [2007], who found that impairment correlated with degree of impaired imitation ability. These results are consistent with those mentioned previously, which showed decreased activity in the ventral premotor cortex during imitation. These data thus support the hypothesis that mirror neuron function is impaired in autism, in a manner that relates closely to imitation ability.

**Structural neuroimaging.** The mirror neuron regions do not have a specific cytoarchitecture or predefined area, so it has been difficult for structural neuroimaging studies to look at whether they show altered volume in autism. Nevertheless, decreased cortical thickness in the mirror neuron regions in both the parietal and ventral premotor cortex in autism has been demonstrated [Hadjikhani, Joseph, Snyder, &
Tager-Flusberg, 2006]. Furthermore, in a later study, Hadjikhani, Joseph, Snyder, & Tager-Flusberg [2007] found that decreased thickness of the inferior frontal gyrus in autism was associated with decreased BOLD signal in this area during face processing. Less directly, several studies have shown decreased white matter connectivity in autism [Barnea-Goraly et al., 2004; Keller, Kana, & Just, 2007; McAlonan et al., 2005; Waiter et al., 2005], raising the possibility that impaired mirror neuron function in autism could be explained by deficits in connectivity between elements of the network [Minshew & Williams, 2007].

Functional imaging of ToM. If mirror neurons are important for ToM, it might be that an appropriate test of the mirror neuron hypothesis of autism would be to measure mirror neuron activity in ToM studies. At present there is little evidence for specifically reduced mirror neuron activity in ToM in autism. The neural substrate for ToM and other mental state attribution, such as empathy, has now been well investigated and reviewed [Amodio & Frith, 2006; Frith & Frith, 2003]. ToM is served by three brain areas: the medial prefrontal cortex, posterior STS and the temporal pole. Studies of empathy have highlighted roles of anterior cingulate, temporal pole and insula [Singer et al., 2004; de Vignemont & Singer, 2006; Vollm et al., 2006; Wicker et al., 2003].

Gallese, Keysers, and Rizzolatti [2004] have argued that the insula is part of the mirror neuron system as it may also serve to match for actions, and therefore mirror neurons are important for empathy. There is perhaps a stronger case for a more general role of mirror neurons in ToM. ToM is considered by some to be served by cognitive function that is specific to it (“domain-specific” function), which requires separation from other “domain-general” functions, which may also include memory and executive function, but which may nevertheless be vital to ToM development [Perner, Aichhorn, Kronbichler, Staffen, & Ladurner, 2006; Saxe & Baron-Cohen, 2006; Saxe, Schulz, & Jiang, 2006]. Therefore, as will be discussed later, mirror neurons may be very important for ToM, but by serving a domain-general function. In support of this position, fMRI studies of ToM in autism have found differences in activity in STS and Broca’s area [Baron-Cohen et al., 1999; Castelli, Frith, Happe, & Frith, 2002] suggesting that the mirror neuron system may be underactive during ToM function in autism. Baron-Cohen et al. [2006] reported (as a preliminary finding) that Broca’s area showed reduced activity during an empathic task in parents of children with autism compared to control parents, and also that this area was less active in male controls compared to female controls. Thus, they suggest that this area could be hypermasculinized in autism. Most recently, Pfeifer, Iacoboni, Mazziotta, & Dapretto [2008] have also shown a close relationship between activity in Broca’s area during observation and imitation of emotional expression, and measures of empathic ability in children.

Recent Tests of the Cognitive (Self–Other Matching) Hypothesis

This section reviews some of the ways that the self–other matching hypothesis has been tested in autism besides tests for imitation impairment, which have been recently reviewed elsewhere [see above and Rogers & Williams, 2006; Williams et al., 2004].

Emotional contagion. A number of recent studies have explored the role of facial mimicry in autism. In an early study, Platek, Critton, Myers, & Gallup [2003] found reduced contagious yawning in a group of individuals with “schizoid personality traits.” It is possible that a proportion of these participants had ASD. More recently, Senju et al. [2007] found that children with autism were less prone to contagious yawning than controls. McIntosh, Reichmann-Decker, Winkielman, and Wilbarger [2006] found that people with autism showed a reduced electromyographic response in their facial muscles when observing facial expressions but no decrease in activity during intentional expressions. These studies demonstrate that perceived facial expression has a reduced power to evoke spontaneous facial expression in autism. However, a lack of spontaneous facial muscular activity in autism is well known, perhaps resulting from an impairment at the level of the brainstem [Rodier, 2002]. It therefore remains unclear whether reduced emotional contagion effects are the result of a self–other matching deficit or nonspecifically reduced levels of tonic stimulation to facial musculature.

Automatic imitation. Recently, a very interesting study by Cattaneo et al. [2007] used similar methods to McIntosh et al. [2006] by measuring electromyographic activity during observation of an action, in muscles that would be used were the action being imitated. However, they looked at muscle activity that would be expected to occur as a natural consequence of the action being observed. Typical children, but not children with autism, activated their jaw-opening muscles as they observed a piece of food being grasped. The authors argue that children with autism do not perceive intention. This may be the case, but alternatively, children with autism may simply not learn the same associations between actions that are typically contingent upon one another.

Cross-modal influences. The “mirror neuron” hypothesis of autism predicted that there would be a reduced McGurk effect [McGurk & MacDonald, 1976]. The McGurk effect constitutes an influence of observed mouth movements on heard speech, so that the perceived speech is a fusion of auditory and visual stimuli. Williams et al. [2001] drew from Liberman’s theory of speech perception [Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967; Liberman &
Whalen, 2000], which suggests that we hear the sounds according to how we produce them. More recently, mirror neurons have been shown to respond to the sound of actions as well as the sight of them when those actions have a characteristic sound [Gazzola, Aziz-Zadeh, & Keysers, 2006; Kohler et al., 2002].

McGurk effect was not reduced in autism in one study [Williams et al., 2004] but effect sizes were small and perhaps confounded by effects in the monomodal conditions. However, Smith and Bennetto [2007] found reduced effect of visual speech on auditory perception in autism. Williams, Waiter, Perra, Newell, 2007]. To my knowledge there have been no indicators of an autistic spectrum disorder [Mundy & Rogers, 2004]. Early findings therefore support the hypothesis that the mirror neuron system is involved in joint attention and also that the joint-attention problem in autism stems from a self-other matching deficit. However, as with ToM studies of empathy, areas outside the mirror neuron system are emphasized in serving the cognitive function most specific to joint-attention impairment in autism.

**Visual sensitivity to action.** The pSTS (see a more detailed discussion below) provides the visual input to the mirror neuron system [Arbib, 2005; Iacoboni & Dapretto, 2006; Iacoboni et al., 2001; Keysers & Perrett, 2004]. Furthermore, the pSTS is important for controlling attention to visual stimuli and may be sensitive to input from motor areas, which affects sensitivity to action perception [Astañev, Stanley, Shulman, & Corbetta, 2004; Neri, Luu, & Levi, 2006]. Therefore, deficits in this area may reflect dysfunction of the mirror neuron system as a whole. Visual sensitivity to action is studied using films displaying actions as moving light points, measuring the ability to distinguish between genuine actions and randomly moving light points. Moore, Hobson, and Lee [1997] found that children with autism were as equally sensitive to behavioral actions as controls but were less sensitive to actions that conveyed emotions. Similarly, Hubert et al. [2007] found that people with autism were equally as able to label biological action content but were less able to label emotional displays. Yet when a measure of sensitivity not requiring discrimination between action types was used, Blake, Turner, Smoski, Pozdol, and Stone [2003] found that people with autism were less sensitive to all biological motion. In this study, impairment also correlated with autism severity. Others [Klin, 2000; Rutherford, Pennington, & Rogers, 2006] have detected a reduced sensitivity to animations in autism, where two geometric objects move as apparently autonomous agents. This has also been shown to be associated with decreased activity in the area of the pSTS and medial frontal cortex in autism [Castelli et al., 2002].

**Joint attention in autism.** Joint-attention abilities, and particularly behaviors that concern initiation of joint attention perhaps provide the strongest diagnostic indicators of an autistic spectrum disorder [Mundy & Newell, 2007]. To my knowledge there have been no direct assessments of mirror neuron function in joint attention in autism. However, Williams, Waiter, Perrin, Perrett, & Whiten [2005] conducted a relevant study of “joint-attention experience” in a group of normal controls. They showed participants a video clip of a moving spot and asked them to watch it. There was also a person in the video who either watched the spot as well or looked elsewhere. Both conditions were associated with activity in the region of the IPS (though not BA44), but only watching with someone else created a joint-attention experience. This was associated with greater activity in the left frontal pole and the right ventromedial frontal cortex. These findings have recently been replicated by Bristow, Rees, and Frith [2007]. The left frontal pole cluster was in the same location as an area of structural difference in autism the same group [Waiter et al., 2004] had detected previously using voxel-based morphometry. The area is thought to serve an “information integration” function and has a particularly low gray matter density [Ramnani & Owen, 2004].

**Part III: Mirror Neurons: Contributions to Cognitive Function**

**General Functional Neuroanatomy**

Direct recordings of mirror neuron activity demonstrate their presence in the monkey prefrontal cortex [area F5; Gallese et al., 1996] and the parietal area known as “PF” [Fogassi et al., 2005]. Which exact areas of human brain are homologous to these areas, is still a matter of some debate, as the same techniques cannot be used to demonstrate mirror neurons in humans. Neurophysiological experiments [e.g. Fadiga et al., 1999; Hari et al., 1998] provide experimental support in humans for effects that could result from mirror neuron activity, but do not provide high-resolution spatial information. fMRI has been particularly useful (discussed below) but such studies cannot show whether individual neurons have specificity for particular actions or action types. Also, since mirror neurons could have underpinned evolution of human cognitive abilities [Whiten, 2006], new areas, not serving a mirror function in monkeys, could have evolved to serve this function in humans. Notwithstanding these reservations, the mirror neuron system is currently considered to be made up of neurons located in two main areas: the ventral premotor cortex and the inferior parietal cortex. The posterior temporal cortex is considered integral as it provides the visual input to the system [Iacoboni & Dapretto, 2006]. Another important area is the superior parietal cortex [Arbib, 2008; Oztop and Arbib, 2002].

**Parietal cortex.** There are several reasons why parietal cortex may have “mirror neurons” that are important for social cognition. Firstly, there is some evidence that the human parietal cortex has expanded preferentially in
recent evolution compared to other cortex [Astaieiev et al., 2003], suggesting that it could be important for recently evolved social cognitive functions such as imitation [Whiten & Byrne, 1997]. Secondly, it is polymodal association cortex that receives and integrates input from visual, motor and somatosensory areas, used to plan bodily movements and to direct attention [Grefkes & Fink, 2005; Rozzi et al., 2006]. Thirdly, lesions of the parietal cortex result in imitative apraxia [Goldenberg, 1996]. Fourthly, Fogassi et al. [2005] identified a population of mirror neurons in the inferior parietal cortex of the monkey that fired differentially whether an object was picked up to eat it or picked up to place it elsewhere. Fogassi et al. conclude that “mirror neurons” in the inferior parietal region code for commonly observed and executed intention as well as action itself and may serve intention representation in the parietal lobe by matching contextual aspects of action.

Iacoboni et al. [2005] tested a similar hypothesis in humans, asking participants to observe video clips showing a cup being grasped in isolation, or in a context. The context showed whether the cup was being grasped to be drunk from, or for cleaning up. Additional effects of context on observing the action were evident in inferior frontal gyrus but not in inferior parietal cortex. Hamilton and Grafton [2006] identified parietal activity that coded intention by representing goals of actions. These studies highlight differences in the relationships between actions and goals, and actions and contexts. In Iacoboni et al. an action’s context was used to predict its goal and consequently its intention. In the study by Hamilton and Grafton, the goal was determined by the position of the object in relation to the direction of action. Therefore, the object’s physical properties (in this case its position in space relative to the action) or “affordances” [Arbib, 2005] imply the goal. It follows that different mirror neurons may code for the action–goal and action–context relationships.

Parietal cortex is extensive and its many functions have been mapped most accurately in monkeys. It is divided into inferior and superior parietal lobes by the IPS [Grefkes and Fink, 2005]. Anterior IPS and the anterior intraparietal area are closest to somatosensory cortex and are active during grasping and object manipulation. Lateral IPS serves shifts of visual attention, while medial IPS serves planning and monitoring of reaching movements. The ventral aspect of IPS integrates motion processing in different sensory modalities. More caudal aspects closer to visual cortex may process 3D object characteristics [Shikata et al., 2003]. The function of an area is not necessarily specific to effector, as common areas of IPS serve looking, pointing and covert attention [Astaieiev et al., 2003]. Reciprocal connectivity with frontal areas that include the frontal eye fields and premotor cortex contributes to these functions. The Oztop and Arbib [2002] model places most of the parietal cortex outside of the mirror neuron system. More recently, Dinstein, Hasson, Rubin, & Heeger [2007] suggest that more areas of parietal cortex serve neuron functions in humans.

**Broca’s area.** The hypothesis that neurons in Broca’s area could be important for imitation has received support from several studies. Activity in Broca’s area is greater during imitation than during observation or other nonimitative action–execution conditions [Iacoboni et al., 1999; Jackson, Meltzoff, & Decety, 2006]. Heiser et al. [2003] found that applying transcranial magnetic stimulation (TMS) over Broca’s area impaired reaction times on an imitation task, but spared the control task. Koski et al. [2002] found that imitation of a goal-directed action was particularly associated with activation of Broca’s area and Buccino et al. [2004] demonstrated involvement of Broca’s area in sequential imitation. Buccino et al. [2001] found that action observation activated dorsal premotor cortex as well as Broca’s area. A number of studies have questioned the role of Broca’s area in imitation compared to other action–execution conditions [Chaminade, Meltzoff, & Decety, 2005; Grezes, Armony, Rowe, & Passingham, 2003; Makuuchi, 2005; Williams et al., 2006, 2007a,b]. Grezes et al. [2003] suggested that this area may serve a less specific action–execution function such as selecting a response to a visual instruction. There may be several other explanations for these conflicting results. The activity when executed and observed actions match may not be that much greater than when they are executed or observed alone. Repetitive responses or stimulus presentation may also be associated with reduced activity. Also, Broca’s area mirror neurons may have an important role in orienting attention to actions (see further discussion below), resulting in greater activity during nonimitation conditions as well as during imitation. These confounding factors could mean that the additional effect of self–other matching on Broca’s area through imitation is relatively small and is only detected by more powerful 3T scanners.

**The posterior STS.** A number of features of the pSTS make it a promising focus for research into mirror neuron system functioning in autism. As well as being active during imitation [Decety, Chaminade, Grezes, & Meltzoff, 2002; Iacoboni et al., 2001], it is a convergence point for two streams of visual processing, deriving input from the motion-processing “MT” area of visual cortex and the content-characterizing pathway of the ventral stream [Puce & Perrett, 2003]. It also has close connectivity with the orbitofrontal cortex (OFC) and amygdala making it an integral part of Brothers’ social brain [Brothers, 1990; Emory & Perrett, 2000]. Cells in the Macaque STS respond to the sight of intentional actions such as directed gaze and head or eye movements [Jellema, Oram, Baker, & Perrett 2002; Jellema, Baker, Wicker, & Perrett, 2000; Perrett et al., 1989]. In humans, this area is active while viewing faces, mouths and eyes [Puce, Allison, Bentin, Gore, & McCarthy, 1998] and also during mental state representation, both intentional...
attribute [Castelli, Happe, Frith, & Frith, 2000; Allison, Puce, & McCarthy, 2000; Saxe & Kanwisher, 2003; Saxe et al., 2004] and more complex meta-representation [Gallagher et al., 2000; Vollm et al., 2006]. Roles in action planning and visual attention are discussed below.

**Social orienting and attentional control**

It is possible that when attentional functions involve social stimuli such as faces and gestures, they act differently to when they serve other objects. Such social attention processes include joint attention, which is fundamental to the clinical picture of autism [Dawson et al., 2004; Mundy & Newell, 2007]. Attention to social stimuli is also a key component of imitation, and of the tests of mirror neuron function reported above, which require the participant's attention to be drawn to a facial expression or a gesture. This facial expression or gesture, may, at the same time, be directed to the participant. Two possibilities will be discussed. Firstly, that Broca's area is key to differentiating between social and nonsocial attentional function, meaning that differential activity in Broca's area in autism, which has been reported as evidence for mirror neuron dysfunction, could actually be reflecting attentional rather than self-other matching differences. However, a second possibility is that a self-other matching function served by mirror neurons in Broca's area is necessary for developing this social attentional function, which in turn has major developmental implications for the way that we relate to others.

**Broca's area and visual attention.** A widely accepted model for visual attention [Corbetta & Shulman, 2002] divides it into two processes: goal-directed and stimulus-driven attention. Goal-directed (or endogenous) attention is driven by "top-down" executive function processes and is required during search tasks when attention is to be directed to a previously identified object. Such attention is largely under the control of a dorsal fronto-parietal network (DFPN) that includes the dorsal frontal cortex (frontal eye fields), the superior parietal lobe, the IPS and the dorsal parietal cortex.

Stimulus-driven (exogenous) attention occurs in response to an external stimulus, which demands attention. An example would be a flash at the edge of a visual field that will draw attention toward it. This process is under the control of a right ventral fronto-parietal network (RVPN). This involves involving inferior and middle frontal gyrus that includes Broca's area [Downar, 2000, 2001] and TPJ.

The endogenous and exogenous attentional networks are not completely independent. Activity within TPJ (which is close to the pSTS) is modulated by the relevance of the exogenous stimulus to the task; the sensitivity of the dorsal system is modulated by stimulus features and task contingency.

**Mirror neurons and social attention.** Mirror neurons in Broca's area are not just sensitive to the shape of an action, but are also sensitive to the direction in which an action is performed relative to the observer. Gallese et al. [1996] reported that 30 out of 47 mirror neurons tested showed directional preference. It therefore seems very possible that there will be mirror neurons in Broca's area that fire when actions are observed as self-directed, and when they are executed toward another person (i.e. they code for the interpersonal direction of intention).

An important aspect of autism concerns the difficulties that others may have in attracting the child's attention. A child with autism may not look up when his or her parent enters the room, or may not follow a gaze or a point. This seems to reflect a lack of sensitivity of the exogenous attentional network to a familiar action directed toward the individual. As mirror neurons located in Broca's area are sensitive to familiar actions and action–direction, this makes the case for hypothesizing a central role for mirror neurons in the social attention problems characteristic of autism. Furthermore, if cognition is founded upon action, embodied cognitive theory would predict that this deficit in orienting to self-directed action, would be extended in development to have broader effects on attentional control.

Action–direction includes the direction of a facial expression. Williams, Perrett, Watter, & Pechey [2007a] found activity within Broca's area (as well as brain areas serving mentalizing) to be much greater when a face was observed from the front compared to the side. This greater activity was dependent upon intact serotonin function, which may be impaired in autism [e.g. Devlin et al., 2005]. The hypothesis that mirror neurons in Broca's area are important for social attention has several implications as follows:

**Social salience and orienting.** In linking neural codings for the perceptions of self-directed actions to codings for actions that serve orienting behaviors toward others, the mirror neuron system could play an essential role in promoting the salience of exogenous, socially relevant stimuli, which are directed toward the individual. Consequently, when an action is perceived to be directed toward a person, the mirror neuron system is important in promoting a reciprocal action directed toward the actor. Similarly, when an action is perceived to be directed toward an object, this favors the execution of an action being executed by the observer, toward the same object.

In the imitation studies described above, mirror neurons may be important for contributing to premotor activity by fostering orienting actions-to-others in response to observation of self-directed action. Dapretto et al. [2006] suggested that the lack of behavioral differences in their study could be accounted for by the
increased visual attention to the images by the children with autism, as evidenced by greater activity in visual and parietal cortices. This could be further explained as resulting from the absence of the mirror neuron contribution to “bottom-up” stimulus-driven visual attention, resulting in greater “top-down” attentional influences in its absence. Social salience mediated by Broca’s area may also be important for faster reaction times when responses to observed stimuli are imitative, because familiar actions activate mirror neurons and so achieve greater visual salience (see further discussion below).

Preventing fixed patterns of attention. Corbetta and Shulman [2002] propose that the RVPN functions as a “circuit breaker.” Attention is maintained in a “top-down” task by a circuit from visual cortex back to the frontal eye fields. This can be interrupted by the RVPN, which would appear to be well suited to serve this function. For example, if you are looking at me when my attention is directed toward another task, such as reading a book, my attention toward that task will be interrupted to look at you. In autism absence of “circuit breaking,” due to impaired mirror neuron function in Broca’s area, is likely to enhance “top-down” attentional control by the DFPN, perhaps resulting in fixed patterns of attention to nonsocial stimuli [Landry & Bryson, 2004; Ozonoff et al., 2004] as well as enhanced search abilities [O’Riordan, Plaisted, Driver, & Baron-Cohen, 2001].

Development of egocentrism and allocentrism. It has often been suggested that people with Asperger’s syndrome are extremely “egocentric” [Asperger, 1944; Frith & de Vignemont, 2005; Gillberg & Gillberg, 1989]. Williams [2007] has recently challenged this view, arguing that people with autism are in fact the exact opposite—extreme allocentrists. In his early writing, Piaget described “autistic” intelligence [Piaget & Warden, 1926]. The term “autism” had been introduced by Bleuler and Piaget used it to describe “undirected intelligence” as autistic. Undirected intelligence reflects a lack of “egocentricity,” the latter being characterized by framing experience in terms of the way it relates to the self. Even though the condition of autism had not yet been described, use of the term “autistic” was curiously apposite for what we know about autism today, which could be said to be characterized by failure to encode experience in terms of its personal relevance [Williams, 2007]. Developmentally, a deficit in mirror neuron function in autism could result in a failure to discriminate (in terms of learning different patterns of responses) between self-directed and other actions, resulting in a tendency to treat all behavioral observations similarly, regardless of whether they are directed at one’s self or not. This may give rise to an apparent lack of interest in other people’s behavior and attitudes. Consequently, extreme allocentricty may be misconstrued as egocentricity.

**Part IV. Further Development of the Cognitive Hypothesis**

**Mirror Neurons and Mental State Representation**

An important aspect of the self–other matching hypothesis of autism has been the suggestion that the mirror neuron system could play a role in attributing mental states to others (“mentalizing”) through a “simulation” process. The original model [Gallese & Goldman, 1998] has recently been expanded upon by Hurley [2008]. Both these accounts draw upon earlier models of mentalizing [Gordon, 1996; Meltzoff & Gopnik, 1993] arguing that individuals develop models of others’ thoughts by imagining themselves in the other’s position. Gallese and Goldman [1998] suggest that mirroring processes allow others’ perceived behavior to be matched to the neural codings, which represent corresponding memories of the same experience. The mental state that gave rise to that action is then “retrodicted.” Such “off-line” simulation requires that observation of an action “automatically” activates the motor program for this same behavior, but that it is then inhibited for processing “off-line.”

**Intentional and Automatic Imitation**

The Gallese and Goldman [1998] model is therefore predicated on the notion of “automatic” imitation. This idea was originally espoused by William James who stated that “every representation of a movement awakens in some degree the actual movement which is its object; and awakens it to a maximum degree whenever it is not kept from doing so by an antagonistic representation present simultaneously in the mind” [James, 1890; p 1134]. Automatic imitation can be observed in daily encounters, whether through the unintentional copying of another's body posture or the adoption of others’ accents in speech. Experimental evidence for this sort of imitation is also plentiful [e.g. Charrtrand & Bargh, 1999]. More recently this has been referred to as the “Ideo-Motor theory” [Brass & Heyes, 2005] and “automatic imitation” has been demonstrated in several experiments [Brass, Bekkering, Wohlschlager, & Prinz, 2000; Kilner, Pauignan, & Blakemore, 2003; Press, Bird, Flach, & Heyes, 2005] showing quicker or smoother responses to cues when they are imitative compared to when they are nonimitative [though nonspecific problems with cuesalience and stimulus–response compatibility effects may present problems with these studies; Aicken, Wilson, Williams, & Mon-Williams, 2007; Jansson, Wilson, Williams, & Mon-Williams, 2007]. With respect to facial expression, experiments have shown that observation of
facial emotion results in unintentional activation of the corresponding muscle groups in the observer [Hietanen et al., 1998; McIntosh et al., 2006].

Not all imitation is automatic, and it is often intentional and effortful [Grezes, Costes, & Decety, 1999]. If cognitions are founded upon action [Niedenthal et al., 2005], the boundary between automatic and effortful imitation may determine that between empathy and simulation “ToM.” Different types of social learning have been classified according to the cognitive processes upon which they are dependent [Whiten, 2006; Whiten and Ham, 1992]. Imitation that involves learning a novel action by watching someone else perform it appears to be distinct from the imitation that occurs when an action already exists in the motor repertoire, and is evoked by observing another perform it. One test of whether or not imitation is “automatic” is whether action observation without execution necessarily requires inhibition of the motor or premotor cortex. Using fMRI, Brass, Zysset, and von Cramon [2001], and Brass, Derrfuss, and von Cramon [2005] sought evidence for motor inhibition when participants executed a similar but different action to the one observed (they lifted an alternative finger to the one they observed being lifted). They found greater activity relating to self–other discrimination, but not motor inhibition. Williams et al. [2007b] also found no evidence of motor inhibition during a similar task.

In conclusion, imitation learning does not appear to be automatic, and most imitation that falls into the category of “automatic” concerns pre-learnt and salient actions, perhaps achieving salience by being emotionally laden and directed to the observer. As these qualities apply to facial expressions of emotion, it follows that empathy is automatic. Clinicians observe that echolalic content tends to be dominated by emotionally salient aspects of speech. Thus, swear words are not an uncommon form of echolalic content. However, understanding another person’s novel thoughts by imagining ones’ self in his or her position is unlikely to be automatic and will be more effortful. It may depend on additional cognitive function that serves imitation learning.

**Imitation learning.** Imitation learning is the means by which skilled movements are learned through the observation of others [Whiten, 2006]. Imitation is informed by the principles of motor learning involving “inverse models,” which describe the development of motor skills as being practice-dependent [Wolpert, Doya, & Kawato, 2003; Wolpert, Gehleramani, & Flanagan, 2001]. Attempts to achieve desired actions are made that result in error feedback. This is utilized in order to modify programs until the actions achieve their desired trajectories. Error feedback is obtained from sensory functions, which require cross-modal translation before it can influence the motor-programming function. During imitation, observation of another individual’s behavior may serve as the sensory input against which to compare one’s own motor plans. Motor learning and imitation are therefore intentional and incremental learning processes. Oztot, Wolpert, and Kawato [2006] have developed sophisticated computational models that show how mirror neurons may serve imitation.

Williams, Whiten, Waiter, Pechey, & Perrett [2007b] investigated this process in an fMRI study of imitation. Brain activity was compared between imitation and a condition where an alternative learnt action was enacted. Differences in parietal and dorsal premotor cortex reflected mirror neuron function. There were also differences in lateral OFC and caudal anterior cingulate cortex (cACC). The OFC is closely tied to emotional learning and lateral OFC is thought to serve behavioral change in response to error feedback. Error detection also resulted in conflict-related activity in cACC.

Williams, Whiten, Waiter et al. [2007b] conceive of imitation as a tracking function that continuously compares and contrasts one’s self with others, recognizing differences as well as similarities between self and other. This is illustrated by an fMRI study [Calvo-Merino, Glaser, Grezes, Passingham, & Haggard, 2005] that looked at expert dancers’ brain activity as they observed other dancers. The dancers being observed exhibited a common expertise with the observer or an expertise in a different form of dance (ballet or capoeira). Calvo-Merino et al. concluded that the experiment identified the brain areas serving action recognition. Williams et al. [2007b] suggested that it also identified brain areas serving self–other comparison. Observers ask how they differ to others as well as how they are similar, perhaps asking whether they are better or worse at performing the skill. Comparing (as opposed to “matching” or “identifying”) ourselves with others is a fundamental aspect of social processes that is closely related to imitation. Furthermore, the close relationship between imitation and the OFC underlines the importance of emotional learning in imitation and the typical presence of a strong drive to be like others or better than them.

One implication of this is that motor skills should not only be measured in terms of dexterity, accuracy or speed, but also their capacity for encapsulating and communicating mental states such as emotions. This is an important dimension of motor development that is deeply relevant to autism. Consistent with this, Perra et al. [2007] found that imitation impairments in people with autism cluster with performance measures on ToM tasks to discriminate between children with and without autism. A strong theoretical case has been made for abnormality of the amygdala–OFC circuit in autism [e.g. Bachevalier & Loveland, 2006; Baron-Cohen et al., 2000a; Dawson, Meltzoff, Osterling, & Rinaldi, 1998; Schultz, 2005]. Reduced influence of the amygdala–orbitofrontal circuit upon the mirror neuron system could be important in influencing the socialization of action–learning in autism. This could be an important developmental influence and offers a feasible mechanism.

---

**82 Williams/Mirror neurons in autism**
for developmental dysfunction of the mirror neuron system in autism.

**Imitation Learning and “ToM”**

The following paragraphs outline an alternative model that could explain how self–other comparison is required for ToM development. It may be comparable to recent computational models [Oztop, Kawato, & Arbib, 2005; Wolpert et al., 2003] that also considers how we can estimate others’ mental states by comparison of predicted and actual sensory feedback.

Perner [1991] classified mental state representation abilities into a hierarchy that included secondary representations. These sit between primary representations (that reflect relatively well-grounded models of reality, e.g. perceptions) and meta-representations (mental states that represent mental states such as belief attributions). Secondary representations consist of models of reality. They are decoupled from the real world in some way but help to predict or understand its behavior [Suddendorf & Whiten, 2001]. Secondary representations are necessary for processes such as causal understanding, object permanence and symbolic reasoning.

Action may also be represented at a secondary level within motor control structures. The “meaning” of an action may be constituted by a learnt understanding of how the character of a generic form of an action is affected by differing contextual and emotional factors. For example, a wave-good-bye has a generic form in the sense of it being a hand moved in a rhythmic fashion as the palm is held up, facing out. The manner of a wave then varies according to a wide variety of situational, emotional and cultural contextual influences in which it takes place. These might include such diverse influences as whether the relationship is intimate, whether the separation is to be brief or prolonged, whether the departure is amicable, the age of recipient, the distance between participants and the local cultural norms.

Imitation of a model by an observer allows the observer to form the same associations between action plans and memories in the observer as those that control the model’s actions. Thus, through imitation, the observer identifies differences as well as similarities between different forms of an action (such as waving) and relates these to emotional and contextual differences through experience. Consequently, imitation results in an observer developing an understanding of how emotion and contextual memory impact upon the basic form of action.

Imitation may therefore serve to develop a representational knowledge by a continuous process of self–other comparison. In this way mentalizing is not just about simulation, but also about understanding why individuals differ from one another, and how differences between other people’s behavior stem from different motives. Tomasello, Carpenter, Call, Behne, and Moll [2005] have discussed how the experience of sharing intentions and goals forms a basis for the development of social cognition. The suggestion here is that the notion of shared experience should be broadened to include all secondary representations of action, and also that mirror neurons may play an important role in relating differences as well as similarities in experience, to differences and similarities between observed and experienced actions.

If secondary representational models of action are developed through forming memories of an action’s motor codings and its emotional and contextual associations, we would perhaps expect their neural basis to depend upon connectivity between premotor, limbic and orbitofrontal systems. Indeed, these systems meet each other in medial prefrontal cortex, and this is also the primary location in the brain for ToM function [Frith & Frith, 2000; Amadio & Frith, 2006].

Within this alternative model, therefore, the contribution made by mirror neurons to ToM development occurs through imitation learning but not just by ‘simulation.’ Self–other differences are also important. Furthermore, mirror neurons are indirectly involved by serving imitation, which then facilitates development of ToM by fostering connectivity between motor, limbic and OFC, particularly in the medial prefrontal cortex.

**Secondary Representation of Action and Autism**

In autism, secondary representation that is embodied in action seems to be particularly affected, whereas cognition that is disembodied from action such as that involved in recognizing oneself in a mirror and object permanence may be spared. Language and symbolic understanding are relatively unaffected in Asperger’s syndrome. The most diagnostically discriminative features of autism, identified by the Autism Diagnostic Interview-Revised (ADI-R) [Lord et al., 1994] and Autism Diagnostic Observation Schedule-Generic (ADOS-G) [Lord et al., 2000] concern secondary representation of action and include gesture, imitation, following and directing attention and imaginative play. Imperative pointing (pointing to things to ask for them) is less discriminative than declarative pointing (pointing to direct attention).

Moreover, it appears that when secondary representations of action require intentional execution, this is particularly diagnostic of autism. Therefore, expression of emotion in facial expression is discriminative, but whether such emotion is directed toward another individual is more so. During joint-attention activities, following another’s attention discriminates children with autism less than whether they initiate joint attention.
Gesture recognition is impaired [Smith & Bryson, 2007] but gesture production more so. Imitation of gesture is performed less accurately than imitation of actions on objects [Williams, Whiten, & Singh, 2004]. Action planning is impaired [Ozonoff et al., 2004], but intentional understanding is spared [Aldridge, Stone, Sweeney, & Bower, 2000; Carpenter, Pennington, & Rogers, 2001; Hamilton & Frith, 2007].

Part V: Conclusions and Synthesis

Across the spectrum of autism-related disorders, it appears to be the cognitive functions that are embodied by action that are most affected. While communication skills based on gesture, facial expressions and eye movements are diagnostic, those abilities that are less dependent upon integration with action, such as certain sensory, mathematical and mnemonic abilities may be enhanced. It follows that the mechanisms that underpin imitation and self–other matching are fundamental to our understanding of autism. In this article, the relationships between mirror neurons and self–other matching functions in autism have been considered at neurobiological and cognitive levels. Two important issues emerge. One is that different features of actions may be coded for by different groups of mirror neurons. A second is that different social cognitive functions may be differentially served by these different groups. These two issues will be considered in turn.

Action Features

A traditional model conceives of mirror neurons as homogeneous groups of neurons that fire in response to specific actions, either when they are observed or when they are executed. The more the character of an action matches a mirror neurons’ prespecified character, the stronger it will fire. Primate mirror neuron studies have examined action character with respect to features such as grasp type. However, actions also vary for other features that include their speed, force, direction relative to observer, emotional content and relationship to goal and context. As different features of actions are coded for by different brain areas, whether in the parietal or the motor cortex, it follows that self–other matching of these different features is also likely to be served by different groups of cells, either in the classic mirror neuron areas or outside. Therefore, a second model for mirror neuron function, suggested by the work of Dinstein et al. [2007], is that different populations of cells with mirror properties, each serve self–other matching for separate action features. A simple extension of this model is that there are also other groups of cells serving aspects of self–other matching functions that are more independent of action and lie beyond the mirror neuron system, in areas such as the superior frontal gyrus for joint attention, and temporal pole, anterior cingulate and insula for aspects of emotions [Gallese et al., 2004].

Social Cognitive Functions

A further issue that follows from this more complex model of mirror neuron function is that the different cell groups with mirroring functions are likely to be differentially engaged in the various social cognitive functions of relevance to autism. The role of mirror neurons in four main elements of social cognitive development has been discussed. Firstly, mirror neurons are likely to be involved in empathy, emotional contagion and “automatic imitation,” where pre-existing motor knowledge is “released” by observation of others’ actions. Secondly, mirror neurons may be important for mediating influences of observed actions on attentional systems. This could occur by engaging mirror neurons that code the direction of action relative to the self. Influences of the mirror neuron system on attentional systems may be important for development of joint-attention skills and fostering the development of attentional salience for personally relevant actions. Thirdly, mirror neurons may be important for imitation learning, whereby the motor repertoire is developed through experience of self–other comparison. Fourthly, mirror neurons may be involved in a ToM process that differs from empathy by drawing upon an accumulated knowledge of action obtained through self–other comparison.

Prediction and Tests

Mirror neurons may therefore serve a range of self–other comparison functions, with the result that self–other comparison can show variable profiles of impairment in different individuals in autism. Both of two novel ideas contained herein are readily testable. The first is that autism can be associated with a specifically reduced tendency to discriminate between self-directed and other action, and that this is mediated by a reduced function of Broca’s area, perhaps by reduced connectivity with the orbitofrontal–amygdala circuit. The second is that meta-representational ToM and empathy develop through different mechanisms, though both involve the mirror neuron system. Empathic impairment in autism will be predicted by reduced tendency toward automatic imitation. Meta-representational impairment will be associated with impairment on aspects of motor learning and imitation. Further separation seems unlikely as both meta-representational and empathic development will be affected by social attentional impairment. A key issue that remains to be addressed concerns whether there are specific features of action for which imitation is more specifically impaired. A prediction that follows from the model of learning described here will be that people with
autism may particularly struggle to capture differences between generic and context-specific forms of action during imitation. For example, imitating a gesture such as “thumbs-up” for “okay” may not be such a problem for people with autism, as is learning to imitate “thumbs-up” with the nuances that convey just how “okay” things are. Developing experimental methods that can capture these more subtle effects presents a challenge, though the “emphatic gesture” component of module 4 of the ADOS-G [Lord et al., 2000] has already demonstrated some success.

This self–other comparison/mirror neuron model of autism does not make any assumptions about causes of dysfunction. Important potential causes include impairment of the orbitofrontal–amygdala circuit [Bachevalier & Loveland, 2006] memory [Minshew & Goldstein, 2001], differential development of white matter pathways or variable lateralization of function. Serotonin may be important in modulating differential responses to self-directed and other action [Williams et al., 2007a], and the suggestion has been made that Broca’s area could be hypermasculinized in autism [Baron-Cohen et al., 2006].

In summary, this model offers several avenues to be explored, both in testing the model and developing strategies to diminish autism-related disability. For example, it is possible that the influence of stimulus-driven attention could be enhanced pharmacologically [Corbetta & Shulman, 2002]. Aspects of social skill training that may be worthy of development include training motor skills to express emotion and developing motor awareness. Further research into the self–other matching function in autism promises to be a fruitful line of research.

Acknowledgments

This research is supported by the Northwood Charitable Trust. I am also grateful to Nina Williams, David Perrett, Morven McWhirr, Michael Arbib, and Sally Rogers and to two anonymous reviewers for helpful comments on earlier drafts.

References


