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The Simulating Social Mind: The Role of the Mirror Neuron System and Simulation in the Social and Communicative Deficits of Autism Spectrum Disorders

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The mechanism by which humans perceive others differs greatly from how humans perceive inanimate objects. Unlike inanimate objects, humans have the distinct property of being "like me" in the eyes of the observer. This allows us to use the same systems that process knowledge about self-performed actions, self-conceived thoughts, and self-experienced emotions to understand actions, thoughts, and emotions in others. The authors propose that internal simulation mechanisms, such as the mirror neuron system, are necessary for normal development of recognition, imitation, theory of mind, empathy, and language. Additionally, the authors suggest that dysfunctional simulation mechanisms may underlie the social and communicative deficits seen in individuals with autism spectrum disorders.

Keywords: mirror neuron system, autism spectrum disorders, simulation, social impairments, communication impairments

> "When we see a stroke aimed, and just ready to fall upon the leg or arm of another person, we naturally shrink and draw back on our leg or our own arm . . . The mob, when they are gazing at a dancer on the slack rope, naturally writhe and twist and balance their own bodies, as they see him do."
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> —Adam Smith, *The Theory of Moral Sentiments*

Over 200 years ago, Smith (1759) described the perception of others not simply in terms of simple observation but rather a result of active embodied understanding. Unlike inanimate objects, for which actions are processed visually and can be successfully predicted based on physical characteristics and physical laws, this type of processing—*systemizing* (Baron-Cohen, 2002)—is not sufficient for understanding human behaviors, because they are motivated by internal states that typically do not follow objectively predictable patterns. Thus, many researchers have proposed a simulation account of human perception (Barsalou, 1999; Gallagher & Meltzoff, 1996; Gallese, 2001; Gallese & Goldman, 1998). Though each researcher uses his or her own terminology, all generally claim that the understanding of human actions and internal states relies on both the capacity of the observer to perceive other humans as "like me" (Meltzoff & Moore, 1995) and the capacity to simulate the observed actions and internal states of other humans within the observer's own motor, cognitive, and emotional representations.

This simulation account proposes that when typically developing individuals perceive another person in a certain situation, they will automatically and unconsciously project that perception back onto the observer's own motor, cognitive, and emotional representations in order to run an offline simulation (Gallese, 2003). This offline simulation, in turn, allows the observer to create an embodied understanding of the observed person's behaviors, thoughts, and feelings. Thus, according to this view, it follows that the mechanisms by which we understand an action, thought, or emotion in another individual share an underlying neural circuitry with the mechanisms by which we execute such actions, thoughts, or emotions ourselves—what Gallese (2001) calls the "shared manifold of intersubjectivity" (p. 34) or "Intentional Attunement" (Gallese, 2004). Similarly, in the theory of mind literature, the simulation theory proposes that the observer performs an internal simulation of the perceived actions and then uses knowledge of his or her own actions and intentions in such a situation to infer others' mental states (Gordon, 1986; Heal, 1986). Finally, from the motor control literature, computational models describe cells called *emulators*. Emulators receive an efferent copy of the motor command and produce a simulation of the sensory signals that would be produced in the organism as a result of that same command (Ito, 1984). Emulators would theoretically aid in motor control, prediction of actions, and motor imagery (Grush, 2004).

Barsalou (1999) united the aforementioned simulation theories with his perceptual symbol systems (PSS) framework. The first tenet of this theory is that knowledge (or internal representations) regarding perceptions, actions, and introspective states are represented in the same systems in which they are experienced. According to PSS, neurons, called *conjunctive neurons*, receive efferent copies of the input signal from all of the senses (including

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vision, audition, olfaction, gestation, haptics, proprioception, and introspection) and store it for future cognitive use. Once these so-called conjunctive neurons are established, they can be activated by cognitive simulation in the absence of bottom-up input. PSS also establishes two central constructs: simulators and simulations. *Simulators* are distributed networks of conjunctive neurons that activate in response to a specific category. They are responsible for integrating the modality-specific information and forming a supramodal representation of a concept. Once a simulator is established for a certain category it enables the individual to reenact its content in the form of simulations. *Simulations* can be used to draw inferences about a given concept or to internally represent it through memory, language, and thought.

The original framework was extended by Barsalou (2003) to provide a mechanism by which these simulated concepts are incorporated into specific contexts. In this system, *situated conceptualizations* are proposed to contain simulations of four basic components of a context: people and objects, actions and body states, introspective states, and settings. According to PSS, each component of the conceptualization is simulated in the respective brain areas that originally encoded the information. Just as the simulators for a concept integrate the activity of the conjunctive neurons to formulate supramodal representations of that concept, the simulators for each component combine to form a supramodal simulation of the situation. Barsalou proposed that this simulation, which incorporates multiple core components of a context, allows the conceptualizer to have the experience of being in the situation. Barsalou's PSS framework brings together all of the simulation literature across the multiple domains and provides a unified theory for how humans interpret and interact with their environment.

Though the simulation account has a long history (dating back to at least 1759) and is intuitively appealing, debate continues over the theory's validity in multiple domains. Additionally, it is unclear whether the underlying neural circuitry that mediates the simulation processes for perception, social interaction, and communication is centered in the motor system or whether multiple brain regions are capable of simulation. Recent research, which spans psychology, linguistics, cognitive science, and neuroscience, has begun to answer some of these questions. These answers also give hope for finding a possible neural basis for many of the behavioral deficits seen in autism spectrum disorders (ASD; including autism, Asperger's syndrome, and pervasive developmental disorder–not otherwise specified).

In this review, we argue that when simulation processes are deficient, one is left with an individual with qualitative impairments in social interaction and communication skills, two of the three defining characteristics of ASD. Though rule-based knowledge clearly may influence how people perceive and interpret their environment, it cannot be sufficient, as evidenced by the deficits in ASD. Individuals with ASD have no difficulty understanding rule-based accounts of the environment (Baron-Cohen, 2002). In fact, high-functioning individuals with ASD often have superior systemizing ability (Baron-Cohen, Richler, Bisarya, Gurunathan, & Wheelwright, 2003).

Although researchers note that individuals with ASD have a clear impairment in understanding and interacting with other individuals, the underlying mechanisms that may mediate the spectrum of deficits in the areas of perception, social interaction, language, and behavior have remained a mystery. Historically, theories have focused on either (a) behavioral accounts (e.g., Baron-Cohen, 1995; Frith, 2001) in which one behavioral deficit is explained in terms of other behavioral deficits or (b) neuroanatomical accounts that implicate individual regions in the brain such as the amygdala or cerebellum (e.g., Baron-Cohen et al., 2000; Courchesne, 1997). These theories have had enormous hueristic appeal and must be considered in future theories, but one limitation of strictly behavioral and strictly neuroanatomical accounts is that neither explains the underlying functional mechanisms that mediate the deficits.

The most informative theories are those that are able to link neuroanatomy and functional mechanisms to the behavioral impairments that are unique to ASD. One theory, which does exactly this, suggests that a dysfunction in a specific functional system, the mirror neuron system (MNS), underlies the behavioral impairments in ASD (Altschuler et al., 2000; Williams, Whiten, Suddendorf, & Perrett, 2001). Our group has provided preliminary evidence for this theory on the basis of both a case study (Altschuler et al., 2000) and a follow-up study including 10 individuals with ASD (Oberman et al., 2005). Though we believe this theory is on the right track, there is no reason to believe that the MNSs in premotor and parietal cortices are the only systems in the human brain that have mirror or simulation properties. In this review, we propose a more comprehensive explanation implicating a deficit in simulation. As this review shows, there are multiple brain regions (including those that have been implicated in anatomical accounts of ASD) that appear to have simulation properties. Thus, the simulation system, similar to the immune system may be functionally modular but localized in multiple brain regions rather than a single region—a similar theory to the intentional attunement theory proposed by Gallese (2006).

This review discusses behavioral, neurophysiological, neuromagnetic, and neuroimaging findings that independently and collectively support the simulation view of perception, theory of mind, empathy, and language, all of which are essential skills for understanding and interacting with the environment in which we live and are core deficits in ASD. Though other views can account for some of these findings, our goal is not to provide a complete review of each of the alternative accounts but rather to stress that in all cases, the simulation view accounts for the data in the most parsimonious manner.

Perception and Imitation of Actions

Evidence for the Role of Simulation in Typically Developing Individuals

Though anecdotal references to simulation have been recorded as early as 1759, this view was made popular by philosophers including William James and Rudolph Hermann Lotze in the late 1800s, as evidenced by the following quotations:

> The spectator accompanies the throwing of a billiard ball, or the thrust of a swordsman, with slight movements of his arm; the untaught narrator tells his story with many gesticulations; the reader, while absorbed in the perusal of a battle scene, feels a slight tension run through his muscular system, keeping time as it were with the actions he is reading of. (Lotze, 1852; as quoted in James, 1890, p. 525)

> An educated man . . . on entering his house one day he received a shock from crushing the finger of one of his little children in the door. At the moment of his fright he felt a violent pain in the corresponding finger of his own body, and this pain abode with him three days. (James, 1890, p. 712)

Behavioral studies as early as those performed by Darwin indicate that when individuals are in the presence of others, the observer tends to synchronize his or her movements to match those of the others (Condon & Ogston, 1967; Darwin, 1872/1965; Kendon, 1970). More specifically, people tend to mimic others' gestures and body postures (Chartrand & Bargh, 1999), facial expressions (Dimberg, 1982; Dimberg et al., 2000; Wallbott, 1991), tone of voice and pronunciation patterns (Goldinger, 1998; Neumann & Strack, 2000), as well as breathing rates (McFarland, 2001; Paccalin & Jeannerod, 2000).

It has been proposed that the increased likelihood of one performing a certain behavior when observing the same behavior in another individual is a result of automatic stimulus–response relationships, similar to priming in which the presence of the stimulus increases the probability of a response. Along these lines, James (1890) stated that "every representation of a movement awakens in some degree the actual movement which is its object" (p. 526). Another view suggests that the tendency toward mimicry can be accounted for by contagion similar to contagious yawns or laughter (Hatfield, Cacioppo, & Rapson, 1994; Laird et al., 1994). Neither of these accounts necessarily suggests that observation and execution of actions share a common mechanism, as required by strict simulation theories. Other recent behavioral studies, however, have provided evidence that in addition to action observation influencing the occurrence of concurrent execution of that same action, the performance of an action influences the concurrent perception of that action. A simple stimulus–response relationship or contagion accounts have less explanatory power in this case. The finding that action execution and action observation reciprocally modulate each other is taken as evidence for the simulation account of mimicry—that is, behavioral mimicry occurs because action observation and action execution are mediated by overlapping neural mechanisms.

One piece of evidence for a shared action observation–execution representation is found in a study conducted by Pietromonaco, Zajonc, and Bargh (1981). In this study, participants were asked to remember a set of 78 predominantly neutral faces (taken from a yearbook) while the participants either imitated the head and facial configuration, made simple judgments about the stimuli, squeezed a sponge, or chewed gum—a manipulation designed to prevent imitation. Results indicated that participants asked to imitate the faces performed the best at the later memory test (73% correct), whereas those who squeezed a sponge were at 65%, those asked to make judgments were 64% correct, and those who chewed gum (and thus were not able to imitate) were 59% correct. Unfortunately, this study leaves many questions unclear. First, the study dealt with delayed memory of the expressions, so it is impossible to determine whether mimicry aided the initial perception of the stimuli or the encoding of the stimuli into memory, rehearsal, or retrieval. Second, it is possible that differences in task motivation or attention could account for the differences in memory between the three conditions. Still, despite these interpretational and empirical ambiguities, this pioneering empirical work continued to inspire research on the role of somatic feedback in perception.

Reed and Farah (1995) also investigated the influence of motor action on perception. They, however, used arm and leg actions rather than facial expressions. Reed and Farah's studies showed significant improvements in recognition of other's actions when the observer moved the same limb. In other words, if the observer was moving his own arm, he was more likely to recognize that the confederate moved her arm than her leg. This finding held up even when selective attention and conscious mimicry were controlled. Reed and Farah concluded that the participants used the same body schema to process both their own and others' movements. The same conclusion was also drawn by Ramachandran and Rogers-Ramachandran (1996) when studying patients with anosognosia that was due to damage to the right parietal and frontal cortices. These patients denied not only their own paralysis but also the paralysis of another individual. The authors concluded that damage to an individual's own body schema may lead to deficits in making judgments about another individual's actions, possibly as a result of damage to a specific system of neurons, namely the MNS, which is discussed later in this review.

Taken together, these behavioral studies support the conjecture that there is considerable overlap between the neural mechanism for action observation and action execution. However, behavioral studies are not able to speak to how this shared representation aids in recognition. This task has been undertaken by researchers in computational neuroscience who have developed models of neural networks that account for the role of simulation, or what they call *emulators* in the motor system.

An *emulator* is defined as "a device that mimics the input/output operation of a target system" (such as the motor system; Grush, 2004, p. 387). Grush (2004) suggested that an emulator is beneficial in many ways. First, it allows an individual to control his or her actions online and inhibit inaccurate or inappropriate actions prior to their occurrence. More important for this review, however, is its role in perception. Grush proposed that perception is a result of activation in an emulatory loop that receives input and mimics both the input from the senses and feedback from the observer's own outputs, thus creating a mechanism that unites perception of the outside world and knowledge of the internal state of the observer.

These so-called emulators have been proposed to exist in the dentate of the cerebellum, according to behavioral, lesion, and neurophysiological studies (Grush, 2004; Ito, 1984; Wolpert, Zoubin, & Flanagan, 2001; see Decety, 1996, for a review). However, the question still remains whether the cerebellum emulators are unique or whether they are a part of a larger network of neural systems that serve to simulate their content. Neurophysiological and imaging studies conducted over the past 50 years appear to support the latter—that is, that multiple regions of the brain are capable of offline simulation.

Electroencephalography (EEG) studies from as early as 1954 demonstrated neural activity in the region of sensorimotor cortex when nonmoving subjects watched other individuals performing specific actions. While studying the changes in brain activity, French researchers Gastaut and Bert (1954) recorded EEG activity while subjects performed actions as well as while they were presented with visual stimuli. Though recordings were taken across the scalp at various frequency ranges, of particular interest

to this review are the electrical changes recorded over the rolandic (sensorimotor) region. Gastaut (1951) had previously reported that oscillations recorded over this region, in a frequency close to the alpha frequency (7–11 Hz), were reduced in amplitude when subjects performed a directed action or simply shifted their posture.

Just 3 years later, Gastaut and Bert (1954) found that the so-called rolandic *arceau rhythm* (named for its characteristic arc shape) was also reduced when subjects identified themselves with an active person represented on a screen, for example, when they viewed a film of a boxing match. Gastaut and Bert concluded that "the relation between the blocking of the 'arceau' rhythm and the image of boxers in action is unquestionable." It is currently thought that suppression of this rhythm represents increased activity in the neural networks located in the rolandic region (Andrew & Pfurtscheller, 1997). Thus, as early as 1954, there was neurological evidence that the observation of actions in others activates neural systems in the observers' sensorimotor systems, evidence for the role of simulation in visual perception.

Over 40 years later, Altschuler, Vankov, Wang, Ramachandran, and Pineda (1997) corroborated Gastaut and Bert's (1954) finding by showing a reduction in power of the arceau rhythm (now referred to in the standardized Greek naming system as the *mu wave*) during the execution, imagination, and observation of human action and suggested that the suppression of the mu wave could be used as an EEG index of mirror neuron activity. Cochin, Barthelemy, Lejeune, Roux, and Martineau (1998) replicated this finding, showing mu wave suppression during the observation of actions performed by humans. No such modulation was present when subjects watched object movements. In a follow-up study, Cochin, Barthelemy, and Martineau (1999) reported that the response in the mu wave when subjects observed another performing an action was not significantly different from the response when subjects performed the action themselves. The authors cited this as evidence that action observation and execution share the same neural network.

Subsequently, multiple laboratories have reported similar results, finding a reduction in the power in the mu wave over central leads during the observation of others' actions, and have supported the link between mu wave suppression and mirror neuron activity (Babiloni et al., 2002; Muthukumaraswamy & Johnson, 2004a, 2004b; Pineda, Allison, Vankov, 2000). Additionally, over the past several years, other techniques have also been successful in identifying activity in the area of the sensorimotor cortex during action observation. Hari and colleagues (Avikainen, Forss, & Hari, 2002; Hari et al., 1998) have successfully used magnetoencephalography to measure the activity of the precentral gyrus following stimulation of the median nerve. Beta frequency rebound over sensorimotor cortex showed a significant reduction during both action execution and action observation. The readiness potential, an event-related potential (ERP) marker of motor preparation recorded over the central leads prior to movement onset, has also been shown to be modulated by observed actions (Kilner, Vargas, Duval, Blakemore, & Sirigu, 2004). Furthermore, when the nature and onset of action is predictable, the rise of the readiness potential precedes the observed movement's onset. Kilner et al. (2004) proposed that this type of timing might allow the observer not only to react to others' actions but also to anticipate actions that will be performed in the near future.

Though electrophysiological recordings from the scalp give us broad estimates of neural systems involved in certain behaviors, their spatial resolution is less than satisfactory for exact localization of neural mechanisms. The ideal technique for such research is to record directly from individual neurons in awake human volunteers, but such an opportunity is rarely available to researchers. Alternatively, animal studies on closely related species can be incredibly informative for such an investigation. The macaque monkey has been the prime subject for investigation of the mechanisms underlying action observation and execution.

The most relevant finding in macaque single-unit electrophysiology research was made by Giacomo Rizzolatti and his colleagues (Di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992; Fogassi, Gallese, Fadiga, & Rizzolatti, 1998; Gallese, Fogassi, Fadiga, & Rizzolatti, 2002). While studying the premotor cortex in the macaque, they came across a system of neurons in area F5 that responded not only when the monkey performed an action but also when the monkey watched the researcher perform a similar action (Di Pellegrino et al., 1992). The team named this system of neurons the *mirror neuron system* because it appeared that the observed action was mirrored or simulated within the monkey's own motor system. In addition to the original mirror neurons found in the macaque's premotor cortex, neurons in the inferior parietal lobule have also been found to have mirror properties (Fogassi et al., 1998; Gallese et al., 2002).

The discovery of the MNS gained immediate attention and raised two questions: Does a similar system exist in humans? And could this be the elusive neural mechanism subserving the action observation–execution link that enables humans to imitate?

Though few researchers referred to this system by name prior to the late 1990s, there was already a large body of behavioral, physiological, and lesion data (discussed earlier in this review) supporting the existence of the MNS in humans. However, these techniques gave little clue to the spatial location of such a system. The first attempt to localize the human MNS was in a study conducted by Fadiga, Fogassi, Pavesi, and Rizzolati (1995). Using transcranial magnetic stimulation (TMS), Fadiga et al. investigated whether the premotor cortex in humans responded when the participants watched others' actions. It was determined, on the basis of anatomical cytoarchitecture, that the homologous region to the macaque F5 is human Brodmann's area 44/45, also known as Broca's area. Fadiga et al. found that TMS applied over Broca's area resulted in greater motor-evoked potentials (MEPs) when the subject observed another person moving as compared with a baseline rest condition. Furthermore, the pattern of muscle activation evoked by TMS during action observation was very similar to the pattern of muscle contractions present during the execution of the same action.

Subsequent to this neuromagnetic study, positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) studies (Decety et al., 1997; Iacoboni et al., 1999) showed selective activity in the frontal operculum (Brodmann's area 44/45) and the anterior parietal cortex when subjects watched human actions. Further, this activity, similar to the macaque correlate, was somatotopically distributed in both premotor and parietal regions (Buccino et al., 2001).

Two additional regions of the human cortex that appear to have mirror properties have recently been discovered. The first, the superior temporal sulcus (STS), was originally identified for its

selective response to the observation of biological motion in monkeys (Oram & Perrett, 1994; Perrett, Harris, Mistlin, & Hietanen, 1990) and in humans (Bonda, Petrides, Ostry, & Evans, 1996; Puce, Allison, Bentin, Gore, & McCarthy, 1998). More recent studies have also indicated that this region has a somatotopic representation with the observation of hand actions activating inferior posterior regions, mouth actions activating mid posterior regions, and eye movements activating superior posterior regions (Pelphrey, Morris, & McCarthy, 2005). In addition to its visual properties, human STS also responds to the imitation of an action (Iacoboni et al., 2001). This activation is greater during imitation than during control motor tasks and continues to respond even when the subject's view of his or her hand is obscured. The second region is the so-called extrastriate body area. The extrastriate body area was first classified by Downing, Jiang, Shuman, and Kanwisher (2001) on the basis of its response to the visual perception of human bodies and body parts. A recent study conducted by Astafiev, Stanley, Shulman, and Corbetta (2004) report fMRI activation in this region in response to the subject's moving his or her own arm or leg toward a target in the absence of visual feedback. This activation was also present after attention and sensory properties of the target were controlled. Thus, it appears that multiple regions of the human brain may be capable of motor simulation (inferior frontal gyrus, inferior parietal lobule, superior temporal sulcus, extrastriate body area, and the dentate of the cerebellum).

The aforementioned findings provide strong evidence that an action observation–execution matching system, similar to that found in the macaque premotor and parietal cortices, albeit in a more extensive system, exists in the human brain. Since the original studies, specific properties of the human MNS have been investigated. Studies have reported that unlike the macaque system, human mirror neurons respond to goal-directed, non-goal-directed, and pantomimed actions (Buccino et al., 2001; Grezes, Armony, Rowe, & Passingham, 2003), whereas the monkey system responds only to goal-directed actions (Gallese, Fadiga, Fogassi, & Rizzolati, 1996).

Additionally, human mirror neurons are selective to actions within the observer's motor repertoire. In other words, if the observer is unable to match the observed action to a motor representation within his or her own system, the mirror neurons will not respond (Buccino et al., 2004; Stevens, Fonlupt, Shiffrar, & Decety, 2000). The individual need not be familiar or skilled at the action but only physically capable of performing it. For example, actions such as grasping and biting, which humans share with other primates, will activate the human MNS whether the observed action is performed by a human or a macaque. However, observing a dog barking, which is not part of the human motor repertoire, does not activate this system but rather is processed in lower level perceptual systems (Buccino et al., 2004). Furthermore, actions that are part of the human motor repertoire but are not familiar will not activate the MNS as much as actions that are familiar to the observer. This property was demonstrated in a study conducted by Calvo-Merino, Glaser, Grezes, Passingham, and Haggard (2005), who recorded fMRI data from expert dancers and found increased activity to the observation of others performing familiar styles of dance movements, as compared with unfamiliar styles matched for low-level visuomotor properties.

Though it is hard to argue against the existence of a human MNS, its function has yet to be clearly delineated. In their original article, Di Pellegrino et al. (1992) proposed that the MNS may help an observer understand the actions of another by mapping them onto his or her own motor representations. Additionally, studies have demonstrated that when human subjects are instructed to observe actions with the intent to imitate (as opposed to remember), the MNS is selectively involved in the imitation condition (Decety et al., 1997). This finding has led researchers to add imitation to the list of functions of the MNS. Critics, however, are quick to point out that macaques and many other primates do not typically show imitation behaviors (Visalberghi & Fragaszy, 2001; Whiten & Ham, 1992) but do have mirror neurons. Thus, this system cannot be sufficient for the ability to imitate, but it may have been an evolutionarily necessary step that led to the ability to imitate in humans (Arbib, 2005; Ramachandran, 2000).

In a review article, Iacoboni (2005) outlined his theory of the neurological basis of imitation. He suggested that the circuit begins in the superior temporal cortex in which the visual properties of the observed action are coded. The signal is then sent to the posterior parietal cortex that provides somatosensory information regarding the observed action by means of the parietal mirror neurons. Finally, the signal is sent to the frontal mirror neurons to code the goal of the action to be imitated. Before the action is ultimately sent to the primary motor cortex, however, an efferent copy of the motor command is sent back to the STS in order to match the predicted sensory consequences of the planned imitation to the visual description of the original observed action. If there is a good match, the action is initiated by the primary motor cortex. This series of connections has strong anatomical support from nonhuman primate studies (Jeannerod, Arbib, Rizzolatti, & Sakata, 1995; Rizzolatti, Luppino, & Matelli, 1998; Seltzer & Pandya, 1994; Taira, Mine, Georgopoulos, Murata, & Sakata, 1990).

A PET study (Grezes, Costes, & Decety, 1998) supported the activation of the dorsal visual stream extending up into premotor cortex while subjects watched meaningful actions with no instructions, as well as both meaningful and nonmeaningful actions with the intent to imitate at a later time point. Additionally, Ruby and Decety (2001) reported findings from a PET study finding that simulation of both self and other actions activate the inferior parietal cortex in the area of inferior parietal lobule where mirror neurons have been identified. Thus, although the MNS may not be sufficient to mediate imitation in the macaque, it is possible that, through evolution, the human MNS with the endowed ability to match action observation and action execution utilized this system for imitation.

The aforementioned data in both macaque single-unit physiology and human imaging provide convincing evidence that regions of the cerebral cortex primarily designated as part of the motor system are activated by the observation of others' actions. Furthermore, this activity is thought to aid in visual recognition and imitation. Thanks to the discovery of the MNS, the simulation view of perception has gained new momentum and inspired collaboration between behavioral psychologists, cognitive scientists, and neuroscientists. One question that has yet to be answered, however, is whether the impairments in perception and imitation as seen in individuals with autism spectrum disorders are a result of dysfunction in these simulation systems.

Role of Simulation in the Perception and Imitation Deficits of ASD

A multitude of studies performed over the past several decades (reviewed by Williams, Whiten, & Singh, 2004) have suggested that children with autism suffer from impairments in imitation. As imitation is thought to be a critical building block for early affective, social, and communicative development, an impairment in this domain can have quite deleterious consequences to a developing child (Rogers & Pennington, 1991). The first suggestions of an imitation deficit in ASD was made in 1953 (Ritvo & Provence, 1953) with an anecdotal report of a mother describing the inability of her 21-month-old to make pat-a-cake simply from watching her. The only way the child could learn the game was to have the mother hold his hands and put them through the appropriate movements. Since this time, dozens of empirical studies have been published detailing the specific characteristics of the imitation deficits seen in ASD.

The first review of this literature was conducted by Rogers and Pennington (1991), who found strong evidence for the existence of an imitation deficit affecting simple body movements and actions with symbolic meaning. What became very apparent as more studies were conducted was that the imitation deficits in ASD varied on the basis of the specific task the child was asked to perform and the child's age. For example, it has been suggested that the development of language might be necessary to derive benefit from the symbolic meaning of a gesture in an imitation task that includes meaningful gestures (Williams et al., 2004). Thus, older children and those with better language skills will perform better on imitation of symbolic gestures as compared with younger children or those with poor language skills (Green et al., 2002; Rogers, Bennetto, McEvoy, & Pennington, 1996). Second, imitation of actions with objects appears to be relatively less impaired compared with actions without objects in individuals with ASD (Aldridge, Stone, Sweeney, & Bower, 2000; Hammes & Langdell, 1981; Roeyers, Van Oost, & Bothuyne, 1998; Sigman & Ungerer, 1984). Finally, a common characteristic of the imitation deficit in individuals with ASD is reversal errors (that is, producing the movement with a 180° transformation). Reversal errors are also commonly seen in typically developing preschoolers (Ohta, 1987), suggesting that the imitative deficit in ASD may be characterized as a delay of normal development rather than an absolute deficit (Whiten & Brown, 1999).

Though recognized over 50 years ago (Ritvo & Provence, 1953) and studied in detail, the cause the imitation impairment has yet to be identified, though many theories exist, including Curcio's (1978) symbolic representation hypothesis, Jones and Prior's (1985) dyspraxia hypothesis, Rogers and Pennington's (1991) self-other representation hypothesis, and Trevarthen and Aitken's (2001) motivation hypothesis. The most recent review of the literature conducted by Williams et al. (2004) suggested that Rogers and Pennington's hypothesis of a deficit in self-other mapping explains the findings from the empirical studies much better than the other proposed theories. Williams et al. suggested that typically developing individuals automatically and unconsciously use the same mechanism to process both self and other actions and use this mechanism for imitation, whereas Rogers and Pennington's review (1991) suggested that infants with autism lack the ability to "form and coordinate social representations of self and other via amodal or cross-modal representation processes" (p. 137). They further suggested that this deficit in self–other mapping leads not only to their imitation impairments but also to their other social–communicative impairments.

If Williams et al. (2004) are correct, and the imitation deficits are not a result of impaired perception or impaired motor skills, but rather self–other mapping impairments underlie the imitation deficits, then it is plausible that the MNS, which appears to be involved in imitation in typically developing individuals, may also be the neurological basis of the imitation deficits in ASD. Five independent research groups have reported findings supporting this proposal. The first study to find evidence for MNS impairments in ASD was conducted by Altschuler et al. (2000), who recorded mu wave suppression in one child with autism. Preliminary results showed a lack of suppression to the observation of actions in others, suggesting a possible impairment in the MNS. In a follow-up study from the same laboratory, Oberman et al. (2005) corroborated this finding by demonstrating an absence of mu wave suppression in a sample of 10 individuals with ASD while they watched videos of another person's actions. Although typically developing individuals showed significant mu-wave suppression, indicating normal MNS functioning, participants with ASD showed no significant change in mu power from a baseline condition.

Nishitani, Avikainen, and Hari (2004) also found evidence of an impairment in the MNS using magnetoencephalography. Subjects were presented with still pictures of a woman performing orofacial gestures and were instructed to imitate these gestures. Cortical activations were recorded from 8 adult subjects with Asperger's syndrome and 10 control subjects. In both groups, activations were recorded over occipital cortex, superior temporal sulcus, inferior parietal lobe, inferior frontal lobe, and primary motor cortex. Though the two group's activations were similar for occipital cortex, superior temporal sulcus, and inferior parietal lobe, activations in inferior frontal lobe and primary motor cortex were weaker and had a greater latency in the Asperger's syndrome group as compared with the control group. These findings suggest that the deficit is not in low-level visual processing, but rather in higher order cognitive processes in the prefrontal regions. Similarly, Villalobos, Mizuno, Dahl, Kemmotsu, and Muller (2005) found that area 44, the prefrontal mirror neuron area, had reduced functional connectivity with primary visual cortex in individuals with autism, as compared with matched controls. Both groups concluded that their findings provided evidence for impairment in the prefrontal MNS in individuals with ASD.

Another group (Theoret et al., 2005) recorded TMS-induced MEPs while subjects watched videos of finger movements that were directed either toward or away from the observer. In the control group, both types of actions resulted in increased MEPs recorded from the observer's right first dorsal interosseus and abductor pollicis brevis muscles. The clinical group, consisting of individuals diagnosed with ASD, showed increased MEPs only to actions directed toward the observer (self-directed or allocentric) and no significant change from baseline in the away (other directed or egocentric) movement condition. The researchers explain this result in terms of a mirror neuron deficit leading to impairment in simulating egocentric actions and a general self–other representation deficit.

Most recently, Dapretto et al. (2005) published a study in which they used fMRI to investigate activity in the MNS in individuals with ASD. Participants were asked to both imitate and observe emotional facial expressions while experimenters analyzed the blood-oxygen-level dependent (BOLD) signal in regions thought to be part of the MNS. Whereas typically developing individuals showed activation in visual cortices, primary motor, premotor (including the inferior frontal gyrus, MNS region), limbic, and cerebellar regions, individuals with ASD did not show this pattern and, specifically, showed no significant activation of the inferior frontal gyrus. Additionally, the activity that was observed in the MNS regions in individuals with ASD was correlated with symptom severity, as indexed by the Autism Diagnostic Observation Schedule (Lord et al., 1989) and the Autism Diagnostic Interview (Lord, Rutter, & Le Couteur, 1994).

The aforementioned studies represent the developing body of literature supporting the role of an impaired premotor MNS in individuals with ASD. One of the first proposed, and most strongly supported, functions of the MNS is to support internal simulations of actions. Without the ability to internally simulate the visual perception of an action, motor imitation would likely be more difficult and performance would likely be abnormal. We would thus propose that the imitation deficits seen in individuals with ASD are best explained by their impairments in simulation caused by an impaired MNS.

Theory of Mind and Empathy

From Imitation to Theory of Mind and Empathy in Typically Developing Individuals

Arguably more socially relevant than understanding an action's motor properties and being able to reproduce them (imitation) is the ability to understand the thoughts, intentions, and emotions that guide the observed action. Over the past 25 years, researchers in the areas of developmental psychology, cognitive science, and philosophy have devoted their careers to understanding how humans learn to attribute thoughts and intentions to others, referred to as *theory of mind* (TOM; Humphrey, 1976; Premack & Woodruff, 1978). Despite the over 25 years of research, there is still much debate over the underlying mechanism that leads to this ability. Thus, a vast literature has been developed by researchers proposing, testing, dissecting, and criticizing various theories. A thorough review of this literature, however, would be outside of the scope of this article. Thus, an overview of the two main competing theories is presented below (the reader is referred to Carruthers & Smith, 1996; Davies & Stone, 1995, for a balanced review of the debate).

The two major competing theories of the development of TOM are theory–theory and the simulation theory. The theory–theorists have suggested that individuals develop TOM over the first few years of life by testing given rules regarding the functions of the objects and organisms with which they interact as a child and eventually come up with a cognitive theory of what others are thinking (Gopnik & Meltzoff, 1997). Support for the theory–theories comes from behavioral studies finding that children appear to develop a theory of other minds at around age 3. Also, behavioral studies have found that 3-year-olds make errors about other's knowledge that they do not make about their own knowledge. Finally, Saxe (2005) argued that when errors in others' beliefs are made, the errors reflect knowledge of psychological laws governing how minds work.

Opposing theory–theories are simulation theories that propose that TOM is simply an outgrowth of the ability to interpret others' actions through simulation. By creating an internal simulation, individuals can step into the mental shoes of another person and understand the thoughts, emotions, and intentions behind their behavior (see Gordon, 1986; Heal, 1986; Goldman, 2000, for reviews). In its strongest form, this view implies that understanding others' minds requires no conceptual thinking or theory formation. Similar to the action simulation processes discussed earlier, the simulation theory simply requires that the observer reflect back on his or her own experience and use that knowledge to infer the mental state of the other individual. Simulation theories have been supported by brain-based studies that have found MNS activity in response to the goal or intention of an action and regions of the brain that respond both when subjects are asked to reflect on their own mental state and when they are asked to infer the mental state of another individual, which is discussed later in this section.

Although not traditionally discussed in reference to the simulation view of TOM, the "Mary" scenario provides an excellent example for the necessity of reflection on self-knowledge for the understanding of internal states of others. Jackson (1986) proposed a theoretical question of whether Mary, a color-blind neuroscientist who knows everything about the physiological processing of color but has never experienced it herself, would really know what it is to see red. Jackson proposed that the theory–experience gap would preclude Mary from understanding the internal qualia, or mental content, of the experience of seeing red. This theoretical scenario demonstrates the importance of an individual's own experiences in the understanding of others' mental states. Further, it suggests that simply having the cognitive theory of what others may think or feel in a certain scenario is not sufficient for TOM. Similar to Mary, oftentimes individuals with autism can be taught rules regarding what individuals may think or how they may feel in a certain situation; however, they will often still have difficulty really knowing the other's mental state.

Similar to the simulationist view of TOM, empathy by definition incorporates a simulation of an observed person's internal state. The term *empathy* was originally introduced by Theodore Lipps (1903) and used to describe the feeling one gets when watching an acrobat walking on a suspended wire. Lipps wrote, "I feel myself inside of him" (p. 121). This concept was further elaborated on by Edith Stein (1912/1964) in her book *On the Problem of Empathy*. Stein proposed that empathy is not simply the understanding of other's feelings or emotions in a cognitive or theoretical manner. Rather, empathy is a function of one individual's experiencing the same feelings as another individual through an appreciation of similarity. Most recently, Preston and de Waal (2002) proposed the perception–action model of empathy, which states that the attended perception of an individual's state automatically activates the observer's representations of the state and situation. Further, the activation of these representations automatically primes or generates the associated autonomic and somatic responses unless inhibited.

Strong empirical evidence exists for the role of simulation in the ability to empathize with others' emotions. When subjects are presented with stimuli of others displaying emotional facial ex-

pressions, typically developing individuals will automatically (without instruction to do so) mimic the facial expression of the stimuli (Bush, Barr, McHugo, & Lanzetta, 1989; Dimberg, 1982; Dimberg & Lundqvist, 1988). This mimicry occurs even when the stimuli are subliminally presented (Dimberg et al., 2000). This automatic facial mimicry response has been proposed to facilitate recognition and empathy for the observed emotion through a process of internal simulation of the corresponding facial expression (Lipps, 1907; Niedenthal, Brauer, Halberstadt, & Innes-Ker, 2001; Pietromonaco et al., 1981; Wallbott, 1991).

One study that provided evidence for the simulation account was conducted by Wallbott (1991). In this study, participants were videotaped while they performed an emotional facial recognition task. Each participant was then brought back to the laboratory for a subsequent session and asked to guess, on the basis of the videotape of his or her own face, what facial expression they were watching. The participants' judgments of their own facial expressions were above chance, suggesting that the participants were imitating the facial expressions of the people they were judging. Additionally, a correlation was found between the participants' judgment of the stimuli and their judgment of their own (videotaped) facial expression. Finally, the recognition rate from the original study correlated with individuals' recognition rate of their own (videotaped) facial expressions. Though the first two findings in this study do not discriminate between the emotional contagion and emotional recognition accounts, the correlation between the recognition score from the study and their self recognition score (when they were presented with only their own videotaped mimicry responses) is interpreted as evidence that the facial mimicry in fact aided in the recognition of the facial expressions in others.

Most recently, Niedenthal et al. (2001) provided direct evidence for the role of mimicry in recognition of facial expressions. Participants were asked to hold a pen sideways in their mouths, between their teeth and lips (preventing facial mimicry). Performance of the experimental group was compared against a group of participants that was free to move their faces naturally. Both groups were asked to identify a point at which a morphed face changed from happy to sad and vice versa. Participants who were prevented from using facial mimicry detected the change later in both directions than those who were able to move their face freely, indicating that the disruption of facial mimicry leads to impaired recognition of facial expression.

Although these studies support a relationship between automatic mimicry of facial expression and recognition, and theoretically, empathy has been conceived as a result of both external and internal mirroring (Lipps, 1903), a missing step exists in this theoretical account. If empathy is a result of internal and external mimicry, it would follow that individuals who are more empathetic should produce more facial mimicry than those who are less empathetic. In order to investigate this question, Sonnby-Borgstrom, Jonsoon, and Svensson (2003) compared automatic facial mimicry in individuals who scored high and low on an emotional empathy scale (Questionnaire Measure of Emotional Empathy). Highly empathetic individuals displayed electromyography (EMG) activity consistent with mimicry of the presented facial expressions at both automatic (56 ms) and controlled (2,350 ms) exposure levels. Low-empathetic individuals did not display mimicry at any exposure level. Thus, this study supports the role of simulation in emotional empathy.

If the simulation account of empathy and TOM is accurate and there is a link between motor and mental simulation, one would expect to see regions of the brain that respond to both the experience of a certain thought or emotion and the perception of that thought or emotion in another individual. One may also expect that the premotor MNS would not simply code the physical properties of actions but also be sensitive to the goals and intentions of those actions. Consistent with these claims, preliminary data suggest that mirror neurons in the premotor cortex may be sensitive to the goals and intentions of actions (Gallese et al., 1996; Iacoboni et al., 2005). fMRI studies have identified overlapping regions that respond to both the experience and perception of thoughts and emotions (Mitchell, Banaji, & Macrae, 2005; Morrison, Lloyd, DiPellegrino, & Roberts, 2004; Singer et al., 2004; Wicker et al., 2003). Finally, fMRI and lesion studies have indicated that sensorimotor cortices are not only involved but also necessary for normal performance on TOM and empathy tasks (Avenanti, Bueti, Galati, & Aglioti, 2005; Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000).

In accordance with the simulation theory, single-unit studies with macaques have suggested that a proportion of mirror neurons are broadly congruent (Gallese et al., 1996), meaning they respond to the performance of an action and the observation of an action with a similar goal even if the exact physical properties of the action differs. Similarly, a recent fMRI study supported the claim that the human premotor MNS is sensitive to the intentions and goals of observed actions. In a recently published study, Iacoboni et al. (2005) showed participants videos of four different types of actions. The first video showed a person grasping objects in the absence of any context. The second video depicted scenes containing objects in a context with no actions. The third video showed someone grasping a cup with the intention to drink, and the fourth video showed the same action but in a different context that implied the intention to clean. Results suggested that the posterior part of the inferior frontal gyrus and the adjacent portion of the ventral premotor hand area (both within regions thought to be part of the human premotor MNS) were more active in the two intention conditions as compared with the other two nonintention videos. Additionally, the drinking intention condition resulted in significantly more activation than the cleaning intention videos. These findings suggest that, like that of the monkey, the human premotor MNS is sensitive to the underlying intention that motivates perceived actions.

As would be expected from the facial mimicry research, fMRI conducted while people observe emotional facial expressions in others activates a similar network as imitation of those same expressions. Notable areas in this shared network include inferior frontal cortex, superior temporal cortex, insula, and amygdala, all of which not only showed increased activity in both observe and imitate conditions as compared with a rest condition but also showed more activity in the imitate condition as compared with the observe condition (Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003). This study indicates that there is an overlapping region of premotor cortex that responds both when subjects are asked to interpret emotional facial expressions and when they observe hand actions.

Another brain region that may contain neurons with "mirror-like properties" for internal mental states is the medial prefrontal cortex, Brodmann's Area 9. Though not traditionally thought of as

part of the MNS, this area responds both when subjects are asked to make judgments regarding their own abilities, personality traits, and attitudes (S. C. Johnson et al., 2002; Kelley et al., 2002) and when they are asked to attribute intentions to characters in a comic strip (Brunet, Sarfati, Hardy-Bayle, & Decety, 2000) or infer another person's knowledge about a familiar or unfamiliar object (Goel, Grafman, Sadato, & Hallett, 1995). In a recent study, subjects were asked to evaluate their own emotional responses to a picture and to infer the mental state of the individual in the picture (Ochsner et al., 2004). The medial prefrontal cortex responded during both conditions. Thus, it seems conceivable that the same region of the brain that is involved in representing the mental state of ourselves is also involved in inferring the mental states of others. Similar to the shared representation for the perception and performance of actions mediated by premotor mirror neurons, a system of neurons in the medial prefrontal cortex may serve to create a mirror-like shared representation for the experience and perception of mental states. In this way, the medial prefrontal cortex may be the neural substrate that simulation theorists have been waiting for.

In three fMRI studies (Morrison et al., 2004; Singer et al., 2004; Wicker et al., 2003), empathy for specific emotions activated networks of cerebral cortex similar to the actual experience of that emotion. Both the experience of disgust (while inhaling foul smelling odorants) and the observation of others performing facial expressions of disgust activates the same regions of the insula and the anterior cingulate cortex (Wicker et al., 2003). Additionally, both the experience of a physically painful stimulus and the knowledge that a loved one is experiencing the same painful stimulus activates the anterior insula and rostral anterior cingulate cortex bilaterally (Singer et al., 2004). These areas were also correlated with individual empathy scores, indicating that the more an individual was able to use this shared network of cortices, the better his or her ability to empathize with others. Similarly, another study (Morrison et al., 2004) found that receiving a painful pin prick and watching a stranger receive the same pin-prick activated dorsal anterior cingulate cortex.

Though the emotional aspects of pain are surely a large part of the pain matrix, the full experience of pain also contains sensory input, which has been shown to be important for empathy. In a study conducted by Avenanti et al. (2005), subjects watched actors being pricked by a sharp needle in either the hand or the foot. MEPs were obtained from the same muscles in the observer while TMS was applied over the motor cortex. MEPs in the observer were reduced in the muscle that corresponded to the muscle that was pricked in the actor. The reduction in MEPs was also correlated with the observer's subjective rating of the sensory aspects of the pain attributed to the actor and with sensory but not emotional, empathy measures. This finding suggests that modulation in activity in an observer's motor cortex is related to their perception and ability to empathize with another individual's pain, further supporting simulation theories.

These fMRI results provide evidence for the involvement of sensory cortices in emotion recognition but do not answer the question of whether this activity is necessary for normal recognition of emotion. One technique that begins to answer the question of necessity is lesion studies. These studies have identified paired deficits in the production and recognition of specific emotions. Damage in the amygdala, for example, appears to impair both the expression and recognition of fear (Adolphs et al., 1999; Adolphs, Tranel, Damasio, & Damasio, 2002; Sprengelmeyer et al., 1999). Similarly, damage in the insula and basal ganglia results in a paired impairment in the experience and recognition of disgust (Calder, Keane, Cole, Campbell, & Young, 2000). Finally, both the experience and recognition of anger appears to depend on the dopamine system, with a dopamine antagonist impairing both processes (Lawrence, Calder, McGowan, & Grasby, 2002).

Though these studies provide excellent support for the shared representation of experience and perception of emotions, the sample sizes were extremely low, resulting in limited statistical power and generalizability of the findings. This potential limitation was not a factor for Adolphs et al. (2000), however, who addressed the question of the necessity of sensorimotor cortices for recognition of emotions in visually presented facial expressions in 108 focal brain lesion patients and 30 healthy control participants. Subjects participated in three visual emotion recognition tasks. In the first task, participants were asked to rate the intensity of basic emotional facial expressions. In the second task, participants were asked to match a facial expression with the name of the emotion it is meant to convey. The final task required participants to sort facial expressions into emotional categories. Though each task identified a slightly different group of regions, damage to primary and secondary sensorimotor cortices impaired performance in all three tasks, supporting the critical role of sensory and motor cortices in the recognition of visually presented facial expressions.

Role of Simulation in the TOM and Empathy Deficits in ASD

The aforementioned behavioral and neuroimaging studies offer strong evidence in support of the role of motor and sensory systems in social skills (such as TOM and empathy) in typically developing individuals. Additionally, as noted earlier in this review, an impaired motor system may play a critical role in the neural pathology of the imitative deficit in ASD. If the same system underlying motor simulation is a critical component in TOM and empathy (as suggested by Gallese, 2001) and if this system is in fact dysfunctional in individuals with ASD, then one would expect that these individuals would have both TOM and empathy deficits. Consistent with this argument, many researchers believe TOM and empathy deficits are central to the clinical manifestation of autism (see Baron-Cohen, 1995; Gillberg, 1992).

Anecdotally, Gillberg (1992) reported that children with autism are sensitive to affective change. In other words, individuals with autism can feel whether an interaction is full of strong emotion even if they do not show appropriate behavioral reactions to emotional events. It is possible, then, that these children can also feel emotions such as happiness, anger, and fear but that they have difficulty making sense of these feelings in others, leading to a behavioral impairment of empathy. This anecdotal report was subsequently supported with controlled studies showing individuals with ASD to have appropriate autonomic arousal (Blair, 1999; Shenk & Ramachandran, 2003) but impaired behavioral responses (Bacon, Fein, Morris, Waterhouse, & Allen, 1998; Corona, Dissanayake, Arbelle, Wellington, & Sigman, 1998; Sigman, Kasari, Kwon, & Yirmiya, 1992) to the observation of individuals in distress.

In addition to these behavioral studies, direct evidence for a facial mimicry deficit in individuals with ASD was seen in a recent study conducted by McIntosh, Reichmann-Decker, Winkielman, and Wilbarger (2006). In this study, adults with ASD, as well as age-, gender-, and verbal intelligence-matched typically developing individuals viewed pictures of happy and angry facial expressions. In one condition, automatic mimicry was measured by EMG when participants were instructed to "watch the pictures as they appear on the screen." In a second condition, voluntary mimicry was measured by instructing the participants to "make an expression just like this one." EMG electrodes placed over the cheek and brow recorded muscle activity. Despite displaying a normal pattern of voluntary mimicry, results from this study suggested that, unlike the participants in the matched control group, participants with ASD did not show automatic mimicry.

Similarly, TOM studies have found severe deficits in individuals with autism. In a series of studies conducted by Baron-Cohen, Leslie, and Frith (1985, 1986), typically developing children, children with Down's syndrome, and children with autism were given the "Sally-Anne" test. Whereas the vast majority of both 4-year-old typically developing children and mental age-matched children with Down's syndrome passed the test, the majority of children, adolescents, and adults with autism failed, even when the task was controlled for verbal ability. This study also included a control task that asked subjects to order a picture sequence on the basis of physical causality, in which children with autism performed at a normal level, indicating that their impairment in understanding beliefs as psychological causes of behavior was not a result of a general inability to understand causality.

A similar finding was also reported in another study (Perner, Frith, Leslie, & Leekam, 1989) in which subjects were shown a Smarties box and asked to guess what was inside. When subjects were shown that in fact pencils (rather than the expected Smarties) were inside, they were then asked to predict what the next child who comes in would guess is in the box. Typically developing children answered "Smarties," their original false belief. In answering this way, typically developing individuals clearly were able to inhibit their current belief in order to represent the counterfactual knowledge that the next child would have. The majority of children with autism, however, answered "pencils," reflecting an inability to inhibit their own current beliefs and infer false beliefs in others. These findings have been interpreted as support for a selective deficit in understanding the psychological mechanisms that underlie human behavior.

It is important to note, however, that a debate exists over whether the deficits in these tasks are a result of impaired TOM or simply a deficit in executive functioning (see Perner & Lang, 2000, for a review of this debate). Though there is much evidence supporting the correlation between TOM abilities and executive function abilities in both typically developing individuals (see Russell, Mauthner, Sharpe, & Tidswell, 1991) and children with autism (see Ozonoff, Pennington, & Rogers, 1991; Russell, 1997), it appears that TOM abilities are a prerequisite for executive functioning rather then vice versa (Perner & Lang, 2000). Additionally, studies conducted by Charman and Baron-Cohen (1995) showed no deficit in individuals with autism on tasks that have a similar working memory load as false-belief tasks (e.g., false photograph and false model tasks), indicating that though a working memory or executive functioning deficit may exist in autism, it cannot account for the poor performance on these false-belief tasks.

One prominent theory of the social deficits in ASD was put forth by Baron-Cohen et al. (2000), who implicated the amygdala as the core brain region responsible for this impairment. This theory was based on several lines of evidence. The first was primate lesion studies, which found that lesions of the amygdala resulted in monkeys that failed to initiate social interactions and failed to respond appropriately to social gestures (Kling & Brothers, 1992). Other evidence comes from post-mortem studies finding increased cell density in the amygdala of individuals with autism (Bauman & Kemper, 1994). Functional imaging also showed significant reductions in activity of the amygdala during a mentalizing task (Baron-Cohen et al., 1999). Additionally, there is physiological evidence supporting a dysfunction in limbic connections resulting in an abnormal pattern of skin conductance response to visual stimuli in individuals with ASD (Hirstein, Iversen, & Ramachandran, 2001). Though Baron-Cohen et al. stressed the role of the amygdala in autism, they were quick to highlight that other neural regions also show abnormalities.

Another brain region thought to play a role in the social deficits of autism is the medial prefrontal cortex (paracingulate cortex). This region responds during TOM tasks in typically developing individuals when subjects are asked to reflect on both their own and another individual's mental state. (Brunet et al., 2000; Castelli, Happé, Frith, & Frith, 2000; Fletcher et al., 1995; Gallagher et al., 2000; Vogeley et al., 2001) The same region also has reduced activity during similar tasks in individuals with ASD (Baron-Cohen et al., 1999; Castelli, Frith, Happé, & Frith, 2002; Happé et al., 1996).

The third region that has been implicated in the social deficits in ASD is the superior temporal sulcus. Nonhuman primate single unit studies (Oram & Perrett, 1994) as well as human neuroimaging studies (Bonda et al., 1996; Grossman et al., 2000) have shown activity in this region in response to biological actions (see Allison, Puce, & McCarthy, 2000, for a review), as well as detection of eye gaze in monkeys (Perrett et al., 1985) and humans (Hoffman & Haxby, 2000; Puce et al., 1998) and inferred intentional action of inanimate shapes on the basis of movement patterns in humans (Castelli et al., 2000). This brain region has also shown abnormal activity patterns in individuals with ASD during face recognition tasks (Critchley et al., 2000; Schultz et al., 2000), a mentalizing task (Castelli et al., 2002), and an eye gaze-processing task (Pelphrey et al., 2005). Studies have also found reduced grey matter volume in anatomical MRI scans (McAlonan et al., 2004).

In conclusion, though more research needs to be done, there is much evidence that multiple brain regions including the amygdala, medial prefrontal cortex, and superior temporal sulcus are involved both in the development of normal social cognition and in the impairments seen in ASD. Additionally, each of these three regions appears to possess mirror properties. Thus the simulation theory of autism not only is supported by these findings but also subsumes these findings under a more unified theory.

Language

Evidence for Role of Simulation in Language Comprehension and Production

Thus far this review has discussed evidence for the role of simulation in three social abilities that are central in the behavioral

pathology of autism: understanding of others' behaviors, thoughts, and feelings. We now move to address a fourth ability that can also be explained in terms of a deficit in simulation, language comprehension and production.

A leading theory of language comprehension may provide a clue as to why individuals with autism have both social and communicative deficits. This theory, termed the *motor theory of speech perception* (Liberman & Mattingly, 1985) has two main claims. The first claim is that the objects of speech perception are not the acoustic signals (as proposed by Ohala, 1996) but rather the phonetic gestures. The phonetic gestures of the speaker are represented in the brain of the observer as motor commands that signal movements of the mouth, lips, and tongue in specific configurations. The second claim of this theory is that speech perception and speech production are intimately linked.

Supporting evidence for this theory comes from behavioral, neuromagnetic, and neuroimaging studies. One paradigm that has been used to support the linkage between speech perception and speech production is similar to the response interference paradigms used to support the role of simulation in action perception. In a series of studies performed by Kerzel and Bekkering (2000), participants were asked to speak the syllables /ba/ and /da/ while watching silent videos of others articulating the same or different syllables. For both the /ba/ and /da/ conditions, a significant interference effect was noted with a slower reaction time when the response-irrelevant visual stimulus was inconsistent with the target response.

Additional evidence comes from neuromagnetic studies. Fadiga, Craighero, Buccino, and Rizzolatti (2002) performed a TMS study in which participants listened to auditory speech and nonspeech sounds while a magnetic pulse was applied to their left motor cortex. The MEP in the tongue was recorded using electrodes placed on the surface of the tongue. When tongue movements were required to produce the heard speech sounds, there was an increase in MEPs as compared with nonspeech sounds or speech sounds that did not require the tongue.

In a similar study, Watkins, Strafella, and Paus (2003) investigated whether hearing or watching speech sounds being produced would increase MEPs in the lips. Subjects listened to speech and nonverbal sounds and watched speech-related lip movements as well as eye and brow movements. In both the auditory and visual speech conditions, TMS over primary motor cortex resulted in an increase in MEPs in the lip muscles, further supporting the involvement of the motor cortex in the comprehension of speech.

fMRI studies have also shown specific activation of speech production areas during the listening of speech sounds. Wilson, Saygin, Sereno, and Iacoboni (2004) scanned 10 subjects while they listened to meaningless monosyllable sounds then produced the same sounds. Production of the sounds activated areas of prefrontal cortex including Brodmann's areas 4a, 6, and 4p. Similarly, listening to speech sounds activated areas of ventral premotor cortex, which largely overlapped with the posterior speech production areas.

Although not directly related to the motor theory of speech perception, further evidence for the involvement of motor actions on language is found in the studies of Rauscher, Krauss, and Chen (1996) and Glenberg and Kaschak (2002). Rauscher et al. found that subjects gesture five times as frequently when describing spatial aspects of a visual scene as compared with nonspatial aspects (0.5 vs. 0.1 gestures per word). Additionally, when subjects were prevented from gesturing, they spoke more slowly. This finding applied only when the content of speech was spatial in nature (116 vs. 100 words per minute). With other kinds of content, however, the subjects' rate of speech was somewhat increased when they could not gesture. This study provides evidence for a shared representation between verbal and motor aspects of language.

A series of studies conducted by Glenberg and Kaschak (2002) found that gestures may share not only a representation with language production but also language comprehension. In these studies, participants were asked to judge whether sentences were sensible by making a response that required moving either toward or away from their bodies. The experimental sentences were specifically created to describe actions that required movements either toward or away from the body (e.g., *Open the drawer*). When the response required a movement that was inconsistent with the sentence, the latency for response was increased as compared with when the response action matched the action described in the sentence. This finding was true for imperative (e.g., *Open the drawer*), concrete transfer (e.g., *Courtney handed you the book*), and abstract transfer (e.g., *You told Liz the story*) sentences. Findings from this and the previous behavioral studies indicate that speech observation interferes with speech production, physical gesturing influences speech production, and language comprehension has a Stroop-like interference effect on action production, all of which support the motor theory of speech perception.

The second claim of the motor theory of speech perception is that speech perception and speech production share an underlying mechanism. Along these lines, recent evidence suggests that the superior temporal sulcus as well as the inferior frontal gyrus respond to both the sight and sound of human speech (Calvert & Campbell, 2003). Given these findings, researchers are beginning to draw connections from the action-related MNS to the utilization of this system for communication. If the purpose of simulating the action with mirror neurons is to understand the observed action, one would theorize that activating the speech production areas while listening to language would lead to better understanding of the verbalization. Electrophysiological and imaging data support this theory.

Hauk and Pulvermuller (2004) recorded EEG while subjects silently read words that related to hand, leg, and head actions. A visual-evoked potential was recorded over the frontal-central leads at approximately 210–230 ms after stimulus onset. Source localization discovered that not only did the reading of these sentences activate regions of sensorimotor cortex, but the activation was somatotopically distinct based on the effector that would be used to perform the action. Face-action words resulted in activations in left frontal regions, whereas leg-action words showed activations in central leads, and arm-action words activated largely right central and right frontal regions.

A follow-up study by the same group (Hauk, Johnsrude, & Pulvermuller, 2004) used fMRI to confirm that simple reading of action-related words activates somatotopically distinct areas depending on the effector used for that action. Results confirmed that reading face-action words specifically activated inferior frontal gyrus, arm-action words activated areas of middle frontal and precentral gyrus, and leg-action words activated pre- and postcentral gyri, superior frontal gyrus, and the dorsomedial frontal region.

For the arm- and leg-movement words, there was a significant overlap with performed leg and arm actions. For face-action words, the activation pattern was just anterior to the region activated by actual tongue actions. This study further confirmed the important relationship between action-related language and the areas of cortex responsible for the performance of the same actions.

A second fMRI study conducted by Tettamanti et al. (2005) confirmed that across all body parts, processing of action sentences selectively activated the pars opercularis of the left inferior frontal gyrus (Broca's area). Mouth sentences activated the pars opercularis in regions rostral, dorsal, and ventral from the focus activated by all body parts. Additionally, the pars triangularis of the inferior frontal cortex was activated during listening of mouth sentences. Hand sentences resulted in a distinctly different pattern of activation, including left precentral gyrus, the left posterior intraparietal sulcus, the left posterior inferior temporal gyrus, the left insula, and the right middle temporal gyrus. Finally, leg sentences selectively activated the left dorsal premotor cortex (within the superior frontal sulcus) and the left inferior parietal lobule.

Finally, a TMS study conducted by Buccino et al. (2005) found a decrease in MEPs recorded from the hand muscles when participants listened to hand-action sentences and MEPs recorded from leg muscles when listening to leg-action sentences. Though modulations in MEPs were an expected result, the direction of the modulation was not as predicted. The authors suggested multiple explanations for the unexpected decrease in MEPs in the muscle consistent with the heard sentence including the modality of presentation and interference. Independent of the direction of the effect, this study provides evidence for specific modulation of the motor areas involved in action production when listening to sentences related to those actions.

Taken together, these findings support a shared mechanism for speech production and speech perception in both auditory and visual domains. Additionally, these findings support a role for simulation in speech comprehension, as the motor effectors involved in actions seem to be modulated when one listens to action-related sentences. The MNS was specifically implicated in the evolution of language in a review article by Rizzolatti and Arbib (1998).

Rizzolatti and Arbib (1998) suggested that the MNS, which mediated action comprehension in monkeys and higher primates, went through six stages of evolution that led to its eventual role in language comprehension in the modern human.

1. The MNS may originally have been for the purpose of recognition of grasping actions through a simulation mechanism in early primates.

2. This primal system evolved in the chimpanzee to support simple imitation for object-directed grasping.

3. In the early homonid, this simple system may have become elaborated to include complex imitation that allowed for learning of novel actions that could be approximated by variants of actions that were already part of the observer's repertoire.

4. The early hominid then developed a system of protosigns, which was a manual-based communication system. This leap from imitation for the sake of instrumental goals to imitation for the sake of communication was likely when the specificity of MNS for object-directed actions was lost.

5. Once protosign had evolved, this provided the scaffold for which protospeech could develop. Once an individual learned a conventional gesture, that gesture could be paired with a vocalization (protospeech).

6. The final stage, language, it is argued, was mediated more by cultural rather than biological evolution in *Homo sapiens*.

If, as suggested by Rizzolatti and Arbib's model and the previously cited studies, the MNS and simulation processes are necessary for the proper evolution, development, and comprehension of language, then it is not at all surprising that individuals with autism, in addition to having social deficits, have language impairments.

Language Deficits in ASD

Beyond the basic function of speech, language also serves the purpose of communication of thoughts, beliefs, and desires between individuals. Though about half of individuals with autism will acquire some level of linguistic knowledge, even these individuals will likely have a specific deficit in the use of language for social communication, pragmatic abilities. When compared with both typically developing and other developmental disorder populations, multiple behavioral studies have revealed a relative sparing of phonological, lexical–semantic, and grammatical aspects of language in verbal individuals with ASD (see Tager-Flusberg, 2000, for a review). The spared abilities may explain why many individuals with autism develop rudimentary aspects of communication, including turn-taking skills, requesting behavior, and regulatory speech.

Some clinical features of the language impairment in autism include echolalia, neologisms, and reversal of pronouns. In lower functioning individuals, often the only language production that is present is echolalic, or the echoing of the words phrases or sentences spoken by others (McEvoy, Loveland, & Landry, 1988). *Neologisms* are idiosyncratic lexical terms that have special or unique meanings to the individual but no meaning to the general public. This tendency is most common in higher functioning individuals with ASD (Volden & Lord, 1991). Finally, verbal individuals with autism will commonly reverse pronouns and refer to themselves as *you* and others as *I*, indicating a difficulty in the notion of self and other (Lee, Hobson, & Chiat, 1994). This tendency is more common in individuals with autism than in any other clinical population (Lee et al., 1994). All of these features characterize the basic impairment in pragmatic use of language in individuals with ASD.

Some have suggested that this selective impairment may be tied to these same individuals' TOM deficits (Locke, 1994; Sperber & Wilson, 1987; Tager-Flusberg, 1993). In the words of Sperber and Wilson (1987), "Communication exploits the well-known ability of humans to attribute intentions to each other" (p. 699). However, explaining the language deficits in autism in terms of behavioral

TOM deficits without suggesting a candidate mechanism for either is explaining one enigma with another and adds little to the understanding of either of these phenomena. Additionally, the direction of this relationship is unclear. Some suggest that TOM is a necessary precursor to developing social communication (Winner, Brownell, Happé, Blum, & Pincus, 1998;Winner & Leekam, 1991; Happé, 1993); others have proposed that experience with social communication leads to proficient TOM skills (Peterson & Siegal, 2000).

Other theories for the language impairment in autism include the weak central coherence theory (Frith, 1989) and an executive dysfunction theory proposed by Liss et al. (2001). The central coherence theory states that pragmatic language requires the ability to not simply attend to the specific semantics of language but rather focus on the global context of the intercourse to comprehend the meaning, whereas the executive dysfunction theory is supported by correlational analyses between executive functioning and language skills in autism (Liss et al., 2001).

Though there is much theory on the underlying mechanisms mediating the language impairment in autism, there is little conclusive evidence for a causal relationship between TOM, central coherence, or executive functioning and the language impairment in ASD. However, the theory presented here, based on the motor theory of speech perception and theories suggesting the involvement of simulation mechanisms such as the MNS in language, provides a functional mechanism that may explain the communication impairments in ASD from a simulation account. One study, conducted in our laboratory, provides preliminary evidence for the role of impairment in the MNS in the language deficits of ASD (Ramachandran & Oberman, 2006). This study used a linguistic multisensory-integration task, the bouba–kiki task, in which participants are asked to name nonsense shapes. For example, if given an amoeboid shape and a jagged shape and asked to name one *bouba* and another *kiki*, 99% of the general population will name the amoeboid shape *bouba* and the jagged shape *kiki*. This is presumably because of mirror neuron-like multisensory systems that integrate the visual shape with sounds (sound–form symbolism). Surprisingly, only 20% of individuals with autism showed this effect. We suggest that the MNS may be involved in multisensory integration in the linguistic domain including the bouba–kiki effect and metaphors, which would explain their deficits in both tasks.

Conclusions

This article has argued that perceptual recognition, motor mimicry, TOM, empathy, and pragmatic language may all be mediated by underlying simulation mechanisms in typically developing individuals. Despite this underlying similarity, historically these abilities have been studied in isolation, and consequently, multiple simulation theories have been developed to explain one or two of these phenomena. However, given that these abilities all seem to be impaired in a single clinical syndrome—autism—it is not only parsimonious but also clinically necessary to develop a unified theory.

Returning to Barsalou's (1999) situated conceptualization model introduced at the beginning of this review, he proposed that simulators exist in multiple brain regions related to the region that first encoded that information. Thus, simulations of actions and body states may occur in areas of the cerebellum, superior temporal cortex, and the MNSs of inferior parietal lobule and premotor cortex (Miall, 2003), whereas the simulator networks for introspective states such as thoughts, beliefs, and emotions are likely to be found in the amygdala, medial areas of prefrontal cortex, insular cortex, and the anterior cingulate cortex.

Not coincidently, the brain regions that have most often been implicated in ASD—cerebellum, amygdala, medial prefrontal cortex, and premotor cortex—all appear (on the basis of the literature cited in this review) to house simulator systems. Thus, we propose that a developmental impairment of functional simulators, especially the MNS, may be the unifying mechanism that underlies the deficits in imitation, TOM, empathy, and pragmatic language. A recent review by Gallese (2006) suggested that a similar mechanism, what he calls *intentional attunement*, is at the root of ASD.

As the data currently stand, this theory has yet to account for behaviors related to restricted, repetitive, and stereotyped patterns of behaviors, interests, and activities. However, it is one of the most comprehensive of any of the theories to date and, with more research, may too account for this aspect of the disorder. The mirror neuron–simulation theory of autism, for which the first experimental support came from preliminary studies conducted in our lab (Altschuler et al., 2000; Oberman et al., 2005), accounts for the wide range of behavioral and neurological deficits that are unique to autism and has the added advantage of fitting well within the literature regarding these abilities in the normal population.

One question that still remains is how simulation systems such as the MNS develop. There are a variety of ways this unique mechanism could be set up, but whatever the mechanism, the research reviewed here suggests that a flaw in its initial setup might be the fundamental deficit in autism. This flaw could affect either the gray matter regions containing the simulator neurons themselves or the white matter tracts that link the regions together as a functional system or a combination of both.

As this review highlights, there is a large body of literature supporting the role of simulation in the areas of perception, motor mimicry, TOM, empathy, and language. Additionally, there is much evidence for deficits in these abilities in individuals with ASD. Two remaining questions that require further investigation include the development of the MNS and how well the behavioral deficits in ASD can be explained by impairments in simulation, especially in the domain of language in which this theory has yet to be applied. The latter is currently being investigated through studies using the same paradigms that were used to establish the importance of simulation in the general population to investigate whether their behavioral deficits are correlated with their performance on simulation tasks.

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