Essay

A mirror up to nature

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Mirror neurons were first documented in the macaque monkey a little over ten years ago. Their discovery has led to the formulation of several theories about their function in humans, including suggestions that mirror neurons are involved in understanding the meaning and intentions of observed actions, learning by imitation, feeling empathy, formation of a 'theory of mind', and even the development of language. Hypotheses have also been made about the consequences of mirror neuron dysfunction; foremost among these is the notion that such a dysfunction during development leads to many of the social and cognitive symptoms associated with the autism spectrum disorders (ASDs). Yet, despite a decade of prolific research on these appealing theories, there is little evidence to support them. In this essay, we review the current state of 'mirror system' research, point to several weaknesses in the field, and offer suggestions for how better to study these remarkably interesting neurons in both neurotypical and autistic individuals.

Mirror neuron research in monkeys

In a seminal experiment, Gallese *et al.* [1] found that approximately 17% of neurons recorded in ventral premotor area F5 of the macaque monkey responded both when the monkey executed a particular movement — for example, grasping, placing or manipulating — and when the monkey observed someone else performing that same movement. The experimenters noted that a third of these 'mirror neurons' (only 6% of all F5 neurons) responded selectively to one particular movement, whether observed or executed, and not to others, while the remainder responded to varying degrees to several 'related' movements. These observations suggest that mirror neurons form a distributed representation of observed and executed movements. According to this idea, different subpopulations of mirror neurons respond selectively to different movements (analogous to the orientation selectivity of neurons in primary visual cortex) and each subpopulation responds similarly to its preferred movement either when it is observed or when it is executed. In this manner, mirror neurons are distinguished from the many other visual, motor and visuomotor neurons that also exist in this area and that are involved in a multitude of visuomotor processes needed for the coordination of movement.

To date, three other monkey electrophysiology studies have been published on this topic in peer-reviewed journals. The first two studies [2,3] reproduced the original finding in area F5, while the third [4] reported neurons with the same functional characteristics in anterior intraparietal area PF/IPL. One of the studies [2] reported that a small number of mirror neurons in area F5 responded selectively not only to particular observed and executed actions, but also to their associated sounds, such as ripping or crushing. The other two studies [3,4] found that a small number of mirror neurons in areas F5 and IPL responded selectively to a particular movement goal or intention rather than to the immediate kinematics and dynamics of a movement. For instance, a 'grasping to feed' movement was shown to activate a different set of mirror neurons than that activated by a 'grasping to place' movement, regardless of whether the movement was observed or executed [4].

These results are remarkable and have, in effect, opened a new field of research into the neural substrate of social cognition. Many theories have suggested a possible role for mirror neurons in mechanisms of action understanding, imitation, empathy, theory of mind, and language [5–7]. All these theories propose that mirror neurons act as a mapping mechanism between the observation of an action and its execution so that, when you observe someone performing a movement, particular mirror neurons embedded in your motor system are activated, enabling you to simulate yourself performing that movement using your own motor system. This simulation then allows you to access your own associated intentions, goals, emotions and social values (perhaps through activity of other brain areas including the limbic system) and assign them to the person you are observing. Mirror neurons can, hence, be thought of as a gateway mediating the formation of an internal representation of the observed person's state and intention. Note that, for this mechanism to work, it is critical that the observed movement (or movement goal) be mapped onto the particular neural circuits used to execute that exact same movement (or goal), otherwise you will assign improper intentions to the person you are observing. Movement selectivity is, therefore, a crucial feature of mirror neuron responses, because successful mapping must be accomplished in a movement-selective manner. In a similar fashion, our ability to understand language has been proposed to rely on a hypothetical group of specialized mirror neurons, which have evolved to map heard vocalizations and words onto the motor structures used to verbalize them, thus enabling a similar process of assigning associated meaning. The great appeal of these theories is that they propose a simple and elegant neural mechanism for associating external visual and auditory stimuli with their appropriate semantic, social and emotional meanings.

Though we share in the excitement about these theories, we note that the neurophysiology experiments described above do not provide much support for them. Imitation, empathy, theory of mind, and the ability to use language were not assessed in these studies, and whether these issues can be studied at all in non-human primates continues to be controversial. Nor was the

ability to understand the actions of others assessed, although it seems reasonable that one might be able to investigate whether this ability depends on mirror neuron activity in monkeys (for example, by pharmacologically inactivating 'mirror' areas, and demonstrating that the monkey has lost the ability to understand an observed movement). Another limitation of the neurophysiology studies described above is that they were qualitative rather than quantitative in nature. In all four studies, the experimenters distinguished responses to a limited number of movements performed by the monkey (executed movements) and experimenter (observed movements) without assessing their precise dynamics and kinematics. The conclusion that intraparietal area, IPL, contains neurons selective for movement goals was based on a qualitative analysis of the responses of only 16 neurons that reliably distinguished between only two movement goals: grasping to eat versus grasping to place [4]. Similarly, the conclusion that area F5 contains multimodal mirror neurons, which are selective for a particular action (for example, crushing a peanut) whether it is heard, observed or executed was initially based on a qualitative analysis of the responses of only 22 neurons. A follow up paper [8], however, presented an interesting quantitative analysis of the ability of these 22 neurons to discriminate among pairs of actions, which serves as an example of how the selectivity of these neurons can be quantitatively characterized.

Do monkey mirror neurons really form a 'dictionary' of movements or movement goals that underlies the monkey's ability to understand the intentions of others? We are of the view that there is still need for a systematic and quantitative characterization of the response selectivity of these neurons as well as a need to establish a causal relationship between their activity and the proposed motor, cognitive, and social abilities, before such claims can definitively be accepted. An experimental setup that seems ideal for performing such quantitative assessment has recently been developed [9], which enables simultaneous measurements of neural activity from areas IPL and F5 as well as kinematics of hand movements performed by pairs of monkeys who are freely interacting with one another.

Mirror neuron research in humans

The relatively clear description of a monkey 'mirror system' composed of two cortical areas that contain mirror neurons has, unfortunately, morphed into a rather vague concept in the search for an equivalent human 'mirror system'. During the past ten years, dozens of studies have used different techniques, including functional magnetic resonance imaging (fMRI), positron emission tomography (PET), electroencephalography (EEG), magnetoencephalography (MEG) and transcranial magnetic stimulation (TMS), in an attempt to identify a human 'mirror system' homologue. We shall focus here mainly on the relevant fMRI studies, but note that studies using other techniques have adopted similar experimental protocols with essentially the same underlying logic and assumptions. In general, these studies have used three types of protocols to elicit mirror neuron responses in humans: passive movement observation, separate observation, and execution of movement, and imitation of movement. In the first protocol, subjects passively view images or video clips of movements, such as a smiling face or a hand grasping an object, and their fMRI responses are compared against a rest condition, following the logic that mirror neurons are active during movement observation and not during rest. In the second protocol, a movement execution condition is added to first isolate cortical areas that respond during execution; fMRI responses during movement observation are then analyzed only within these areas because mirror neurons are expected to respond both during observation and execution of a movement. In the third protocol, subjects passively observe movements, execute the same movements in the dark, or imitate the observed movements (that is, simultaneously observe and execute the movement). The fMRI responses during the imitation condition are compared with responses during observation and during execution with the logic that mirror neurons should be more active during simultaneous observation and execution than during execution or observation alone [5].

There are two concerns with these protocols. The first is that they are unable to measure exclusive mirror neuron activity. For instance, the typical results of passive movement observation and imitation experiments reveal many cortical areas that exhibit larger fMRI responses during observation and imitation, including areas that are not believed to contain mirror neurons; primary visual cortex for example. This is clear evidence that there are many other neurons (in addition to mirror neurons) in diverse cortical areas that increase their responses during these two tasks. These neurons are likely involved in processes of visual recognition, visual motion perception, working memory, movement planning, and movement execution (in the case of imitation), which are all integral components of these tasks. Limiting the analysis to cortical areas that also respond during movement execution (by masking out areas that do not) does not solve this problem. Although this protocol identifies cortical areas that respond during both movement execution and observation, it does not isolate mirror neuron responses from the activity of other (possibly intermingled) visual, motor, and visuomotor neural populations that could underlie the measured fMRI responses.

How then can one know if the fMRI response exhibited by a particular brain area is generated by the activity of mirror neurons or by the activity of any of these other neural populations? Most studies have simply disregarded activity in all cortical areas except for ventral premotor (vPM) and anterior intraparietal sulcus (aIPS), because these two areas are assumed to be homologous to monkey areas F5 and PF/IPL and are, therefore,

expected to contain mirror neurons. Using such circular reasoning, these studies have sidestepped the most important issue, which is to examine whether human mirror neurons actually exist and to characterize their physiology. This circular interpretation has been taken to such an extreme that some recent studies now interpret any fMRI response in areas vPM and aIPS — for example, fMRI responses while observing moving shapes [10] — as being due to mirror neuron activity. Such interpretations grossly ignore the fact that mirror neurons in the monkey account for only a small minority of the neurons in these areas and that the reported fMRI responses could easily be generated by activity of the many neighboring visual, motor, and visuomotor neurons that are not mirror neurons. The widespread cortical responses generated by the movement observation and imitation tasks have also created a vagueness regarding the exact location of the implicated vPM and aIPS areas, whose reported locations vary dramatically among different studies. For instance, the exact location of the implicated vPM area differs by up to 3 cm from one fMRI study to another (see Table 1 in [11]).

The second, and perhaps more important, concern with these studies lies in their lack of ability to assess movement selectivity. As mentioned above, movement selectivity is a defining physiological signature of mirror neurons in the monkey, and is of central importance for theories proposing that mirror neurons play a role in mapping perception to action. If mirror neurons indeed form a 'dictionary' of movements or movement goals, subpopulations of mirror neurons must respond selectively to particular movements or goals. Several 'mirror system' studies have attempted to assess selectivity by comparing fMRI responses to observed movements performed by different effectors (the foot, hand, and mouth). These studies have suggested that mirror neurons are distributed according to the classical somatotopic 'homunculus' organization in premotor and anterior parietal cortical areas [12,13]. Similarly, several TMS studies have reported that primary motor cortex excitability is enhanced in an effector specific manner [14]. Note, however, that these studies suffer from the same drawbacks described above; specifically, that it is unclear whether these somatotopically organized fMRI responses are due to mirror neuron activity or to that of other neural populations. Regardless, testing selectivity at the level of effectors is at a much grosser level of resolution than that of movements or movement goals, which is the level of resolution needed to support mirror system theories.

Assessing neural selectivity using non-invasive techniques in the human brain is difficult in situations where neurons with different preferences may be intermingled within a small volume of tissue (as seems to be the case in vPM and aIPS). Specifically, any particular fMRI voxel within these areas (usually 3 × 3 × 3 mm in size) will contain subpopulations of neurons selective for many different movements and will, therefore, respond when executing or observing many different movements. Showing an overall stronger fMRI response to one condition versus another (for example, to movements with 'goals' *versus* movements without 'goals') does not mean that the underlying neurons are selective (for example, for a particular goal). Such an overall response difference could easily be generated by modulation of the whole neural population within each voxel by processes of attention, arousal, emotional valence and so on, regardless of whether these neurons are selective for movements/goals or not. By contrast, a selective increase in response of one subpopulation of neurons might be complemented by a decrease in the responses of other subpopulations, resulting in no change in the overall level of activity. This distinction between an overall increase in activity and a selective response by a subpopulation of neurons is well-understood in sensory systems [15]. Using imitation and movement observation protocols to look for overall increases in fMRI responses cannot, therefore, be used to test theories regarding the function of human mirror neurons.

The critical challenge in studying the human mirror system is to devise new experimental protocols that can assess response selectivity to movements in the human brain. A common method for assessing neural selectivity using fMRI takes advantage of the fact that sensory neurons adapt/habituate when their preferred stimulus is presented repeatedly [16]. Cortical areas containing neurons selective for a particular stimulus attribute are, therefore, expected to exhibit reduced fMRI responses when the preferred stimulus is repeated in comparison to when it is not repeated. This method can be applied to localize cortical areas that exhibit adaptation when the same movement is repeatedly observed, repeatedly executed, observed and then executed, or executed and then observed (cross-modal adaptation). In this way, one can assess the actual defining feature of mirror neurons: movement selectivity for observed and executed movements.

Three recent fMRI studies [11,17,18] have used such 'adaptation protocols' to assess movement selectivity in the human brain. Two of these studies [17,18] focused on movement observation and showed that several parietal areas exhibited reduced responses to movement repetition. One study [17] attributed this adaptation to the goal of the observed movements (for example, grasp a cookie versus grasp a floppy disk), while the other [18] attributed it to the identity of the movement (the type of grasp being observed). In the third study [11], some of us attempted to isolate mirror neuron responses using a combined visual and motor adaptation protocol. We succeeded in finding five movement selective cortical areas that exhibited adaptation both when the same movement was observed repeatedly and when it was executed repeatedly. Unlike the dispersed imitation responses, our adaptation responses were limited to the anterior inferior frontal sulcus, ventral premotor,

anterior intraparietal, superior intraparietal, and posterior intraparietal cortices. We suggest that all five areas should be considered as candidates for the human mirror system because they contained neurons selective for both observed and executed movements.

Despite our claim that the adaptation protocol is a superior way of identifying candidate mirror system areas, however, we still were unable to demonstrate the existence of mirror neurons in the human brain as we did not find any cortical areas exhibiting cross-modal adaptation [11]. Such adaptation, in trials where the same movement was observed and then executed or executed and then observed, would have provided strong evidence that visual and motor adaptation were taking place in a single subpopulation of visuomotor mirror neurons. It is possible that the overlapping visual and motor adaptation effects that we did find were generated by two separate (possibly intermingled) subpopulations of visual and motor neurons that adapted independently during repeated observation and execution of the movements. Nonetheless, these results show for the first time that five specific areas of human cortex contain movement selective neurons both for observation and execution. If mirror neurons exist at all in the human brain, it is likely that they lie within these areas. We hope that future human mirror system studies use similar and novel protocols for assessing movement selective responses rather than relying on the circular reasoning commonly used to interpret imitation and passive movement observation experiment results.

Mirror neuron research in autism

A recent theory [7] that has received considerable attention both in expert and popular science literatures posits that a dysfunction in the 'human mirror system' serves as the physiological basis of some of the core behavioral impairments which characterize ASDs. ASD refers to a set of complex, polygenetic neurodevelopmental disorders of unknown etiology and is diagnosed according to three behavioral characteristics: social and communication deficits, repetitive behavior and restricted interests. Among the behavioral impairments are deficits in joint attention, imitation, social interaction and communication (the ability to use language and gestures), empathy and theory of mind (the ability to understand the intentions of others). As mentioned above, several researchers have suggested that mirror neurons embody a neural mechanism enabling these precise cognitive capabilities in neurotypical individuals and this is why a connection has been made between ASD and mirror system dysfunction. Yet, as is the case with the theories regarding mirror neuron function in neurotypical individuals, there is surprisingly little evidence to support the claim that a dysfunction in mirror neurons is the neural mechanism underlying ASD.

A small number of studies have used the passive movement observation and the imitation protocols described previously to compare 'mirror system' responses in ASD and neurotypical individuals. While some studies report differences in 'mirror system' responses to these two tasks (response differences in the general vicinity of vPM and/or aIPS) [19–22], other studies do not [23,24]. Assuming that there are cortical response differences to these tasks, the more pressing question in connection with the 'mirror system' hypothesis is whether these differences (as measured by fMRI, EEG, and TMS) are due to differences in mirror neuron activity or to that of other neural populations.

Numerous studies have reported multiple cortical and sub-cortical response differences between ASD and neurotypical populations in connection with just about any task tested (for example, a visual search task [25]). Even while participants are performing imitation tasks, several brain areas have been reported to respond differently between ASD and neurotypical individuals, including areas not expected to contain mirror neurons, such as the amygdala [24]. Such heterogeneity of cortical response differences suggests that a more widespread cortical deficit could underlie ASD symptoms. It is important to note that there are a few alternative theories that could account for a difference in 'mirror system' responses during imitation and movement observation. For instance, it has been shown that resting state brain activity in ASD individuals is different from that of controls [26]. As most fMRI studies compare responses during a given task with a baseline at rest, their results depend on resting state activity just as on activity during the task. Any relative increase in resting state activity could, therefore, mistakenly be interpreted as reduced task-related activity (for example, reduced responses during imitation in area vPM).

Yet another hypothesis suggests that some forms of autism are caused by an increased ratio of excitation to inhibition throughout the brain [27]. Such an imbalance could generate random fluctuations in the activity of many neurons that are no longer under normally strict inhibitory control and will alter the results of statistical parameter mapping methods that are commonly used for fMRI data analysis. These statistical maps depend not only on the difference in response to the two conditions (for example, task *versus* rest) but also on the noise standard deviation. Thus, in a situation where autistic brains exhibit the same mean response amplitudes as controls, yet have greater fluctuations, a decreased statistical significance could mistakenly be interpreted as a lack of activation. These alternative explanations could explain not only the reported 'mirror system' findings, but perhaps some of the response differences in other brain areas not associated with the human mirror system.

A perhaps more important question is whether a 'mirror system' dysfunction could explain the multitude of heterogeneous symptoms associated with ASD. Proponents of this theory often rely on the observation that individuals with ASD have difficulties imitating or understanding the intentions of others [28]. There is, however,

considerable variability in the ability of individuals with ASD to imitate and, importantly, this ability is not correlated with other ASD characteristics such as language development and adaptive behavior, as one might expect given a unitary underlying mechanism for the dysfunction [29]. Furthermore, while autistic individuals are thought to have difficulty understanding the intentions of others in more complicated 'theory of mind' tasks, they do not have difficulty understanding the intentions of simple movements common in daily situations [30,31]. A specific dysfunction in mirror neurons, as the theory suggests, would be expected to affect this ability regardless of the contextual complexity.

There are several other behaviors that are characteristic of ASD, yet seem completely unrelated to the function or dysfunction of mirror neurons. For instance, individuals with ASD are better than neurotypical controls at visual tasks involving the perception of local features of the input [32]. Also, individuals with ASD are often either hypersensitive or hyposensitive to particular sensory stimulation in the auditory, olfactory, gustatory and tactile domains. It is difficult to see how such general differences in several perceptual modalities could be related in any way to mirror neuron function. The connection between ASD and the 'human mirror system' becomes even more tenuous if we focus on other physiological characteristics associated with ASD. Several studies report multiple anatomical differences between neurotypical and ASD individuals that include differences in cell morphology, cortical thickness, overall brain size and sub-cortical volumetric measurements mostly in brain areas not related to the 'human mirror system' [33].

Taken together, it is not obvious how a dysfunction in the 'human mirror system' might account for the multiple behavioral and physiological characteristics of ASD. In fact, the general search for a unitary explanation (a 'missing link') capable of explaining all the ASD characteristics seems like a counterproductive direction for the field [34]. It is indeed interesting to speculate about the possible roles of mirror neurons (and their dysfunction) in human cognition. But we must remember that the connection between mirror neuron dysfunction and ASD is speculative at best and it is worth considering the implausibility that a multitude of behavioral and physiological alterations can be accounted for by a small population of visuomotor neurons that comprise only ~10% of the neurons in only two cortical areas.

Furthermore, in testing the mirror system dysfunction hypothesis, we must also remember the importance of ruling out alternative theories. Showing a correlation with the predictions of a theory provides only weak evidence in support of that theory. Showing a positive correlation with the predictions of one theory and negative correlations with the predictions of alternative hypotheses provides much stronger support. We urge the proponents of the mirror system dysfunction hypothesis to attempt to do just that.

Conclusions

Mirror neurons are exceptionally interesting neurons, which may underlie certain social capabilities in both animals and humans. However, the study of mirror neurons and the 'human mirror system' in particular has been characterized by much speculation and relatively little hard evidence. There is no reason why this should be the case. Further neurophysiology experiments are needed to assess the selectivity of these neurons in non-human primates quantitatively with a particular emphasis on establishing a causal connection between mirror neurons and the ability of the animal to understand observed movements. In parallel, non-invasive techniques in humans should be used to assess movement selectivity as the defining feature of mirror neurons rather than using imitation and movement observation protocols, which activate many brain regions in a non-selective manner.

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Primer

Coral reefs

Nancy Knowlton

Coral reefs, renowned for their diversity and beauty, are often called the 'rainforests of the sea'. They form best in warm, clear, well-lit waters (Figure 1) where they fringe shorelines, form offshore barriers and ring volcanoes, becoming atolls once the volcanoes themselves sink below the surface — a process first outlined by Darwin. Some of the structures coral reefs form can even be seen from space, although in total they occupy just 600,000 km^2, or about 0.1% of the surface of the planet. There are also deep-water coral reefs, but they will not be considered further here.

Today most reefs are primarily built by members of the order Scleractinia, skeleton-forming relatives of sea anemones whose fossil record dates back to the Triassic. The taxonomic relationships of scleractinian corals have been in turmoil for a number of years — many traditional groupings are not supported by modern molecular analyses — and species boundaries are also often difficult to define. Other important reef builders today include fire corals, blue corals and coralline algae. In the geological past, reefs have been formed by many kinds of organisms, including microbes, sponges and clams.

Although all corals, like other members of the phylum Cnidaria, can capture prey using their stinging cells, the ability of some corals to grow at rates sufficient to form reefs is due to their nutritional symbiosis with single-celled algae — a group of dinoflagellates that are broadly referred to as zooxanthellae. Zooxanthellae provide their coral hosts with the products of photosynthesis, and in turn the corals provide nutrients to the zooxanthellae. For many years it was thought that all zooxanthellae belonged to a single species, but it is now recognized that zooxanthellae represent a highly diverse collection of symbionts that differ in their light and host preferences and in their life histories.

Most coral reef biodiversity lies not with the corals themselves (~1000 species) but rather with the many other organisms that live on reefs. Their numbers are highly uncertain, with estimates ranging from about one million to about 9 million species, and we know little about the extent to which

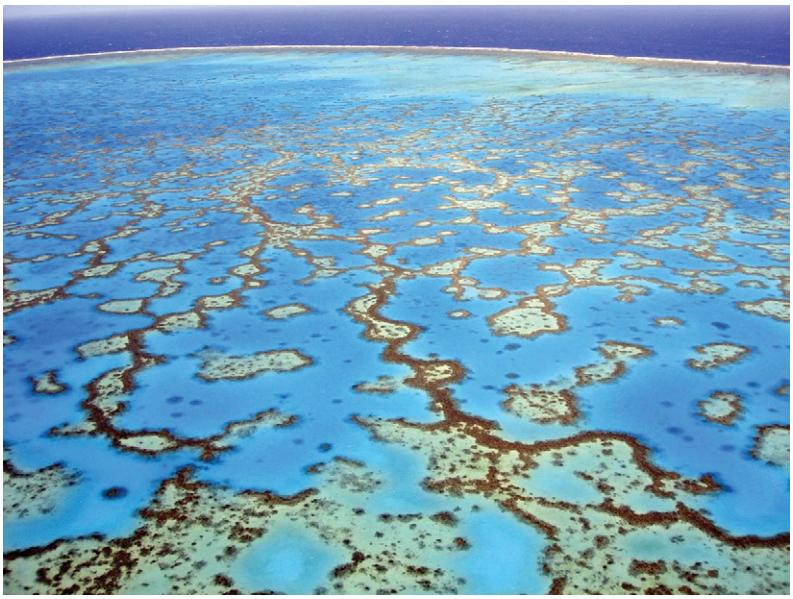

Figure 1. Aerial view of the coral reefs of Heron Island, Great Barrier Reef, Australia. Photo courtesy of Ove Hoegh-Guldberg.