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Mirror Neuron Forum

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Question 1: Do Mirror Neurons in Macaques or Humans Make an Important Contribution to Action Understanding?

Initial answers to Question 1

Vittorio Gallese (VG). To answer this question, we should first clarify how motor acts and actions are mapped within the cortical motor system and what the notion of *action understanding* means. One might think that motor neurons would discharge in association with the activation of specific muscle groups or during the execution of elementary movements. In fact, however, a crucial functional property of macaque premotor area F5 (and of the posterior parietal regions reciprocally connected to it) is that most of its neurons are active only during motor acts, which are movements executed to accomplish a specific motor goal such as grasping, tearing, holding, or manipulating an object. The most interesting F5 neurons are those discharging any time the monkey grasps an object, regardless of the effector used, be it the right hand, the left hand, the mouth, or both hand and mouth (see Rizzolatti, Fogassi, & Gallese, 2000).

What are those F5 neurons doing? The data strongly suggest that they map between the observer's goals and the acting animal's goals. Umiltà et al. (2008) demonstrated the independence between how the effector moves and the motor end-state it attains. All tested neurons in area F5 and half of the neurons recorded from the primary motor cortex discharged in relation to the accomplishment of the goal of grasping, regardless of the movements made to accomplish it. This property also fully applies to mirror neurons (MNs, Rochat et al., 2010). The sensory-to-motor direct mapping enabled by MNs goes beyond the mere kinematic features of movement. That is, the mapping is between the goal of an animal's executed actions and the goal of another animal's actions, even if the other's movement are only partially seen (Umiltà et al., 2001) or, indeed, even if the other's movement is not seen but the consequences are

heard (Kohler et al., 2002). Also, fMRI evidence shows that posterior parietal and ventral premotor areas are activated in humans by the observation of goal-related motor acts or by listening to action-related sounds (see Rizzolatti & Sinigaglia, 2010). A similar functional property was revealed in congenitally blind patients (Ricciardi et al., 2009). Goal-dependency has also been demonstrated with humans using transcranial magnetic stimulation (TMS; Cattaneo, Caruana, Jezzini, & Rizzolatti, 2009). TMS was used to measure the amplitude of motor-evoked potentials (MEPs) recorded from participants' hand muscles during the observation of action. It is important to note that the MEPs measured when grasping were similar both when using regular pliers so that the hand closed to effect the grasp and when using reverse pliers so that the hand opened to effect the grasp.

MNs provide the first neural mechanism allowing a direct mapping between the visual description of a motor act and its execution. This mechanism provides a parsimonious solution to the problem of translating the visual analysis of an observed movement—in principle, devoid of meaning for the observer—into something that the observer understands because it is directly mapped onto the observer's motor representations. Some critics of the hypothesis that MNs contribute to action goal recognition suggest that MNs function much like neurons in extrastriate visual areas (e.g., the superior temporal sulcus, or STS), which are sensitive to biological motion (Hickok, 2009). However, the extrastriate neurons do not show goal relatedness (Cattaneo, Sandrini, & Schwarzbach, 2010).

Let us now turn to mindreading. For decades, the prevalent opinion has been that in humans, action understanding predominantly—or even exclusively—relies upon reading the

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minds of others (Baron-Cohen, 1995; Baron-Cohen, Leslie, & Frith, 1985). This view is based on the assumption that the observable behavior of others is intrinsically intentionally opaque as it only consists of biological motion. According to the same view, only mind reading can translate a ''moving hand'' into a ''grasping hand.'' The discovery of MNs in the macaque monkey brain and subsequent evidence of mirroring mechanisms in the human brain suggested a more straightforward mechanism enabling the understanding of others' behavior.

Two recent papers showed that a high percentage of macaque premotor and parietal MNs respond during the execution and observation of grasping depending on the overarching goal of the action in which the grasping is embedded: Some MNs only respond when grasping leads to bringing the object to the mouth, but not when it leads to putting the object into a container, and other MNs respond when the grasp is for putting the object into a container but not the mouth (Bonini et al., 2010; Fogassi et al., 2005). Thus, MNs map integrated sequences of goal-related motor acts (grasping, holding, bringing, placing) clustering them into ''syntactically'' separate and parallel intentional actions. Compelling evidence shows the same in humans (Brass, Schmitt, Spengler, & Gergely, 2007; Cattaneo et al., 2008; Iacoboni et al., 2005).

Several neuropsychological studies (Buxbaum, Kyle, & Menon, 2005; Moro et al., 2008; Negri et al., 2007; Pazzaglia, Smania, Corato, & Aglioti, 2008; Saygin, Wilson, Dronkers, & Bates, 2004; Tessari et al., 2007) and TMS-induced inactivation studies (Urgesi, Calvo-Merino, Haggard, & Aglioti 2007; Urgesi, Candidi, Ionta, & Aglioti, 2007) show a strong correlation between action execution impairments and action recognition/understanding deficits. The occasional single-case dissociations within such cohorts (e.g., as discussed by Hickok, 2009) need explanation, but they cannot override the overall group-level congruence.

In conclusion, it is fair to say that action understanding, even at a basic level, does not necessarily require the activation of MNs. It has been shown that communicative actions, when implying motor acts outside of the human motor competence (e.g., observing a barking dog) are easily understood without any involvement of the observer's cortical motor system (Buccino et al., 2004). However, this does not imply that action understanding obtained without mirroring is the same as that based upon it. I submit that it is only through the activation of MNs that we can grasp the meaning of others' behavior from within. In virtue of the translation of others' bodily movements into something that the observer is able to grasp as being part of a given motor act accomplished with a given motor intention, the observer is immediately tuned in with the witnessed motor behavior of others. This enables the observer to understand others' motor goals and motor intentions in terms of her/his own motor goals and motor intentions.

Greg Hickok (GH). I will focus on MN function in macaques because this provides the foundation on which many theories regarding the human mirror system are built. If there is reason to question the role of MNs in action understanding in macaques, there is likewise reason to question to the role of the mirror system

in human action understanding as well as generalizations of this idea to concepts such as empathy and theory of mind.

It is instructive to consider the motivation for the action understanding theory of MN function. MNs were discovered in the context of research on how visual object information can be used to select appropriate motor actions for grasping (Rizzolatti et al., 1988). It was found that cells in motor cortex responded both during object observation and object grasping; in many instances, the object and grasp shape complemented one another. It was argued that visual features were used to access a motor ''vocabulary'' of possible grasping actions (Jeannerod, Arbib, Rizzolatti, & Sakata, 1995; Rizzolatti et al., 1988). Notably, the visual response properties of these cells were not considered to be the basis of *object understanding*—a function that was relegated to semantic systems in the temporal lobe. Rather, the visual response properties were thought to reflect a nonsemantic, ''pragmatic'' function of the dorsal processing stream (Jeannerod et al., 1995).

Like the visual object-responsive cells described above, MNs were found to show a mirror correspondence between the visual and motoric properties. A natural interpretation of MNs, then, would have been that they were part of the pragmatic dorsal stream and reflected a mechanism whereby actions can access a motor vocabulary for executing similar actions. There is no question that others' actions are important to action selection (di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992), but an action-selection theory of MN function was rejected from the start in favor of the action-understanding theory—why?

The reason appears to be that no behavior in the macaque repertoire was clearly available to explain the existence of MNs. Imitation is the obvious behavior that a MN might support, but macaques, it was noted, don't imitate (Rizzolatti & Craighero, 2004). It was instead proposed that MNs support action understanding. In other words, because the most likely interpretation seemed to fail empirically, a different interpretation, one at odds with the model for object-responsive neurons, was put forward (see Figure 1). But more recent evidence shows that forms of imitation (observational learning, cultural transmission) are in the repertoire of macaques and a range of other species (Hickok & Hauser, 2010).

Infant macaques, for example, have been reported to imitate overtly (Ferrari et al., 2006), and adult macaques can learn and replicate an abstract sequence of actions (tapping on a set of pictures in a prescribed order) by observing an ''expert'' macaque (Subiaul, Cantlon, Holloway, & Terrace, 2004). Observational learning of this sort has been described in a range of species including domesticated dogs (Range, Viranyi, & Huber, 2007), mongooses (Muller & Cant, 2010), bottlenose dolphins (Krutzen et al., 2005), bats (Page & Ryan, 2006), fish (Schuster, Wohl, Griebsch, & Klostermeier, 2006), and invertebrates (Fiorito & Scotto, 1992). Thus, the ability to observe an action and use that visual input to select a similar action appears to be a common ability in animals.

These observations breathe new life into the more straightforward interpretation of MNs, namely that they support ''action selection'' just like canonical neurons (Hickok &
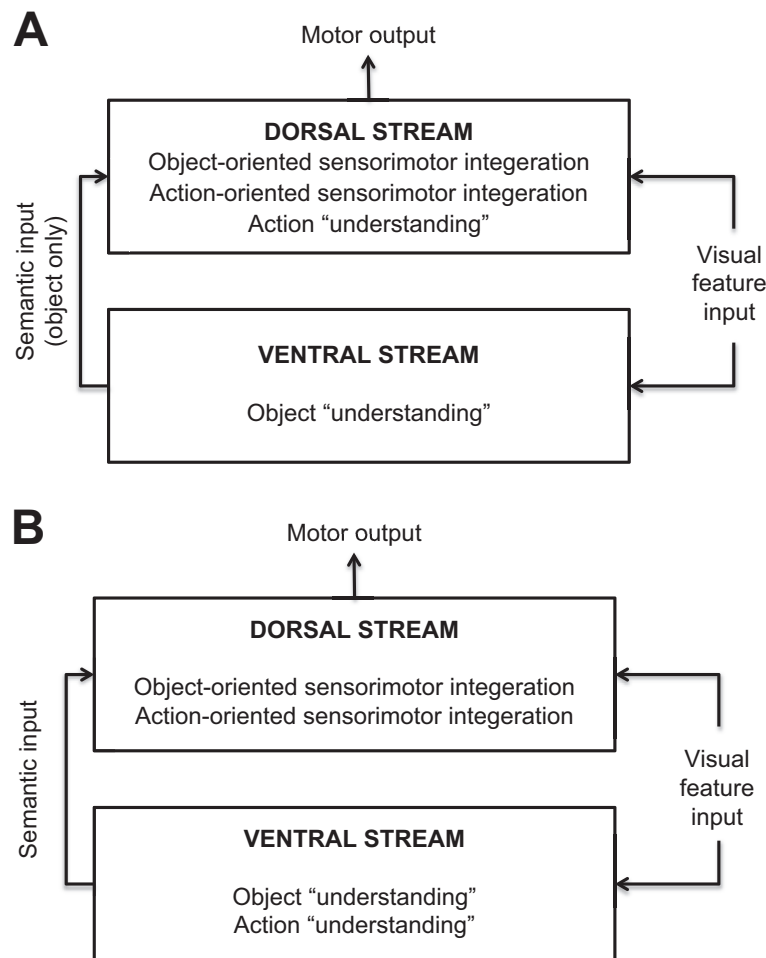
Figure. 1. Schematic models of dorsal and ventral stream function. A: The mirror neuron theory of action understanding, which holds that object- and action-oriented processes for sensorimotor integration and "understanding" are organized differentially: Action understanding is part of the dorsal sensorimotor stream and object understanding is part of the ventral stream. B: An alternative model, promoted here, in which object- and action-oriented processes for sensory-motor integration and understanding are organized similarly. Both models assume that semantic information from the ventral stream can modulate sensorimotor processes in the dorsal stream. Figure from Hickok & Hauser, 2010.

Hauser, 2010). On this view, action understanding is a function of the ventral semantic or "what" stream while the dorsal stream, which includes the mirror system, supports a sensory-motor pragmatic or "how" function. It seems likely that MNs are only a subclass of cells that respond to dynamic actions: Although mirror responses may be appropriate in some situations, in many others (such as an attack), a nonmirror response (flight) may be most appropriate. This predicts the existence of "antimirror neurons" that take one action as input and select a different but appropriate action response.

The action selection theory isn't the only alternative to the action understanding theory. Others have suggested that MNs acquire their properties purely by association between executing an action and observing the visual consequences of one's own actions (Heyes, 2010). The extent to which MNs

support action understanding, action selection, or simple association is an empirical question. Given these alternatives, an empirical reevaluation of the functional role of MNs in both monkeys and humans is much needed.

Marco Iacoboni (MI). MNs discharge while performing actions and while perceiving the actions of others. This pattern of discharging activity would provide a functional mapping between the motor aspects of one's own actions and the perceptual aspects of the actions of others. The internal motor knowledge of the observer becomes the frame of reference to which the perceptual aspect of the actions of other individuals is mapped.

Admittedly, it is very difficult to obtain empirical evidence that unequivocally proves this hypothesis. There is, however, both imaging and neurological evidence that is compellingly consistent with it. Tasks requiring predictions about perceived

actions differentially activate premotor areas in comparison with matched action-related cognitive tasks not requiring prediction (Stadler et al., 2010). Furthermore, neurological patients with lesions in inferior frontal (Fazio et al., 2009) and inferior parietal (Kalénine, Buxbaum, & Coslett, 2010) cortex—two MN areas—have selective impairments in action understanding. For instance, patients with inferior frontal lesions and no apraxia are impaired in temporal re-ordering of pictures of human actions but not of physical events (such as a falling object; Fazio et al., 2009).

An objection to this interpretation is that we understand actions we can't perform (Hickok, 2009). My mom does not play tennis, but she still has some kind of understanding of what is going on when Federer hits a backhand volley. This objection, however, misrepresents the kind of understanding MNs would provide. When I watch the same backhand volley, my internal motor knowledge of how to hit a backhand volley gives me a much richer understanding (an understanding from 'within'; Rizzolatti & Sinigaglia, 2010) of that action. That is, I can typically predict ball direction, speed, and placement—even a few moments before Federer's racquet hits the ball—according to racquet orientation, body position, and motion.

An alternative interpretation is that cognitive, inference-based mechanisms would activate MNs after action recognition has occurred (Csibra, 2007). The discharge of MNs during action perception would then simply be a by-product of this high-level, inference-based cognitive recognition of the action. Although this hypothesis does not explain the imaging and neurological data discussed above, it does make a specific prediction with regard to the timing of visual and motor discharges during action recognition. The visual discharge must necessarily precede the motor discharge. Empirical data in both monkeys and humans, however, demonstrate that the timing of visual and motor discharge is equivalent (Ferrari, Gallese, Rizzolatti, & Fogassi, 2003; Fogassi et al., 2005; Gallese, Fadiga, Fogassi, & Rizzolatti, 1996; Kohler et al., 2002; Mukamel, Ekstrom, Kaplan, Iacoboni, & Fried, 2010). Data from single cells in humans also show that temporal and frontal units with mirroring properties discharge at the same time during action perception (Mukamel et al., 2010). Taken together, the empirical data support the hypothesis of an important contribution of MNs to action understanding.

Replies to Question 1

VG. I will respond to some of the points raised by GH. First, the major premise of his initial argument is debatable. According to GH, if one can question the relevance of MNs to action understanding in monkeys, then this would automatically jeopardize any conclusion about the role of MNs in human social cognition. Why? Do we assume that a given trait or neural mechanism found in different species must necessarily preserve identical characteristics? Evolutionary theory patently contradicts this assumption.

Second, GH's parallel between the functional relevance of canonical and MNs, both explained in terms of action

anticipation, can be questioned. According to GH, both classes of neurons instantiate the action-oriented coding typical of the dorsal stream, whereas object and action semantics would be exclusively provided by the ventral stream. However, an exclusive action-oriented characterization of the dorsal stream falls short of explaining the functional role exerted by the ventral part of the dorsal stream (the *ventro-dorsal stream*) that reciprocally connects cortical areas of the inferior parietal lobe to ventral premotor areas (see Gallese, 2000, 2007a, 2007b; see also Rizzolatti & Gallese, 2006; Rizzolatti & Matelli, 2003). The meaning we attribute to objects is not exclusively the outcome of their visual description as instantiated by extra-striate visual areas within the ventral stream. That is, objects are not merely identified and recognized by virtue of their physical "appearance" but also in relation to the effects of the potential interaction with an agent, like looking at the object by moving the eyes, moving around the object, interacting with the object, etc. (Gallese, 2000, p. 31). Indeed, in 1997, Rizzolatti and I wrote: "[. . .] objects, as pictorially described by visual areas are devoid of meaning. They gain meaning because of an association between their pictorial description (meaningless) and motor behavior (meaningful)" (Rizzolatti & Gallese, 1997, p. 223). Object perception is not simply intertwined with action. Action also constitutively shapes the content of perception by characterizing the perceived object in terms of the motor acts it may afford, and this characterization occurs even in the absence of any effective movement, as epitomized by the firing of canonical neurons during object observation (Gallese & Sinigaglia, in press).

Third, GH's statement that action understanding is a function of the ventral semantic or "what" stream can also be questioned. Where is the "what" of action in the ventral stream? Perhaps, GH would argue, it can be found in the STS. However, no evidence supports this argument, and in contrast current evidence demonstrates quite the opposite. As recently shown by Cattaneo et al. (2010), only the motor system—and not the STS—can generalize a given motor goal independently from the effector accomplishing it.

I agree with GH that more work is needed to firmly establishing a relationship between MNs and action understanding. However, the present evidence shows that this relationship is very likely.

Morton Ann Gernsbacher (MAG). There is a very obvious argument against the need for MNs in action understanding: Simply put, we are able to understand a myriad of actions we've never executed. For example, the vast majority of ballet patrons are likely to have never donned a pair of toe shoes, much less executed a relevé or arabesque. Yet, for centuries, humans have understood these and other actions that they've never executed and can only observe. If this were not true, then spectator sports—from wrestling to whale watching—would conjure nary a fan.

VG responds to this well-known fact by conceding that MNs are "not necessarily" required for action understanding. MI agrees. However, MI claims that previous experience executing an action provides "a much richer understanding"

of that action. Similarly, VG claims that "only through the activation of MNs" can we fully "grasp the meaning" of actions.

Thus, according to VG and MI, prior experience executing an action engenders a deeper understanding of the action, which is achieved through more robust MN activity. Although MI agrees that it's "very difficult to obtain empirical evidence that unequivocally proves this hypothesis," it's not very difficult to obtain empirical evidence that consistently falsifies this hypothesis because such evidence already exists (Muhlau et al., 2005; Peigneux et al., 2004; Rumiati et al., 2005; Tanaka, Inui, Iwaki, Konishi, & Nakai, 2001).

Every study ever published that manipulates, in MI's words, participants' prior "internal motor knowledge" of an action—that is, every study to contrast imitation of meaningful, familiar, well-known actions with meaningless, novel, never-executed-before actions—demonstrates the same thing: Increased experience executing actions is associated with decreased, not increased, activation in putative MN regions.

For example, Tanaka et al. (2001) measured activation while participants imitated well-known symbolic finger gestures, such as the ok sign or the peace/victory sign, or performed unknown (and therefore meaningless and nonsymbolic) finger gestures. "No significant [additional] activation was detected" (p. 1173) for well-known symbolic gestures compared with unknown meaningless gestures. Rather, it was the unknown (i.e., never executed before) gestures that were associated with significantly more "bilateral parietal activation" (p. 1172).

As another example, M[u]hlau et al. (2005) measured activation while participants repeatedly imitated each of only two "stereotyped" finger or hand gestures or while participants imitated a wide set of meaningless, novel, never-executed-before finger or hand gestures. Again, it was the novel gestures, not the stereotyped gestures, that were associated with the greatest activation in putative MN regions: "bilateral activation of the inferior parietal cortex (BA 40), the superior parietal lobe (precuneus, BA 7), the inferior frontal cortex (opercular region including BA 44 on the right and BA 9/44 on the left" (p. 1091).

Similar findings are reported by Rumiati et al. (2005), who manipulated the proportion (0%, 30%, 50%, 70%, or 100%) of meaningful, familiar, well-known, and understood actions versus meaningless, novel, never-executed-before actions, and Peigneux et al. (2004), who contrasted meaningful limb, hand, or finger gestures that the participants knew so well that they could easily name them with meaningless limb, hand, or finger gestures that were unknown to the participants.

The repeated finding that increased experience executing actions is associated with decreased, not increased, activation in putative MN regions, lies completely opposite that predicted by MI for executing actions that are more "common," more "prepotent," and are "learned early on in human development" (Iacoboni, 2005, p. 83, 2007, p. 441). MI predicts that executing these more familiar actions that "yield better [behavioral] performance . . . should produce greater activity in mirror areas" (Iacoboni, 2005, p. 83). However, as reviewed above, increased experience executing actions is not associated with increased activity in putative MN regions. Although prior experience executing an action might engender a deeper understanding of the action, that "deeper understanding" is not achieved by more robust MN activity during execution of that action.

GH. VG makes a strong case for the view that MNs are not enabling action understanding via strict motor simulation. He argues instead that MNs are responding to the goals of the movements, not the movements themselves. The question then becomes, what are these goals? VG characterizes them as "motor goals." But how can the goal be motoric if the movements are independent of the goal? Instead, the goal must be cognitive (to possess an object) or sensory (to taste a raisin, satiate hunger). Stated more generally, we don't move for the sake of movement. We move to effect a change in the environment or ourselves. There is no specific meaning that is inherent to a movement. Reaching for a cup has different meanings depending on whether the outcome is a sip of water or the clearing of a table, and the outcome can be achieved via any number of movements, including asking someone else to do it. Rather, movements inherit their meaning from the consequences they effect.

Now, if the goals (meaningful effects) of movement are not motoric and if, as VG claims, MNs "map between observer's goals and the acting animal's goals," then it follows that MNs have nothing to do with the motor system. This is not likely what VG intends to argue and indeed seems like the wrong result.

There's a better solution. MNs are part of the motor system and code a relation between cognitive/sensory goals, represented elsewhere in the brain, and possible motor solutions to achieving those goals. That is, MNs reflect one stage in the process of activating and selecting among the possible actions. Why do they respond during action observation? Because the actions of others are relevant for selecting our own actions in the same way that the shape of an object is relevant for selecting an action directed toward it. We are not surprised when neurons in F5 respond both during object observation and object-directed actions and we do not endow such cells with the power to understand objects. Why should we treat MNs any differently?

A comment on the neuropsychological evidence cited by VG is warranted. These studies do find correlations between action execution and recognition deficits, but correlations can be misleading and dissociations are also attested in the literature (Hickok, 2009; Mahon & Caramazza, 2008). Regarding the latter, VG writes, "The occasional single-case dissociations within such cohorts . . . need explanation, but they cannot override the overall group-level congruence." These dissociations are not occasional—they comprise 33% of a sample in a recent prominent paper (Pazzaglia et al., 2008)—and the MN theory of action understanding has no explanation of them.

GH. Iacoboni writes, "The internal motor knowledge of the observer becomes the frame of reference to which the perceptual aspect of the actions of other individuals is mapped . . . Admittedly, it is very difficult to obtain empirical evidence that

unequivocally proves this hypothesis. There is, however, both imaging and neurological evidence that is compellingly consistent with it."

There is also evidence that is compellingly consistent with the hypothesis that the sun revolves around the earth. We need to dig deeper, though, considering both evidence that is consistent with the claim as well as evidence that is inconsistent with it. Often times, it is the inconsistent evidence that is so revealing. By looking beyond the sun's "motion" and considering the (apparent) retrograde motion of planets, we now appreciate that a heliocentric model does a better job of explaining all the evidence.

We are in the same situation regarding the interpretation of mirror neuron function. On first glance, we see the neural equivalent of the sun rotating around the earth: cells in motor cortex that fire both during action execution and observation, associations between motor (in)activity and action perception, and so on. But on closer examination, there are anomalies: individuals who can perceive speech without the ability to produce it (Bishop, Brown, & Robson, 1990), others who can recognize emotion in facial expression without the ability generate the expressions themselves (Bogart & Matsumoto, 2010), and the fact that we all can recognize and understand actions that we can't execute (e.g., flying, coiling). These facts, like apparent retrograde planetary motion, demand explanation.

Two classes of models have been proposed to deal with these facts. One retains the motor-centric view of understanding (from "within") and explains the anomalies as falling under a different, impoverished, perceptual mechanism (Rizzolatti & Sinigaglia, 2010). The other aims for a unified perceptuo-centric approach in which all actions are understood via the same non-motor-dependent mechanism (Hickok, 2009; Hickok & Hauser, 2010; Mahon & Caramazza, 2008).

Iacoboni underlines the notion that the motor system provides understanding "from within," noting that having played tennis he can "typically predict ball direction, speed and placement." There is no doubt that motor knowledge can augment perception in this way. But how? Is it that the ball direction and velocity (the sensory consequence of the action and Iacoboni's operational definition of "understanding" in this example) are directly coded in the motor programs for generating a tennis swing? No. These are visual features that can be detected and coded even in non-tennis players. Rather, what tennis experience adds is the ability to better predict the sensory consequences of a swing as a result of prior experience with the association between a movement and its consequences (Hickok, Houde, & Rong, 2011). Sensory events, including actions, are analyzed and "understood" by sensory systems; motor knowledge just allows the sensory systems to do the job sooner or more efficiently. The motor system isn't the only source of such predictions. Just watching a player's repertoire of swings repeatedly would also allow the viewer to predict direction and velocity of the ball (i.e., sensory learning exists). Further, in expert tennis players it may be helpful to use sensory learning rather than one's own motor knowledge if one's adversary has a different kind of swing. Sensory learning of this sort is exactly what a prey animal, for example, would need in order to predict an eminent attack by another animal with a very different motor repertoire, such as the coiling of a snake.

MI. The ventral/dorsal dichotomous interpretational framework (as invoked in GH's answer to Question 1) is too simplistic to account for extant data on the MN system (and probably other systems too). We now know that MNs exist in areas well outside the classical dorsal stream (Mukamel et al., 2010). Furthermore, it is unclear why hypotheses about the functions of MNs must be mutually exclusive. It is unlikely that MNs play an important role in action selection (a role typically attributed to more classical premotor neurons, especially in the dorsal premotor cortex) since they fire throughout the action, well after the action has been selected. It is likely, on the other hand, that association learning plays some role in shaping the responses of MNs (as suggested in CH's answer to Question 4). However, there is no reason to assume, as GH does, that because association learning plays a role in shaping MN responses that MNs cannot implement any form of action understanding.

Cecilia Heyes (CH). In their initial responses, VG and MI supported the view that MNs make an important contribution to action understanding, whereas GH opposed it. Curiously, however, the two sides did not seem to disagree about the data. For example, VG and MI didn't criticize studies suggesting that there can be action understanding without MN involvement, and GH didn't question the data reviewed by VG in his first three paragraphs. This makes me wonder whether Question 1 is really an empirical question—whether the claims and counterclaims about action understanding currently appearing in the MN literature could really be resolved by empirical means.

So what is the dispute really about? My guess is that it concerns not what causes action understanding, but what constitutes action understanding. The Parma group, influenced by the philosophical tradition of phenomenology (Rizzolatti & Sinigaglia 2008), believe that MN activity constitutes or "amounts to" a basic kind of action understanding. According to this view, it is appropriate to say that I have "understood" Action X, if observing X activated in me neural mechanisms that are also involved in producing X. It is not necessary for this activation to have any further consequences: for example, enabling me to name X or to make logical inferences about X. In contrast, GH and others with a background in cognitive science believe that this usage of the term *action understanding* is, at best, misleading. It is perilously close to obscuring the distinction between responding and understanding and to giving the impression that high-level "semantic" understanding of actions depends on MNs. If this reading is correct, the debate is substantial but not empirical—there are significant theoretical issues at stake, but they can't be resolved by collecting more data.

VG emphasizes that monkey MNs match observed and executed action "goals," not "the mere kinematic features of movement." This emphasis suggests another potential rationale for the claim that MN activity constitutes action understanding. Perhaps the emphasis on goals, and on what MNs "are doing," is an implicit appeal to the idea that MNs were
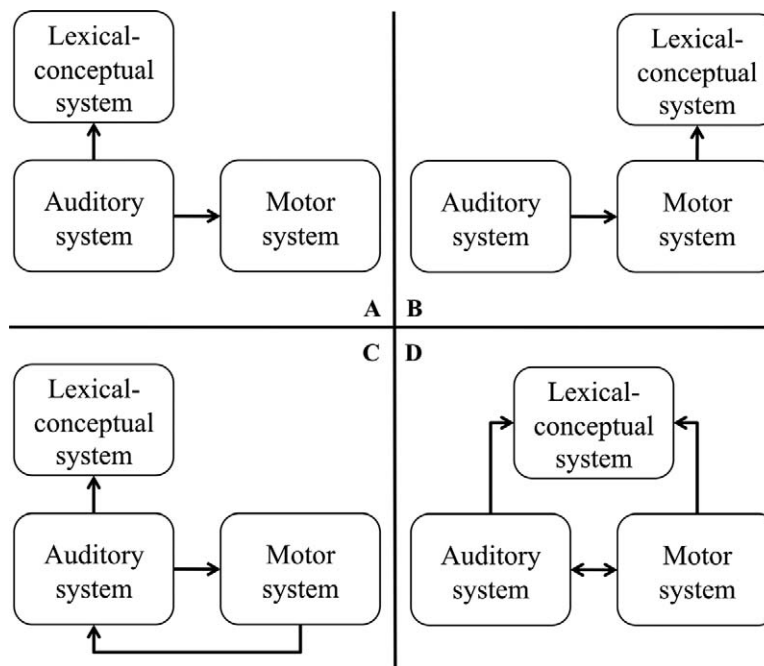
Fig. 2. Diagram of different theories of speech perception: auditory (A), motor (B), auditory allowing for motor modulation of auditory analysis (C), and a sensorimotor hybrid (D).

"designed" by evolution for action understanding. If so, it is important to note that the current evidence suggests that MNs are forged by sensorimotor associative learning and that they may not evolve for action understanding or any other function (Heyes, 2001, 2010; see Question 4). This associative account does not preclude the possibility that MNs are recruited in the course of development to make a contribution to action understanding (Press, Heyes, & Kilner, in press), but it does suggest that the properties of MNs are very far from being fixed (Press, Catmur, Widman, Heyes, & Bird, 2011). For example, it predicts that if monkeys were given contingent experience of seeing and doing kinematically defined movements, then they, like humans, would develop kinematic-matching, in addition to goal-matching, MNs.

Question 2: Do Mirror Mechanisms Causally Contribute to Speech Perception and Language Comprehension?

Initial answers to Question 2

   GH. This, in my view, is a critical question with import not only for speech perception but for the whole of the MN debate. Speech perception is an ideal test case for the action understanding theory of MN function, much like speech served as a test case for the idea of cortical specialization in the 19th century. MN theory was generalized to speech perception in the earliest publications, and there is a rich theoretical and empirical literature on which to draw. If the action understanding

interpretation fails for speech perception, it raises serious questions about the theory generally.

   In considering claims about the role of the motor system in speech perception, it helps to frame the question in terms of simple speech perception architectures. According to auditory theories of speech perception (Diehl, Lotto, & Holt, 2004; Holt & Lotto, 2008), speech sound recognition happens in the auditory system and these auditory representations serve as the input to the lexical system which in turn drives lexical-semantic access (Fig. 2A). According to motor theories (Fadiga & Craighero, 2006; Liberman, Cooper, Shankweiler, & Studdert-Kennedy, 1967; Liberman & Mattingly, 1985), speech perception happens in the motor system, which, although typically not discussed, must serve as the input to the lexical system (Fig. 2B).

   These are the extreme positions but there are intermediate models. One is an auditory theory that allows for motor modulation of the auditory analysis (Hickok, 2009, 2010; Hickok, Holt, & Lotto, 2009; Schwartz, Basirat, Menard, & Sato, 2009; Fig. 2C). Another is a sensorimotor hybrid in which speech recognition happens as a consequence of joint auditory and motor processing. These composite auditory-motor representations then serve as the input to the lexical system (cf. Pulvermüller, 1996 Fig. 2D).

   The motor theory architecture is easy to rule out as there is unequivocal evidence that one does not need an intact motor speech system to perceive speech (Hickok, 2010). High levels of speech perception ability have been demonstrated in (a) patients with severe motor speech deficits and damage to the mirror system (Rogalsky, Love, Driscoll, Anderson, & Hickok,

2011); (b) individuals who failed to develop a functional motor speech system due to neurological disease (Bishop, Brown, & Robson, 1990; Lenneberg, 1962); (c) infants who have not yet developed motor speech control (Eimas, Siqueland, Jusczyk, & Vigorito, 1971); and (d) even chinchilla and quail (Kuhl & Miller, 1975; Lotto, Kluender, & Holt, 1997), which don't have the biological capacity for speech. Further, complete and acute functional disruption of the motor speech system via left intracarotid amobarbital injection (Wada) induces only a $\sim 8\%$ phonemic error rate in speech recognition (Hickok et al., 2008) and much of this effect may be caused by deactivation of left auditory systems.

On the other hand, recent research showing that modulation of the motor system can affect speech perception (D'Ausilio et al., 2009; Meister, Wilson, Deblieck, Wu, & Iacoboni, 2007; Mottonen & Watkins, 2009; Watkins & Paus, 2004), at least under some task conditions (Hickok, 2010; Hickok et al., 2009), suggests that a purely auditory theory doesn't capture the whole story either. Some form of intermediate model is needed.

I suggest that an architecture that is fundamentally auditory but allows for motor modulation is most consistent with the facts. Specifically, motor effects on perception tend to be small ($\sim 10\%$ modulation), have been demonstrated only in partially ambiguous acoustic stimuli (speech in noise), and at least a portion of this effect may be attributable to postperceptual factors rather than perceptual discrimination (Sato et al., 2011). These facts coupled with the observation that speech perception is at or near ceiling levels even with the motor system severely compromised, is evidence for an asymmetry in computational importance between the auditory (driving) and motor (modulatory) speech systems.

If the motor system has only a small modulatory effect, why is it involved in perception at all? The answer, we suggest, is that the perceptual modulation developed to support motor function (Hickok, Houde, & Rong, 2011). Specifically, motor commands generate a corollary discharge that generates an internal forward model predicting the sensory consequences of motor commands. The sensory prediction is realized as an auditory gain modulation (prediction is much like attention). Given the existence of such a mechanism in the motor control system, perhaps it can be co-opted (exapted, in the evolutionary sense) for perception of others' speech under some circumstances.

The fundamental claim of the action-understanding theory of MN function—that MNs are the "basis" of action understanding—does not hold in the speech domain despite early claims to the contrary (Gallese et al., 1996; Rizzolatti & Arbib, 1998). This fact, I suggest, questions the validity of the fundamental claim generally.

MI. TMS and fMRI studies have demonstrated activation of motor areas during speech perception (Fadiga, Craighero, Buccino, & Rizzolatti, 2002; Pulvermüller et al., 2006; Watkins, Strafella, & Paus, 2003; S.M. Wilson, Saygin, Sereno, & Iacoboni, 2004). Furthermore, connectivity analyses show that the speech production motor areas active during speech perception are functionally connected with auditory areas in the superior temporal cortex traditionally associated with the analysis of speech sounds. Crucially, when TMS modulates activity in these speech production areas, speech perception is modulated accordingly (D'Ausilio et al., 2009; Meister et al., 2007; Sato, Tremblay, & Gracco, 2009). Mirror mechanisms may implement top-down motor-based models of the acoustic input (Iacoboni, 2008b; S.M. Wilson & Iacoboni, 2006) that could be useful for speech perception in noisy contexts that are almost the rule in real life situations. Indeed, fMRI and magnetoencephalography (MEG) data for correct and incorrect responses during acoustic phonetic identification in noise are consistent with this model (Callan, Callan, Gamez, Sato, & Kawato, 2010). Arguments against this model have invoked the mostly production deficits in Broca's aphasia (i.e., if MNs in Broca's area used in production are also important in speech perception, then damage to Broca's area should affect both production and perception) and the limited interference effects of TMS (Hickok et al., 2009). However, Broca's aphasia is associated with speech perception deficits, especially when input is degraded (S.M. Wilson, 2009). Also, TMS only produces a painless and very transient modulation of brain activity, and dramatic TMS effects should not be expected. Taken together, these data and arguments suggest a causal role of mirror mechanisms for speech in speech perception.

Evidence for a causal role of mirror mechanisms in language comprehension is less direct. Although there are many behavioral studies demonstrating a modulatory role of motor behavior on language processing (Glenberg & Kaschak, 2002; Glenberg et al., 2008; Havas, Glenberg, Gutowski, Lucarelli, & Davidson, 2010) the neural correlates of these effects are yet to be investigated. However, a causal role of mirror mechanisms in these effects is plausible. Indeed, several fMRI studies have demonstrated relationships between neural mirroring and language processing (Aziz-Zadeh, Wilson, Rizzolatti, & Iacoboni, 2006; Buccino et al., 2005; Hauk, Johnsrude, & Pulvermüller, 2004; Tettamanti et al., 2005). The logic of these studies is that a role of neural mirroring in language comprehension predicts overlapping activation of specific body part maps or coordinated action maps for action production, action perception and the processing of linguistic stimuli that describe either corresponding body parts or corresponding coordinated actions (Fernandino & Iacoboni, 2010). The results of all these studies are consistent with a relationship between mirror mechanisms and language comprehension, although these studies have yet to provide evidence causally linking mirror mechanisms to language comprehension. The results of these studies also support the embodied cognition model that argues that human cognition is grounded in our perceptual and motor experiences (Barsalou, 2008; Glenberg, 1997; Glenberg & Kaschak, 2002).

Finally, there is an evolutionary hypothesis linking area F5 of the macaque (where MNs were originally discovered) with a key language region of the human brain, the posterior part of Broca's area (Rizzolatti & Arbib, 1998). This hypothesis is in line with theories suggesting a critical role of gestures in the origins of language. MNs, by coding gesture production and perception may

have provided the neural mechanism for a preverbal, gesture-based form of communication (Corballis, 2010).

Replies to Question 2

VG. According to GH, the roles of MNs in speech perception and language understanding are to be considered tightly related: If a relationship between MNs and speech perception cannot be established, so the argument goes, it would follow that the connection between MNs and language understanding would be falsified.

I disagree with this logic. It is now clear that motor resonance can be detected at multiple levels. In humans, the cortical motor system is activated during the observation of a variety of motor behaviors, ranging from intransitive movements of body parts to goal-related motor acts and actions. Similarly, motor resonance has been demonstrated at the phono-articulatory level, as well as during the processing of action-related linguistic expressions (words and sentences) and of morpho-syntactical aspects of language (for review, see Gallese, 2008; Jirak, Menz, Buccino, Borghi, & Binkofski, 2010). A recent study, for example, shows that motor behavior shares with linguistic syntax an abstract representation, namely, a "means–end parse" independent of specific actions and goals (Allen, Ibara, Seymour, Cordova, & Botvinick, 2010). Even granting only a peripheral role to the motor system in speech perception—as argued by GH—wouldn't affect the impressive evidence showing a systematic involvement of the motor system in language processing and understanding. For example, it was recently shown that right handers preferentially activate the left premotor cortex during lexical decisions on manual-action verbs (compared with nonmanual-action verbs), whereas left handers preferentially activate right premotor areas (Willems, Hagoort, & Casasanto, 2010). Thus, right and left handers, who perform actions differently, use correspondingly different areas of the brain for representing action verb meanings.

Let us now briefly turn to the abstract and nonliteral uses of language. Aziz-Zadeh and Damasio (2008) proposed that the verbs in "to kick" (literal) and "kick off the year" (idiomatic) imply the same "kick" motor representation. TMS evidence supports this view. Glenberg et al. (2008) showed that abstract transfer sentences (e.g., "give the news") activate the motor system exactly as concrete transfer sentences do (e.g., "give the pizza"). In conclusion, all of these results, although preliminary, strongly suggest causal contributions of the motor system (and of MNs in particular) to language processing and understanding. It would be a mistake to dismiss the hypothesis as quickly as GH would like.

MI. In a "virtual lesion" repetitive TMS (rTMS) study on speech perception, the TMS effects over premotor cortex were, if anything, a little stronger than the TMS effects over the auditory cortex (Meister et al., 2007). However, the effects were not reliably different, suggesting that both structures participated in the functional process, in contrast to GH's suggestion that motor processes play a small, modulatory role in speech perception. Again, I find it counterproductive to focus on dichotomous models ("it's auditory," "no, it's motor"). These models, although didactically useful, tend to provide a limited understanding of the functional processes at play. Indeed, consistent with the model in GH's Figure 2D, the most successful recent computational models of action and perception disclose the intimate relationship between motor control and perception (Friston, Daunizeau, Kilner, & Kiebel, 2010; Friston, Mattout, & Kilner, 2011). Eventually, we will have to get rid of these labels altogether, because they seem to get in the way of a better understanding of the phenomena under investigation.

MAG. MI claims that MNs contribute causally to speech perception and offers three types of empirical data to support his claim. Unfortunately, the studies do not provide consistently robust and replicable evidence of "a causal role of mirror mechanisms in speech perception."

The first type of empirical evidence MI cites comprises three fMRI studies (Callan et al., 2010; Pulvermüller et al., 2006; S.M. Wilson et al., 2004). These studies are reported to have shown overlap in activation during speech perception and speech production. But in none of these studies were the critical analyses corrected to avoid false positives. For example, when identifying regions of the motor cortex active during both speech perception and speech production, Pulvermüller et al. (2006) corrected for false discovery rate when identifying regions active during speech production, but when determining whether these regions were also active during speech perception, Pulvermüller et al. (2006) left the analysis uncorrected while failing to adjust their very liberal significance level.

Only a minority of all published fMRI studies fail to correct for false positives (Bennett, Baird, Miller, & Wolford, 2010), because failing to do so is considered a "serious" shortcoming (Bennett, Wolford, & Miller, 2009, p. 418). If mirror mechanisms are necessary for speech perception—if, as MI claims, motor cortex plays a causal role in speech perception—then surely activation observed in that region during speech perception would be robust enough to withstand conventional corrections. But that doesn't appear to be the case, as illustrated by the fact that no meta-analysis or systematic review based on whole brain analyses has ever identified motor cortex as a region involved in, much less required for, speech perception (e.g., Turkeltaub & Coslett's, 2010, meta-analysis of over 20 experiments and 300 research participants; see also Price, 2000, 2010).

The second type of empirical evidence MI cites comprises two studies that apply TMS to regions of motor cortex and measure MEPs from the participants' lips or tongues while they perceive speech. The assumption is that if motor mechanisms play a causal role in speech perception, then MEPs measured from participants' lips or tongues should be amplified during speech perception. However, in one of the two studies MI cites (Watkins et al., 2003), MEPs recorded from participants' lips didn't differ in magnitude when participants perceived speech or nonspeech (i.e., sounds such as glass breaking, bells ringing, or guns firing—sounds that humans clearly do not produce with their lips). If motor mechanisms are causal for speech perception, then surely motor potentials would be expected to show a statistically significant boost when participants actually perceived speech. But that

didn't appear to be the case. Moreover, another study that MI doesn't cite (Sundara, Namasivayam, & Chen, 2001) failed to find any boost in MEPs measured from participants' lips while they were perceiving speech.

A third type of empirical evidence MI cites comprises studies that apply TMS to either the primary motor cortex or the ventral premotor cortex and examine directly the effects on speech perception performance. For example, D'Ausilio et al. (2009) applied double-pulse TMS to regions of the motor cortex believed to be involved in lip versus tongue movement and reported that TMS led to 10% faster identification of speech sounds produced with the lips (/ba/ and /pa/) versus tongue (/da/ and /ta/). However, a study that MI doesn't cite (Mottonen & Watkins, 2009), which applied rTMS to lip regions of the motor cortex, failed to find any modulation of perception of /pa/. Similarly, another study that MI does cite (Sato et al., 2009), which applied rTMS to the left ventral premotor cortex, also failed to find an effect on identifying speech sounds (/ba/ or /pa/); neither did this study find an effect on discriminating speech sounds (/ba/ vs. /pa/). However, the study did find an effect of TMS to the left ventral premotor cortex on a more complex task that interchangeably required discriminating speech sounds and discriminating speech syllables (/put/ vs. /bon/).

A characteristic of this third group of studies is that, for the most part, the studies reporting an effect of TMS on speech perception embedded their speech sounds in white noise, whereas the studies that didn't report such effects did not. It's well known that embedding speech stimuli in white noise and degrading speech in other deleterious ways encourages participants to adopt atypical strategies, which reveal atypical neural processes, including an atypical reliance on the ventral premotor cortex (e.g., Eisner, McGettigan, Faulkner, Rosen, & Scott, 2010; Wong, Lee, & Parrish, 2005; Zekveld, Heslenfeld, Festen, & Schoonhoven, 2006). Given the very slow response times reported in the studies MI cites (nearly 300% slower than normal, cf. Diehl, Kluender, Foss, Parker, & Gernsbacher, 1987; Foss & Gernsbacher, 1983), it's very likely these participants were engaging in atypical processing, which could well have led to atypical results.

In sum, the empirical studies MI cites to support his claim of a "causal role of mirror mechanisms for speech in speech perception" do not provide consistently robust and replicable evidence. As McGettigan, Agnew, and Scott (2010) recently concluded, "unambiguous evidence supporting an obligatory role for motor representations in the perception of normal speech remains hard to find" (p. E42). Therefore, I concur with Lotto, Hickok, and Holt's (2009) conclusion that although no speech scientist would deny that the processes involved in speech production interact with those involved in speech perception, the strong claims that speech production is causal to speech perception "are probably untrue given empirical evidence" (p. 112).

GH. I agree completely with MI that, "[m]irror mechanisms may implement top-down motor-based models of the acoustic input." I have proposed a similar view (Hickok et al., 2011). We differ, perhaps, in terms of the weight of functional contribution of these motor-based models to perception. Based on the

small size of the modulatory effect on perception, I argue that the motor system plays only a marginal role in speech perception (Hickok, 2010; Hickok et al., 2009; Hickok et al., 2011). I further argue that motor simulation of speech modulates the *primary* analysis in the auditory system. Thus, the motor system is but one of many sources of top-down constraint on acoustic analysis of the speech signal; others include lexical, syntactic, and visual speech information.

MI seems to give the motor system a more prominent role, at least in his other writings (Iacoboni, 2008b): "Speech perception . . . requires the integration of sensory and motor information" (p. 33), and this integration "would generate a . . . phoneme production simulation to be used for phoneme categorization" (p. 33). So, MI appears to subscribe to some version of Figure 2B in which the motor system is required for speech recognition and appears to be the pinnacle of the speech processing chain (phoneme categorization).

The idea that speech perception requires the integration of sensory-motor information is proven incorrect by a range of neuropsychological, developmental, and comparative data, as pointed out in my primary answer. MI takes issue with one of these sources of data: Broca's aphasia. It is true that previous research reported deficits on speech perception tasks in Broca's aphasics (Baker, Blumstein, & Goodglass, 1981; Miceli, Gainotti, Caltagirone, & Masullo, 1980). A reexamination of these data, however, has shown that this effect was due to the use of a biased dependent measure, percent correct, rather than a measure that controls for response bias, such as d′ (Hickok, 2010). Further, a recent study of patients with large lesions involving the mirror system reported at or near ceiling performance on speech perception/recognition tasks (Rogalsky et al., 2011). Damage to Broca's area, and the mirror system generally, appears to have little, if any, effect on the perceptual discrimination of speech sounds.

Finally, regarding speculations about a gestural origin of language evolution, it is relevant that a gestural form of human language exists today, namely the signed languages used by the deaf. The study of how these gestural forms of communication are organized in the brain could be highly instructive to theories of the origin of human language and the MN debate generally. Some evidence is available. Lesion work has shown that, as is the case in spoken language, disruption to sign language production does not necessarily cause deficits in sign understanding (Corina & Knapp, 2006; Hickok & Bellugi, 2001; Hickok, Bellugi, & Klima, 1998; Poizner, Klima, & Bellugi, 1987). And in functional imaging, one recent study of deaf signers reported that receptive processing of gestures yielded no activity in the mirror system (Emmorey, Xu, Gannon, Goldin-Meadow, & Braun, 2010).

Question 3: Do Mirror Mechanisms Contribute to Imitation?

Initial answers to Question 3

MAG. Reports of a type of neuron that is activated both when humans execute an action and when humans observe another human executing the same action promised an instant

understanding of the neural basis of imitation. However, this contribution has been more apparent than real due to confusion and misunderstanding.

For example, the process of imitation has, for some researchers, conflated the very meaning of an MN. Some researchers now claim that MNs are those neurons that are activated "during [action] observation and imitation" (Downey, Zaki, & Mitchell, 2010, p. 157; Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2006, p. 1276; Lieberman, 2010, p. 156; Saygin et al., 2004, p. 1800) or, similarly, that MNs are those neurons that are activated "during [both] imitation and [action] observation" (Molnar-Szakacs, Iacoboni, Koski, & Mazziotta, 2005, p. 989; Wolpert, Doya, & Kawato, 2003, p. 593).

But it is important to remember that imitation comprises observation; in fact, simultaneous imitation (used in the vast majority of the laboratory assays of imitation, e.g., Iacoboni et al., 1999) requires simultaneously imitating an action while observing that action. It is therefore unsurprising that cortical areas typically activated during observation would be also activated during imitation; imitation comprises observation, making tautological definitions of MNs that hinge on cortical overlap between imitation and observation (as opposed to observation and execution, without the visual input provided for imitation).

How did this tautology arise? Gernsbacher, Stevenson, and Schweigert (in press) have provided a clue. They analyzed the articles most frequently cited to support the seminal claims about human MNs, for example, claims such as "recent studies have shown activation of 'mirror neurons' in ... area F5 in monkeys and Broca's region in humans ... both during execution of hand actions and during observation of similar actions performed by other individuals" (Nishitani & Hari, 2002, p. 1211) and "macaque monkeys and humans are equipped with so-called 'mirror neurons' in the premotor cortex that respond both when an individual acts in a particular way and when the same individual sees someone else act in this same way" (Hauser, Chomsky, & Fitch, 2002, p. 1575). Of the hundreds of times citations were given to support these claims, the vast majority of citations were to these four studies: Rizzolatti et al.'s (1996) PET study; Grafton, Arbib, Fadiga, and Rizzolatti's (1996) PET study; Fadiga, Fogassi, Pavesi, and Rizzolatti's (1995) TMS study; and Iacoboni et al.'s (1999) fMRI study.

However, with regard to Rizzolatti et al.'s (1996) PET study and Grafton, Arbib, Fadiga, and Rizzolatti's (1996) PET study, the authors themselves warned that neither study replicated in humans the observation reported in monkeys. In neither study did activation detected "during action observation ... overlap with that detected during action execution" (Hari et al., 1998, p. 15061). Compellingly, hundreds of subsequent articles have misunderstood that crucial feature and have subsequently miscited these two studies.

With regard to Fadiga et al.'s (1995) TMS study, the authors also warned that their study failed to provide the requisite evidence to support the claims of MNs in the human inferior frontal gyrus (IFG). Fadiga et al. used TMS to stimulate the left

motor cortex, not the left inferior frontal cortex, and, as Rizzolatti, Fadiga, Fogassi, and Gallese note (1999), the primate motor cortex does not receive visual input—a striking disqualification for a home for MNs. Again, hundreds of subsequent articles have misunderstood that crucial feature and have subsequently miscited this study.

In fact, in 1999 (i.e., years after these seminal studies were published), Rizzolatti and colleagues concluded that "brain imaging experiments carried out in humans failed up to now to convincingly demonstrate the existence of a cortical circuit similar to that described in the monkey" (Binkofski et al., 1999, p. 3276). In 2000, Nishitani and Hari forthrightly stated that "none of these experiments has shown that exactly the same areas would be involved in both action execution and observation" (p. 913). "There is no convincing study showing that the same regions get activated selectively for self executed grasps [and for self executed observation of those grasps]," concluded Arbib, Billard, Iacoboni, and Oztop in 2000 (p. 984).

Thus, over a decade ago, authors of three studies most frequently cited as evidence of MNs in humans (Fadiga et al., 1995; Grafton et al., 1996; Rizzolatti et al., 1996) forthrightly stated that their three studies do not provide the necessary evidence. Nonetheless, their studies continue to be cited erroneously.

And what about the fourth study most popularly cited to support claims such as "mirror neurons are cells that fire during the execution of an action and during the passive observation of the same action performed by somebody else" (Fecteau et al., 2004, p. 2625)? The fourth most popularly cited article is Iacoboni et al.'s (1999) fMRI study, titled "Cortical Mechanisms in Human Imitation," which is fitting because Iacoboni et al. (1999) didn't compare humans spontaneously executing an action with humans passively observing another human executing that action. Rather, Iacoboni et al. (1999) required their participants to observe actions and simultaneously imitate those same actions. As previously argued, it's unsurprising that cortical areas typically activated during observation would also be activated during imitation—imitation comprises observation. And because imitation comprises observation, it's unclear whether action execution apart from imitation (i.e., without observation) would overlap in activation with observation. We know imitation does, by definition, which is most likely the origin of the tautological definition of MNs.

CH. Imitation has been defined in a variety of ways, many of them laden with a great deal of theoretical baggage. In experimental psychology and cognitive neuroscience, *imitation* is now commonly used to refer to the copying by an observer of the topography of a model's action. In this context, *topography* refers to the way in which parts of the actor's body move relative to one another rather than to an external frame of reference. For example, I am imitating you if I copy the way in which your fingers move relative to one another when you make a fist or grasp a cup. I am not imitating you if I merely move my hand in the same general direction or toward the same object.

Imitation is important in promoting cooperative social attitudes (Chartrand & Van Baaren, 2009) and as a foundation for imitation learning (i.e. the acquisition of topographically novel sequences of behavior by observation). Imitation learning is a crucial part of sports and dance training and may contribute to the cultural inheritance of skills (Heyes, in press; see Question 6).

Two studies provide evidence that mirror mechanisms can make a causal contribution to imitation (Catmur, Walsh, & Heyes, 2009; Heiser, Iacoboni, Maeda, Marcus, & Mazziotta, 2003). In both studies, disruptive rTMS of the IFG, a classical mirror area, selectively impaired imitative behavior. Heiser et al. (2003) found that, compared with occipital stimulation, rTMS of the IFG reduced accuracy in an intentional imitation task, in which sequential key pressing responses were cued by finger movement stimuli, but not in a control task, in which the key pressing responses were cued by dot stimuli. Catmur et al. (2009) used an automatic imitation task (Heyes, 2011a) to ensure that any effects of rTMS were due to disruption of perceptual-motor translation processes specific to imitation, rather than to disruption of working memory and other task-general executive processes. Participants made an abduction (outward) movement of the index or little finger of their right hand in response to a colored circle (e.g. orange stimulus – index finger response; purple stimulus – little finger response). A task-irrelevant action stimulus, an image of an index or little finger abduction movement, was presented with the colored circle. In imitation trials, the action stimulus matched the correct response (e.g. index stimulus – index response), and in counterimitation trials the action stimulus was the alternative to the correct response (e.g. little stimulus – index response). The magnitude of the automatic imitation effect was measured by subtracting reaction times in imitation trials from reaction times in counterimitation trials. Catmur et al. found that, compared with posterior parietal stimulation and no stimulation, rTMS of the IFG abolished automatic imitation by delaying the onset of perceptual-motor translation.

It has been argued that mirror mechanisms could not contribute to imitation because monkeys have MNs but are unable to imitate (Rizzolatti & Craighero, 2004). When imitation is defined in an elaborate, theory-laden way—as the copying of an entirely novel action, guided by understanding of the model's intentions—it is true that there is no compelling evidence of imitation in monkeys. However, judged against the same empirically intractable standard, there is no evidence of imitation in humans. When imitation is defined in a leaner and more empirically accessible way—as the copying of action topography—there is evidence that monkeys (Voelkl & Huber, 2000, 2007), and indeed a range of other nonhuman animals (e.g. Mui, Haselgrove, Pearce, & Heyes, 2008; Range, Huber, & Heyes, 2010), can imitate.

The rTMS studies suggest that mirror mechanisms can make a causal contribution to imitation. This does not, of course, imply that mirror mechanisms are either necessary or sufficient for imitation. It is likely that under some conditions (defined by species, developmental history, types of action and other task

parameters) classical mirror areas are unnecessary for imitation, and it is inevitable that other mechanisms (e.g. low-level visual and motor processes) are also involved in generating imitative behavior. Furthermore, even if future research shows that mirror mechanisms are necessary for imitation under a broad range of conditions, this would not imply that mirror mechanisms are a biological adaptation for imitation. There is an extensive cortical network that is typically necessary for reading, but this network was not shaped by natural selection to make reading possible. Thus, the rTMS studies by Heiser et al. (2003) and Catmur et al. (2009) suggest that mirror mechanisms "can do" imitation, not that they are "for" imitation.

MI. Recent single-cell recordings demonstrated audio-vocal MNs in songbirds (Keller & Hahnloser, 2009; Prather, Nowicki, Anderson, Peters, & Mooney, 2009; Prather, Peters, Nowicki, & Mooney, 2008). These MNs innervate striatal structures important for imitative song learning, and their interplay with feedback sensitive neurons suggests they have a computational role in imitative learning.

Although there is no direct evidence that MNs in monkeys are involved in imitative behavior (Ferrari, Rozzi, & Fogassi, 2005; Ferrari et al., 2006; Kumashiro et al., 2003; Voelkl & Huber, 2000, 2007) and in the ability to recognize when others are imitating the monkey (Paukner, Anderson, Borelli, Visalberghi, & Ferrari, 2005; Paukner, Suomi, Visalberghi, & Ferrari, 2009), this hypothesis seems plausible. MNs in monkeys may influence behavior through a direct pathway to motor output structures for immediate imitation and through an indirect pathway via cognitive control structures (prefrontal cortex) for more complex forms of imitation, such as delayed imitation (Ferrari, Bonini, & Fogassi, 2009).

Imitation in humans is often linked to complex behaviors such as social learning and cultural transmission. Humans, however, tend to copy each other rather automatically, as in neonatal imitation or in facial mimicry. Early fMRI studies on imitation used simple actions already in the motor repertoire of the subject (Aziz-Zadeh, Koski, Zaidel, Mazziotta, & Iacoboni, 2006; Iacoboni et al., 2001; Iacoboni et al., 1999; Koski, Iacoboni, Dubeau, Woods, & Mazziotta, 2003; Koski et al., 2002; Molnar-Szakacs et al., 2005) and suggest that human MN areas are activated during imitative behavior. A study of human imitation using MEG is consistent with the fMRI studies and also provides information on timing of activation of these cortical areas (Nishitani & Hari, 2000).

Neuromodulation studies using TMS investigated the causal role of the posterior inferior frontal gyrus (pIFG), a human MN area, in imitative behavior. A high frequency rTMS study demonstrated that there was a selective impairment in imitation when TMS was applied to the pIFG, but not in a control sensory-motor task. Crucially, when TMS was applied to a control site, no TMS effects were observed (Heiser et al., 2003). Two later studies confirmed a causal link between imitation and pIFG (Catmur et al., 2009; Newman-Norlund, Ondobaka, van Schie, van Elswijk, & Bekkering, 2010), although one of them failed to show TMS effects that were specific to imitation (Newman-Norlund et al., 2010).

Imitative learning studies, in which subjects are supposed to learn actions that are not yet in their motor repertoire, have practical difficulties and have not been performed frequently. Two fMRI studies demonstrated that MN areas including pIFG interact with motor preparation areas and cognitive control areas such as the dorsolateral prefrontal cortex during imitative learning. The dorsolateral prefrontal cortex is particularly active while subjects are internally rehearsing and planning the imitative action, rather than during overt imitation (Buccino et al., 2004; Vogt et al., 2007).

Taken together, the evidence clearly supports the hypothesis that mirror mechanisms contribute to imitation. It is still unclear at which functional stage of imitative behavior MNs provide their most important contribution.

Replies to Question 3

VG. MAG's answer was rather surprising in two regards. First, her answer does not cite much of the relevant recent literature such as the meta-analysis reported in Caspers, Zilles, Laird, and Eickhoff (2010). Nor does she cite Gazzola and Keysers (2009), which demonstrates, on a subject-by-subject basis, overlapping activation in the human brain during action execution and observation. This demonstration strongly contrasts with MAG's suggestion that "it's unclear whether action execution . . . would overlap in activation with observation."

Second, MAG's characterization of some of the older literature is in need of correction. As she notes, some authors may have drawn incorrect conclusions from this literature, but those incorrect conclusions in no way diminish the very important (if not yet definitive) contributions of those papers. Contrary to what is implied by MAG, the PET study by Grafton et al. (1996) could not show any evidence of overlapping activation between action observation and execution conditions because it did not involve any participants' active movement! Second, Rizzolatti et al. (1996) never stated that their PET study provided evidence of a mirror mechanism in humans. This study was nevertheless important because it showed for the first time that BA 45 (in the IFG and part of Broca's area) was activated by observation of hand actions. Third, as MAG notes, the Fadiga et al. (1995) TMS study did not show the presence of MNs in the IFG because such a result would have been beyond the reach of their experimental approach. Instead, the study was the first to provide indirect evidence of a system matching action execution and observation in the human brain: When a TMS pulse was directed at the primary motor cortex (M1), it allowed measurement of the modulation of the motor system produced by the observation of grasping actions. As MAG notes, M1 is probably not well-populated with MNs. However, as discussed by Fadiga et al. (1995), the motor facilitation effect induced by TMS during grasping observation is likely due to the input of the ventral premotor cortex to M1, and this input almost certainly involves MNs. It may turn out that MNs are not crucial in action understanding and imitation, but a careful characterization of both older and more recent data suggests the opposite, namely that MNs causally contribute to action understanding and imitation.

CH. MAG shows how scientific "Telephone" can turn speculation into fact in a high-profile field of research. That's why it is important to fish from the ocean of research on MNs and imitation the few studies that demonstrate a specific, causal relationship. MI and I agree that the experiments which come closest to meeting this standard have shown that TMS-induced "virtual lesions" of the IFG impair performance in imitation tasks but not in control tasks (Catmur et al., 2009; Heiser et al., 2003). Previously, I noted that these findings suggest that MNs "can do" imitation, but that they are not "for" imitation. This message is underlined by the most recent experiment in the series (Newman-Norlund et al., 2010), showing that virtual lesions of the IFG impaired performance when participants responded with a power or precision grip, not only to corresponding body movements (imitation task), but also to strategically located dots (spatial task). A result of this kind would be surprising only if one assumed that classical mirror areas, such as the IFG, are dedicated to imitation.

MI. In songbirds, audio-vocal MNs are located in neural circuits important for birdsong learning, and these MNs have functional properties that suggest their role in imitative learning (Keller & Hahnloser, 2009; Prather et al., 2009; Prather et al., 2008). Thus, from an evolutionary perspective, it is likely that MNs in primates have a role in imitation.

MAG's claim that "it's unclear whether action execution apart from imitation (i.e. without observation) would overlap in activation with observation" is wrong. It is, indeed, very clear that activation during action execution without action observation overlaps with activation during action observation. There are many imaging studies that have demonstrated this overlap. Those studies, however, had interpretational ambiguities due to processing steps in group analyses of brain imaging data. A recent study has solved this residual problem (Gazzola & Keysers, 2009). It demonstrates at the single subject level, in all subjects and with unsmoothed data (to obtain the maximal anatomical precision), an overlap of activation between action observation and action execution.

The very first fMRI study on imitation already demonstrated (at the group level) overlapping activation for action observation, action execution (with no concurrent action observation), and imitation (Iacoboni et al., 1999). The argument made in that study is as follows: As MNs fire during both action observation and execution, but their firing rate changes are typically higher during execution, a human brain area containing these cells should be expected to be active during all three main experimental conditions: action observation, action execution (while no action was observed), and imitation. The magnitude of the signal change increase during these three conditions, however, should differ. Assuming a very simple model of roughly proportional relation between spiking rate changes and blood-oxygen level dependent (BOLD) signal changes, one would expect higher signal increase during execution (with no action observation) than during action observation, and even higher signal increase during imitation, because subjects both observe and execute the action while they imitate. This pattern of responses was found in the ventral premotor/inferior frontal

cortex and the inferior parietal lobule. Other studies also demonstrated similar responses in other cortical areas of motor significance, as in the medial wall of the frontal lobe, suggesting that those areas also contain MNs (Koski et al., 2003). Indeed, many years later, single cell recordings of neurological patients with implanted electrodes found human MNs in the medial wall of the frontal lobe (Mukamel et al., 2010). This is a rare case where brain imaging data anticipated findings later obtained with single unit recordings.

Question 4: Do Mirror Neurons Get Their Characteristic Visual-Motor Matching Properties From Learning?

Initial Answers to Question 4

CH. No one now doubts that learning plays a major role in the development of MNs and MN mechanisms. The interesting questions concern the types of experience involved in this learning, and the roles they play in development. In principle, the development of MNs could depend on seeing actions (sensory experience), on performing actions (motor experience), and/or on correlated observation and execution of the same actions (sensorimotor experience). Similarly, each type of experience could play a facilitative "tuning" role or an inductive "forging" role (Gottlieb, 1976). It would be a tuning role if the experience modulates the rate or specificity with which MNs develop the capacity to map observed onto executed actions, but MNs would eventually develop visual-motor matching properties even in the absence of the experience. It would be a forging role if the experience is necessary for the development of MNs—that is, if, in the absence of the experience, neurons in the inferior parietal and premotor cortex areas would not become responsive to the sight, as well as the performance, of certain actions.

As predicted by the "associative sequence learning" account of the origin of MNs (see Fig. 3; Heyes 2001, 2010), the results of training studies indicate that sensorimotor experience plays a forging role in their development. These experiments show that *mirror effects* (i.e., overt behavior, MEPs and BOLD responses that are widely believed to be caused by mirror mechanisms) can be enhanced (Press, Gillmeister, & Heyes, 2007), abolished (Cook, Press, Dickinson, & Heyes, 2010), and even reversed, by sensorimotor experience (Catmur et al., 2008; Catmur, Walsh, & Heyes, 2007).

In one case of reversal, Catmur et al. (2007) first showed that watching an index finger movement produces more activity in the observer's index finger muscle than in her little finger muscle. Then, half of the participants who showed this mirror effect were given incompatible sensorimotor training; they were required to make index finger responses to little finger movement stimuli and vice versa. The other half received control training; they were required to make index responses to index stimuli and little finger responses to little finger stimuli. When tested 24 hr after training, the control group showed the same mirror effect as at the beginning of the experiment, but the incompatible training group showed a reversed, "countermirror" effect; for example, observing index finger movement produced less activity in the observer's index finger muscle than in his or her little finger muscle. A recent replication, using dual pulse TMS, showed that incompatible sensorimotor training produces this effect by changing activity in the ventral premotor cortex, a classical mirror area (Catmur, Mars, Rushworth, & Heyes, 2010).

Reversal effects implicate sensorimotor experience in the development of MNs. During training, the control group watched and performed the actions with the same frequency as the incompatible training group. Therefore, the countermirror effect could not have been due to sensory and/or motor experience alone, but it requires their correlation: sensorimotor experience. The reversal effects also provide evidence that sensorimotor experience forges rather than tunes MNs. If compatible sensorimotor experience—for example, watching your own actions as you are performing them—merely played a facilitative or tuning role in the normal development of MNs, one might expect incompatible sensorimotor experience, received in adulthood, either to have no effect at all or to "blunt" MNs—to reduce the specificity with which they match observed and executed actions. One would not expect it to transform matching visual-motor properties into equally precise nonmatching visual-motor properties.

As far as I am aware, there is no evidence against the hypothesis that MNs are forged by sensorimotor experience. Such evidence could come in several forms. For example, the hypothesis would be challenged by data showing that any trait comparable with MNs can be both "genetically programmed" and reversed (rather than retarded or damaged) by atypical experience in adulthood. The hypothesis would also be challenged by evidence of "poverty of the stimulus"—for example, evidence that MNs show visual-motor matching properties before individuals have had sufficient sensorimotor experience to induce their development (Lepage & Théoret, 2007). Research on "imitation" in newborns does not provide such evidence (Ray & Heyes, 2011); the effect is robust only for tongue protrusion, and there is evidence that it is not mediated by MNs (S.S. Jones, 2009).

Thus, the current evidence suggests that the characteristic visual-motor properties of MNs are forged in the course of development by sensorimotor experience—correlated observation and execution of the same actions.

VG. We do not know whether MNs are innate and how their functions are shaped during development. The earliest indirect evidence available to date on the mirror mechanism in human infants is Shimada and Hiraki (2006). They used near infrared spectroscopy (NIRS) to demonstrate an action execution/observation matching system in 6-month-old human infants. Southgate, Johnson, Osborne, and Csibra (2009) showed with high-density electroencephalography (EEG) that 9-month-old infants exhibit signs of motor resonance (alpha-band attenuation over central electrodes) both during hand action execution and observation. The same authors showed a similar effect in
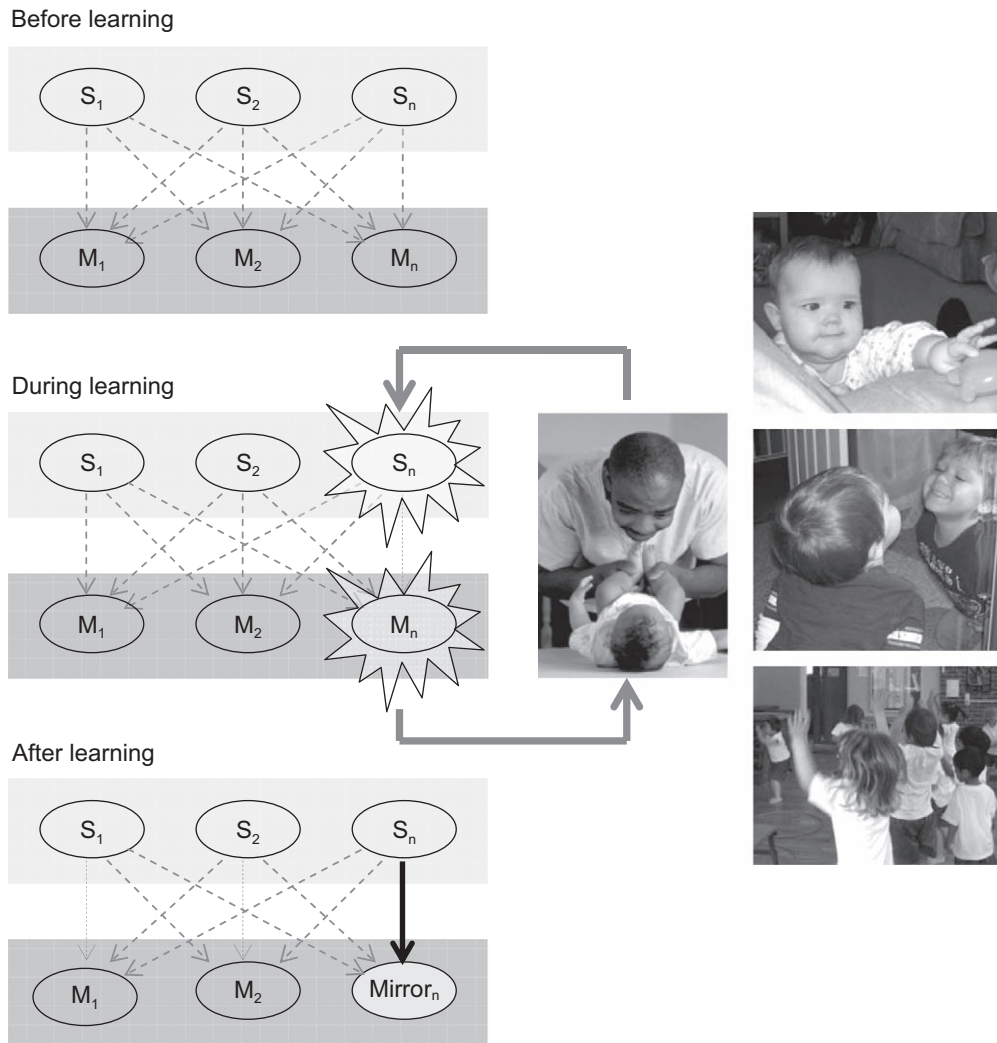
Fig. 3. The associative sequence learning account of the origin of mirror neurons (Heyes, 2010). Before learning, sensory neurons coding different types of action (S_1, S_2, S_n) are weakly and unsystematically connected to motor neurons coding different types of action (M_1, M_2, M_n). Learning occurs when there is correlated activation of sensory neurons (S_n) and motor neurons (M_n) coding similar actions (e.g., during self-observation or when an adult imitates an infant). Correlated activation of S_n and M_n increases the strength of the connection between them, so that activation of S_n is propagated to M_n. Therefore, after learning, M_n is a mirror neuron.

13-months-old infants with an experimental paradigm modeled on that of Umiltà et al. (2001) in which monkeys' MNs were tested during the observation of a hidden hand grasping.

Some authors have proposed that MNs are the outcome of a mere associative mechanism that binds the motor commands enabling action execution with the visual perception of the same action (Heyes, 2010; Keysers & Perrett, 2004). This hypothesis is certainly able to explain the plasticity of the mirror mechanism, but it is highly problematic with respect to its ontogenesis. First, this hypothesis does not account for mirroring mechanisms pertaining to motor acts performed with body parts like the mouth and the face to which neither monkeys nor humans have direct visual access. Second, this hypothesis is forced to downplay or even deny the plausibility of evidence for neonatal imitation both in nonhuman primates

(Ferrari et al. 2005; Myowa-Yamakoshi, Tomonaga, Tanaka, Matsuzawa, 2004) and humans (Meltzoff & Moore, 1977). Third, this hypothesis cannot explain why motor experience obtained without any visual feedback can affect perception of human biological motion related to that experience (Casile & Giese, 2006; Glenberg et al., 2010).

Del Giudice, Manera, and Keysers (2009) recently proposed that MNs might initially develop through experiential canalization of Hebbian learning, allowing for the possibility of some genetic preprogramming. According to this hypothesis, infants view themselves while acting. Then, visual neurons in the temporal cortex that respond selectively to the observed action as it unfolds reinforce the premotor neurons controlling the action and thereby induce Hebbian potentiation. This hypothesis bears the burden of explaining how visual selectivity for specific

actions is achieved by temporal cortex visual neurons. Furthermore, similarly to the abovementioned associative hypothesis, Del Giudice et al. (2009) cannot account for neonatal facial imitation and for the motor bias of perceptual recognition of biological motion.

I have provided an alternative account (Gallese, Rochat, Cossu, & Sinigaglia, 2009). Recent data show that hand motor control in humans is remarkably sophisticated well before birth (Myowa-Yamakoshi & Takeshita, 2006; Zoia et al., 2007). As an example, Castiello et al. (2010) showed that fetal twins, already at the 14th week of gestation, display upper limb movements with different kinematic profiles depending on whether they target the uterine wall or the body of the other twin. Furthermore, between the 14th and the 18th week of gestation, the proportion of self-directed movements decreases while that of the movements targeting the sibling increases. These data show that the human motor system, well before birth, is already instantiating functional properties enabling social interactions and that such social interactions are guided and expressed by motor rules specific to such interactions.

According to my hypothesis, before birth specific connections develop between the motor centers controlling mouth and hand movements and brain regions that will be the recipient of visual inputs after birth. Such connectivity, likely genetically predetermined, tunes the visual areas for spatio-temporal patterns of neural firing that correspond to the spatio-temporal patterns in motor areas during the execution of the mouth and hand movements. This tuning acts as a functional template. Once visual information is provided, the neonate would be ready to respond to the observation of hand or facial gestures that produce spatio-temporal patterns of activity matching the templates, thus enabling neonatal imitation and the reciprocal behaviors characterizing our postnatal life since its very beginning. A similar motor tuning of visual processing could also account for the advantages offered by motor experience, with respect to visual familiarity, observed in a variety of perceptual tasks performed by adults. When the relative contribution of visual and motor experience in processing an observed action is investigated, the results reveal greater activation of the mirror mechanism when the observed actions are frequently performed by the observers than when those actions are only perceptually familiar but never practiced (see Calvo-Merino, Grèzes, Glaser, Passingham, & Haggard, 2006).

Thus, an innate rudimentary mirror mechanism is likely present at birth, to be subsequently and flexibly modulated by motor experience and gradually enriched by visuo-motor learning. Lepage and Théoret (2007) proposed that the development of the mirror mechanism can be conceptualized as a process whereby the child learns to refrain from acting out the automatic mapping mechanism linking action perception and execution. This scenario found recent support from data obtained both in monkeys (Kraskov, Dancause, Quallo, Shepherd, & Lemon, 2010) and humans (Mukamel et al. 2010). Both studies show that MNs can be activated during action execution, but inhibited during the observation of actions done by others. The development of cortical inhibitory mechanisms likely leads the gradual transition from mandatory reenactment to mandatory embodied motor simulation.

MI. MNs for different effectors may be differentially sensitive to experience. Both newborn macaques and humans can imitate facial gestures (Ferrari et al., 2006; Meltzoff & Moore, 1977). Accordingly, preliminary EEG data on observation of facial expressions in one-week-old infant macaques demonstrate a significant suppression of central rhythms (Ferrari et al., 2008) in comparison with control stimuli, a finding that suggests neural mirroring in the very first days of life. It is likely, however, that early face-to-face interactions in humans (Stem, 1985; Trevarthen & Aitken, 2001) and macaques (Ferrari, Paukner, et al., 2009) shape MNs for facial expressions. Indeed, caregivers often imitate infants' facial expressions.

Mirror mechanisms for body parts that can be often seen early in life, such as the hand, may have a different developmental trajectory. The integration of vision and movement is critical in ontogeny for visually guided reaching and grasping (Held & Bauer, 1967). Hand MNs may be the product of such integration. This hypothesis is supported by studies on infants' perception of others' hand actions that seem to depend upon the acquisition of the corresponding motor skills (Bushnell & Boudreau, 1993; Sommerville, Woodward, & Needham, 2005). Indeed, EEG studies on 9-month-old infants demonstrate neural mirroring for reaching actions (Southgate, Johnson, Osborne, & Csibra, 2009), while 14- to 16-month-old infants show more pronounced neural mirroring for crawling rather than walking (van Elk, van Schie, Hunnius, Vesper, & Bekkering, 2008).

Evidence for experience-dependent changes in MN properties later in life, although not conclusive, is suggestive. In a recent study, monkeys were trained to use reverse pliers to grasp food (Umiltà et al., 2008). That is, the monkey had to open up the fingers (extension) to make the pliers grasp the food. After training, some neurons in area F5 had training-specific mirror properties: They fired during execution and observation of the action with the reverse pliers (Rochat et al., 2010).

In another study, after repeated observation of an experimenter using tools, 20% of MNs discharged when the monkey observed tool use actions (Ferrari et al., 2005), even though the monkeys were unable to use the tools. Mirroring responses for repeatedly observed actions that cannot be performed but achieve the same goals of actions within the motor repertoire of the observer may allow some understanding of unfamiliar actions and may represent a first key functional step in imitative learning.

Both fMRI and TMS have been used to investigate experience-dependent changes in human neural mirroring. In a TMS study, subjects were trained in countermirroring—that is, performing an action while watching a different one. After training, motor facilitation during action observation occurred not in the muscles specific to the observed action (the expected effect), but rather in the muscles specific to the executed action that had been associated to the observed one by countermirroring (Catmur et al., 2007). An fMRI study on countermirroring of observed and executed hand and foot

actions produced results in line with the TMS study (Catmur et al., 2008). These results, however, cannot unequivocally be interpreted as due to MNs, because it is well known that motor neurons can learn to associate a motor response to all sorts of sensory stimuli (Cisek & Kalaska, 2010). Taken together, the evidence suggests that the visuo-motor matching properties of MNs can be shaped by experience, but may also be influenced by other factors.

Reply to Question 4

CH. In my initial answer, I contrasted "tuning" and "induction" accounts of the role of learning, and pointed out that two kinds of evidence would count against induction—the forging of MNs by sensorimotor associative learning (Heyes, 2001, 2010). The first would indicate "poverty of the stimulus", and the second would show *innateness with reversability*—that a trait can be both "genetically programmed" and subject to reversal by novel experience in adulthood (Catmur et al., 2008, 2010, 2007).

VG outlined his tuning account and focused on putative evidence of poverty of the stimulus from NIRS and EEG studies of young infants and neonatal imitation. In neither case is the evidence compelling. It is not yet clear whether NIRS and EEG provide valid measures of mirror system activity in infants, and, even if they do, infants get plenty of the experience required to induce MN development in the first 6–13 months of life. On the topic of neonatal imitation, VG (and MI) cited only the high profile studies reporting positive effects. In a comprehensive review, Ray and Heyes (2011) examined 37 experiments testing for neonatal imitation of 18 gestures. Table 1 summarizes the results for the eight gestures that yielded at least one positive result. Consistent with previous reviews, Ray and Heyes found no valid and reliable evidence of imitation in neonates.

VG also cited evidence that the motor system is genetically tuned. Although fascinating, these data have no direct bearing on Question 4. Indeed, the associative account assumes that evolution has laid firm foundations for motor control, and that MNs are a byproduct of those foundations.

It is not clear why VG believes that the associative account of MNs "cannot explain why motor experience obtained without any visual feedback can affect perception of human biological motion related to that experience." Since its inception, the associative sequence learning (ASL) model has stressed that associative learning establishes bidirectional excitatory links between sensory neurons/representations and motor neurons/representations. The sensory-to-motor direction is relevant to imitation and to the visual properties of MNs, whereas the motor-to-sensory direction contributes to motor imagery and the kind of cross-modal adaptation effects reported by Glenberg et al. (2010). A recent study (Cook, Johnson, & Heyes, 2011) shows that, when the actions are more complex, rhythm cues also play a major role in cross-modal learning effects.

The "Hebbian hypothesis" (Keysers & Perrett, 2004) cannot account for mouth and face MNs. However, the original associative account, the ASL model (Heyes 2001, 2010), explains them with reference to sociocultural experience (see Fig. 3).

MI seems to agree that hand and arm MNs are forged by sensorimotor learning. However, relevant to "innateness with reversability," he questions whether the reversal effects reported by Catmur et al. (2008, 2007) were really mediated by mirror rather than motor neurons. In reply, I should first point out that, according to the ASL model, MNs *are* motor neurons—they are born as motor neurons, and they subsequently acquire matching visual properties through associative learning. Second, the latest, dual pulse TMS study (Catmur et al., 2010) confirms that the reversal effects are due to changes in mirror areas of premotor cortex.

In advocating a tuning account of face-related MNs, MI cites an abstract reporting that 1- to 7-day-old macaques show cortical activity in response to human facial gestures (Ferrari et al., 2008). This abstract does not indicate poverty of the stimulus because (a) there is no solid evidence that the EEG measure was indexing mirror system activity, (b) both facial expressions and a moving disk elicited the response, and (c) it does not tell us how much correlated sensorimotor experience the monkeys had received in their young lives.

Question 5: To What Extent Does Variability in Mirror Mechanism Functioning Contribute to the Autistic Phenotype?

Initial answers to Question 5

VG. For many years, a dominant paradigm in the study of the autistic spectrum disorder (ASD) characterized it as the consequence of a defective theory of mind, as a sort of "mind-blindness" (Baron-Cohen, 1995). After the discovery of MNs, several authors have proposed that abnormalities in MN mechanism functioning could be critical in autism (Dapretto et al., 2006; Gallese, 2003, 2006a; Hadjikhani et al., 2006; Oberman et al., 2005; Oberman & Ramachandran, 2007; Oberman, Ramachandran, & Pineda, 2008; Théoret et al., 2005; Williams et al., 2006; Williams, Whiten, Suddendorf, & Perrett, 2001). The malfunctioning of shared representations for perceived and executed actions would impair the capacity to translate others' perspectives into their own, thus potentially shedding light on ASD children's difficulties in imitation (Roger & Pennington, 1991; Rogers, Hepburn, Stackhouse, & Wehner, 2003; Williams, Whiten, & Singh, 2004).

The relationship between MNs and ASD, though, is controversial (see Dinstein et al., 2010; Fan, Decety, Yang, Liu, & Cheng, 2010; Hamilton, Brindley, & Frith, 2007; Southgate & Hamilton, 2008). However, it must be added that critics of the relationship between ASD and MNs are not always fully consistent. For example, Dinstein and colleagues (Dinstein, Hasson, Rubin, & Heeger, 2007; Dinstein, Gardner, Jazayeri, & Heeger, 2008), first concluded that MNs likely do not exist in the human brain, but later

Table 1. Summary of Research Seeking Evidence of Imitation in Human Neonates up to 6 Weeks Old

Gesture	Positive	Negative	Notes
Tongue protrusion	21: Abravanel (84)2 Abravanel (91)1, 2 Anisfeld (01) Heimann (89).1, .2 Jacobson (79) Kugiumutzakis (99).1- .4 Legerstee (1991) Maratos (82).1,.2 Meltzoff (77)1, 2 Meltzoff (83) Meltzoff (89) Meltzoff (92) Meltzoff (94) Vinter (86) (dynamic stimuli)	11: Abravanel (84)1 Fontaine (84) Hayes (81)1, 2 Heimann (85) Koepke (83)1, 2 Lewis (85) McKenzie (83) Ullstadius (98) Vinter (86) (static stimuli)	Reliable but nonspecific effect due to innate releasing mechanism or oral exploratory responses to arousing stimuli
Mouth opening	9: Kugiumutzakis (99).1-.4 Legerstee (1991) Meltzoff (77)1, 2 Meltzoff (83) Meltzoff (94) (duration)	20: Abravanel (84)1, 2 Abravanel (91)1, 2 Anisfeld (01) Fontaine (84) Hayes (81)1, 2 Heimann (85) Heimann (89).1,.2 Koepke (83)1, 2 Lewis (85) Maratos (82).1,.2 McKenzie (83) Meltzoff (92) Meltzoff (94) Ullstadius (98)	Side-effect of reliable tongue protrusion matching: Recovery of mouth opening responses after suppression during tonguing
Hand opening and closing	1: Vinter (86)	4: Abravanel (84)1 Jacobson (79) Vinter (86) (static stimuli) Fontaine (84)	Not reliable. When present, likely to be due to interdependence of facial and manual gestures
Lip protrusion	2: Meltzoff (77)1 Reissland (88)	3: Heimann (89).1,.2 Koepke (83)1	Not reliable. When present, likely to be due to scoring method
Sequential finger movement	1: Meltzoff (77)1	2: Koepke (83)1 Lewis (85)	Not reliable. When present, likely to be due to scoring method
Blinking	4: Kugiumutzakis (99).1-.4	2: Abravanel (84)1 Fontaine (84)	Not reliable. Could be a side-effect of attentional response to tongue protrusion model
Lateral head movement	3: Meltzoff (89) Maratos (82).1,.2		Likely to be due to perceptual tethering
Facial expressions of emotion	2: Field (82) Field (83)	1: Kaitz (88)	Not reliable. When present, could be due to scoring method

Note. Reprinted from Ray & Heyes (2011). Experiments in the "Positive" column reported a positive cross-target comparison (e.g. more tongue protrusion after observing tongue protrusion than after observing mouth opening), and experiments in the "Negative" column did not find a significant difference in cross-target comparison. The number at the beginning of each cell gives the total number of experiments in that cell. Studies are listed in alphabetical order. Each study is identified by the name of the first author and the last two digits of the year of publication. In cases where the published paper included more than one experiment, a digit following the author/year citation indicates the number of the experiment in which the result was found. Decimal digits indicate the stage in the sampling period where an effect was found in a longitudinal study. The "Notes" column summarizes the results of detailed review, reported in the main text and supporting material of Ray & Heyes (2011).

concluded that ASD individuals have normal MNs showing repetition suppression (Dinstein et al., 2010).

A clarification is in order. Being a spectrum disorder, it is unlikely that autism can be exclusively reduced to a deficit in motor cognition. It is even less likely that autism can be simply equated to a mere malfunctioning of the MN mechanism.

Both views appear too simplistic and fall short of capturing the multilayered and diversified aspects characterizing ASD. My point is different: Many of the social cognitive impairments manifested by ASD individuals might be rooted in their incapacity to organize and directly grasp the intrinsic goal-related organization of motor behavior (Gallese et al., 2009).

Several studies have investigated the neurological causes of the social impairments of ASD individuals. Some studies revealed an abnormal neural organization and connectivity during cerebral growth. For example, an increase of white matter seems to be at the origin of the anomalous head and brain dimension most frequently observed in ASD infants (see Courchesne et al., 2007). Also, Hadjikhani and colleagues (2006) found that the cerebral thickness of superior parietal, temporal, and frontal cortices is reduced in adolescents with autism. These regions include areas involved in social cognition, in facial expression and recognition, and, interestingly, parietal and premotor areas displaying the MN mechanism for actions.

Unlike typically developing children, autistic children are more likely to use motor strategies relying on feedback information, rather than on feed-forward modes of control (Schmitz, Martineau, Barthélémy, & Assaiante, 2003). As a consequence, autistic children are less likely to adopt anticipatory postural adjustments. An EMG experiment (Cattaneo et al., 2008) has documented that high-functioning autistic children are unable to organize their own motor acts into the intentional motor chains used by typically developing children. That is, the autistic children were impaired in smoothly chaining sequential motor acts within a reaching-to-grasp-to-eat intentional action. A similar impairment was also found in an action observation condition, and this impairment most likely accounts for the difficulty ASD children have in directly understanding the motor intention of an observed action executed by others. The presence of an atypical organization in action chaining has been further demonstrated by a recent study investigating the kinematics of intentional actions in autistic children (Fabbri-Destro, Cattaneo, Boria, & Rizzolatti, 2009). Furthermore, Boria and colleagues (2009) demonstrated that when presented with pictures showing hand-object interactions and asked what the individual was doing and why, children with ASD had no deficit in recognizing the kind (what) of goal-related action they observed. However, in contrast with typically developing children, autistic children had severe difficulties in understanding others' intentions (why) when they had to rely exclusively on motor cues. These results show that in the absence of a context suggesting the use of the object, ASD children guess others' motor intentions by using functional information derived from the object's semantics exemplifying its standard use.

All of these findings (for review, see Gallese et al., 2009), although far from being conclusive, strongly point to a relationship between impaired motor cognition and several aspects characterizing social cognition in ASD individuals.

MAG. Perhaps no other application of MN hypothesizing has been characterized by as much speculation as that of the relation between MNs and the autistic phenotype. Following one highly visible research study (Dapretto et al., 2006), the popular press buzzed that "Autism, Some Researchers Believe, May Involve Broken Mirror Neurons" *(New York Times,* Blakeslee, 2006) and that a "Lack Of 'Mirror Neurons' May Help Explain Autism" *(Scientific American,* Biello, 2005).

These headlines explicitly echoed the claims made by the researchers in their own press release, "UCLA Imaging Study of Children With Autism Pinpoints Broken Mirror Neuron System as Mechanism Behind Social Deficits" (Page, 2005).

Another highly visible research report (Oberman et al., 2005) was similarly heralded in the popular press (including the popular PBS television show, NOVA, 2005) and by the researchers themselves in their *Scientific American* article titled, "Broken Mirrors: A Theory of Autism" (Ramachandran & Oberman, 2006). Because one of these researchers had previously deemed MNs "the driving force behind 'the great leap forward' in human evolution" (Ramachandran, 2000), his subsequent claim that a group of humans lacked this evolutionary mechanism was deemed as "disturbingly . . . prejudiced" (Corwin, 2007) as similar declarations made about other groups of humans a century ago (Jones, 2010).

Because autistic persons, by diagnostic definition, are characterized by atypical social communication, the expanding assumptions that MNs underlie everything from speech perception to social interaction makes for an easy leap—as does autistic persons' "otherness." Attributions of MN efficiency and deficiency have been levied against other minority phenotypes, including persons who are sexually attracted to persons of the same sex (Ponseti et al., 2006), persons who stutter (Saltuklaroglu & Kalinowki, 2005), and persons who smoke cigarettes (Pineda & Oberman, 2006), with the latter attribution derived from the same laboratory paradigm as that used to attribute MN deficiency to autistic persons. However, of the two most prominent studies promoting the broken MN hypothesis of autism, one (Dapretto et al., 2006) failed twice to replicate, and the other (Oberman et al., 2005) not only failed twice to replicate but also failed to control one of the most crucial aspects of the study's design.

More specifically, whereas Dapretto et al. (2006) reported that, when imitating, autistic children exhibited significantly less activation "within the pars opercularis of the inferior frontal gyrus (BA 44) – the site with previously identified mirror properties – as well as in the neighboring pars triangularis (BA 45)" (p. 29), Williams et al. (2006), using Iacoboni et al.'s (1999) seminal mirror-neuron-imitation paradigm, and Martineau et al. (2010), using a similar paradigm, reported no differences between autistic and typically developing children in "the site with previously identified mirror properties."

In fact, in neither Williams et al.'s (2006) nor Martineau et al.'s (2010) attempt to replicate Dapretto et al. (2006) did even typically developing children exhibit a reliable amount of activation in "the site with previously identified mirror properties," a finding supported by a recent meta-analysis by Molenberghs, Cunnington, and Mattingly (2009): Of 20 fMRI studies testing samples of typical participants, only two studies reported significant activation in this site (and one of the two studies was from Dapretto et al.'s own lab; i.e., Iacoboni et al., 1999). The vast majority of studies (90%) with typical participants did not report imitation-specific activity in BA 44/45. Thus, Dapretto et al.'s (p. 30) conclusion that the autistic children's lack of imitation-specific activity in BA 44/45

indicated "dysfunction" that "may be at the core of . . . autism" and Iacoboni and Dapretto's (2006, p. 949) recommendation that lack of imitation-specific activity in BA 44/45 could be "an effective bio-marker" for autism lack empirical justification.

As for Oberman et al.'s (2005) study, an attempted replication by Raymaekers, Wiersema, and Roeyers (2009) found no significant differences in mu suppression between autistic and typically developing participants when executing or observing hand actions; neither did Fan et al. (2010), who measured their participants' eye movements to ensure that both groups of participants were attending equally to the stimuli (a design feature absent in Oberman et al., 2005; see also Bernier, Dawson, Webbs, & Murias., 2007, who reported no significant main effect of group or interaction between group and task during execution, imitation, and observation of hand actions).

In contrast to these two highly visible but nonreplicated studies, much larger and more firmly established bodies of data contradict predictions made by MN theory. For example, it has been repeatedly demonstrated that autistic persons of all ages (from preverbal children to mature adults) have no difficulty understanding the intention of other people's actions (Aldridge, Stone, Sweeney, & Bower, 2000; Carpenter, Pennington, & Rogers, 2001; Falck-Ytter, 2010; Hamilton et al., 2007; McAleer, Kay, Pollick, & Rutherford, 2010; Russell & Hill, 2001; Sebanz, Knoblich, Stumpf, & Prinz, 2005). Such well-established data argue against empirically unsupported speculations that autistic persons suffer from "defective intentional attunement" (Gallese, 2006a, p. 15).

As another example, alongside ample empirical documentation that autistic participants are less precise than nonautistic participants when they imitate other people, there are 30 years of empirical documentation that autistic participants are highly responsive when they are imitated by other people (Dawson & Adams, 1984; Dawson & Galpert, 1990, Escalona, Field, Nadel, & Lundy, 2002; Field, Field, Sanders, & Nadel, 2001; Heiman, Laberg, & Nordøen, 2006; Katagiri, Inada, & Kamio, 2010; Nadel et al., 2000; Tiegerman & Primavera, 1981, 1984). Such well-established and repeatedly replicated data contradict the core tenet of most MN proponents' assumptions about autistic people—for example, Gallese's (2006a) conjecture that autistic persons suffer from an "incapacity to establish a motor equivalence between demonstrator and imitator, most likely due to a malfunctioning of the mirror neuron system" (p. 21).

The "broken mirror neuron" hypothesis has led to unusual and unjustified interventions designed to assist autistic persons—from synchronized dance therapy (Ramachandran & Seckel, 2011) to playing with virtual pets (Atlschuler, 2008)—based on a hypothesis with a faulty empirical foundation and eroding empirical support (cf., Avikainen, Kulomaki, & Hari, 1999; Dinstein et al., 2010; Gowen, Stanley, & Miall, 2008; Leighton, Bird, Chairman, & Heyes, 2008; Press, Richardson, & Bird, 2010).

CH. The broken mirror hypothesis suggests that a deficit in mirror mechanism functioning is a primary cause of the autistic phenotype (Williams et al., 2001). More specifically, it suggests that an inborn mirror mechanism abnormality results in the impairment of perception-action matching (i.e., observation of an action does not reliably result in activation of a motor representation of the same action), and, as a consequence of this core impairment in perception-action matching, higher social cognitive functions, such as theory of mind, do not develop normally in autism spectrum conditions (ASC).

Evidence in support of the broken mirror hypothesis comes from studies showing that, relative to typically developing controls, individuals with ASC perform poorly in intentional imitation tasks—that is, procedures where participants are asked to imitate the actions of others (see Williams et al., 2004, for a review). However, intentional imitation tasks demand a range of operations in addition to perception-action matching. For example, when given an instruction such as "Do this!," the participant must use subtle social cues to infer which dimensions of action it is appropriate to copy; they must be motivated to comply with task requirements; attend closely to the modeled actions; and, in many cases, hold sequentially complex modeled actions in working memory until a response is cued. In contrast, automatic imitation tasks provide a more pure measure of perception-action matching by minimizing other cognitive demands. In these tasks, participants are not instructed to imitate. Instead, they are asked to make simple cued responses (e.g., to open their hand as soon as they see movement of a stimulus hand), and the speed of these responses is compared in "compatible" trials, in which the task-relevant cue appears with a task-irrelevant image of the correct action (e.g., an opening hand stimulus is presented when an opening hand response is required), and in "incompatible trials," where the cue appears with an image of the incorrect action (e.g., an opening hand stimulus is presented when a closing hand response is required; Heyes, 2011a). The magnitude of the automatic imitation effect—the strength of the tendency to match action with perception—is calculated by subtracting reaction time (or errors) in compatible trials from reaction time (or errors) in incompatible trials.

In automatic imitation tasks, participants with ASC perform as well or better than typically developing controls. Equivalent performance has been found in a hand opening/closing task, with both human and robotic movement stimuli (Bird et al., 2007); in a horizontal/vertical arm movement task (Gowen et al., 2008); in two tasks involving emotion-related facial movements (eyebrow raising/lowering and mouth opening/closing; Press et al., 2010); and in an index/middle finger lifting paradigm (Cook & Bird, in press; non-social priming groups). A recent study using the index/middle finger paradigm actually found a larger automatic imitation effect in people with ASD than in controls (Spengler, Bird, & Brass, 2010). In combination with data from carefully controlled intentional imitation tasks (e.g. Hamilton et al., 2007), these studies of automatic imitation indicate that the autistic phenotype is not due to a broken mirror mechanism impairing perception-action matching.

Autism is known to be a highly heritable disorder with complex genetic architecture (Yang & Gill, 2007). Some

researchers assume (a) that genetically heritable disorders produce their phenotypic effects by disrupting traits (the normal development of which is under tight genetic control), and (b) that the development of biological adaptations tends to be under tight genetic control. If these assumptions are correct, evidence that people with ASL have "broken mirrors" would support the view that MNs have evolved—that they are a biological adaptation for some kind of social cognition. In a complementary way, the evidence that people with ASC do not have broken mirrors removes an obstacle to the view that, rather than being an adaptation, MNs are an evolutionary byproduct of sensorimotor associative learning (see Question 4; Heyes, 2010).

MI. Among studies using brain activity as the outcome measure, there are 20 published papers that support the idea that variability in mirror mechanism functioning significantly contribute to the autistic phenotype, whereas four studies do not.

Eight fMRI studies demonstrated MN abnormalities in autism. Subjects with autism had reduced activity in pIFG when imitating and observing facial expressions. Activity in pIFG correlated with the clinical condition: the more severe the condition, the lower the activity (Dapretto et al., 2006). Abnormal activity in MN areas has been reported for imitation of finger movements (Williams et al., 2006) and perception of face (Bookheimer, Wang, Scott, Sigman, & Dapretto, 2008; Hadjikhani, Joseph, Snyder, & Tager-Flusberg, 2007), hand motion (Martineau et al., 2010), and emotions in others (Schulte-Rüther et al., 2010). Altered connectivity in autism has been shown between pIFG and other cortical areas (Shih et al., 2010; Villalobos, Mizuno, Dahl, Kemmotsu & Müller, 2005).

Only one fMRI study reported similar adaptation effects in autism and neurotypical subjects in mirroring areas during execution and observation of hand gestures (Dinstein et al., 2010). The neural correlates of fMRI adaptation studies, however, do not necessarily represent the adapting spiking activity of a specific neuronal population (Bartels, Logothetis, & Moutoussis, 2008) and findings from these studies cannot be confidently interpreted as reflecting MN activity.

Two structural MRI studies demonstrated anatomical differences between neurotypical subjects and subjects with autism in MN areas, namely the inferior parietal cortex (Hadjikhani et al., 2006) and pIFG (Hadjikhani et al., 2006; Yamasaki et al., 2010). The reduction of grey matter in pIFG correlates with the severity of social communication problems in autism (Yamasaki et al., 2010).

MEG demonstrated delayed activation of pIFG during imitation (Nishitani, Avikainen, & Hari, 2004) and reduced mirroring during action observation in subjects with autism (Honaga et al., 2010), whereas an earlier MEG study on 8 neurotypical subjects and 5 subjects with autism failed to show group differences during action observation (Avikainen et al., 1999), suggesting that statistical power is a key issue in these studies.

Several EEG studies show reduced mirroring in autism during action observation (Martineau, Cochin, Magne, & Barthelemy, 2008; Oberman et al., 2005, 2008). Reduced neural mirroring correlates also with imitative skills in autism (the more reduced is neural mirroring, the less skilled is the subject in imitation; Bernier et al., 2007). Neural mirroring measured with EEG also correlates with traits of autism in neurotypical subjects, such that the higher the autism traits, the more reduced the mirroring (Puzzo, Cooper, Vetter, & Russo, 2010). These correlations suggest that heterogeneity in both neurotypical subjects and subjects with autism may affect the outcome of these studies, leading to negative results (Fan et al., 2010; Raymaekers et al., 2009) if studies enroll neurotypical subjects high in autism traits and patients with autism that have only mild social cognition problems. Indeed, TMS studies demonstrated reduced neural mirroring not only in subjects with autism (Théoret et al., 2005) but also in neurotypical subjects with high traits of autism (Lepage, Tremblay, & Théoret, 2010; Puzzo, Cooper, Vetter, Russo, & Fitzgerald, 2009). Taken together, the evidence suggests that variability in mirror mechanism functioning significantly contributes to the autistic phenotype.

Replies to Question 5

CH. I'm not an expert on ASC. As a comparative outsider, I was surprised by two features of the first round of answers to this question. First, VG defended the role of impaired motor cognition, and not the role of mirror mechanisms, in producing the autistic phenotype. Second, while reviewing research on the brain activity of people with ASC, neither MAG nor MI explicitly interpreted the question in a causal way—as asking whether atypical mirror mechanism activity generates, rather than merely accompanies, ASC. Replication is certainly important. However, if we're interested in causality, surely what matters is not how many studies report abnormal mirror system activity, but which of them, if any, show that it is not just a corollary of attentional, motivational and/or executive function impairments. For example, eye-tracking studies could tell us whether atypical mirror system activity during action observation is a core feature of ASC or whether it is merely a consequence of people with ASC paying less attention to action stimuli or paying attention to different features of those stimuli.

VG. Two components of MAG's answer need to be addressed. First, when scientists make important discoveries, it is natural for them to be excited and for them to convey their excitement to the popular press and the public. Of course it is unfortunate if that communication leads to a simplification (at best) or distortion (at worst) of the discovery, but such reports in the popular press do not change the scientific facts. Second, as I argued in my answer to Question 5, I do not subscribe (and never did) to the thesis that autism equals broken mirrors. To the best of my knowledge, we do not have any reliable, single physiopathological hypothesis able to coherently explain the multifarious clinical aspects of ASD. At the least, the MN hypothesis has the dual merits of fostering a new way

of looking at autism and strongly challenging previously dominant accounts of autism resulting from a deficient theory of mind or faulty attachment. The motor cognition deficit account of autism is certainly worth further exploration as evidence supporting this hypothesis is accumulating (see Gallese et al. 2009, and MI's answer to Question 5). Similarly worth exploring is the recent evidence that faulty connectivity between the anterior insula and the amygdala (Ebisch et al., 2010) is a possible cause of the atypical gazing behavior of autistic individuals.

MAG. VG claims that autistic persons are characterized by an "incapacity to organize and directly grasp the intrinsic goal-related organization of motor behavior," in other words, VG claims that autistic persons are characterized by an incapacity to understand the intentions of theirs and other persons' motor behavior.

VG supports his claim by referencing head-growth studies (e.g., Courchesne et al., 2007), a cortical thickness study (Hadjikhani et al., 2006), which I review in my response to MI, two motor-control studies (Fabbri-Destro et al., 2009; Schmitz et al., 2003), and a study measuring muscle activation (Cattaneo et al., 2008). But none of those studies directly assess whether, as VG conjectures, autistic persons are incapable of understanding the intentions of action.

Indeed, missing in VG's list of empirical evidence are all but one of a decade's worth of studies, which do in fact directly assess autistic children and adults' understanding of the intentions of theirs and other persons' motor behavior (Aldridge et al., 2000; Carpenter et al., 2001; Falck-Ytter, 2010; Hamilton et al., 2007; McAleer et al., 2010; Russell & Hill, 2001; Sebanz et al., 2005). The studies VG fails to mention are unanimous in demonstrating that autistic individuals of all ages are perfectly able to understand the intentionality of their own actions and of other humans' actions; there is neither "incapacity" nor impairment in understanding of the intentions of action (Gernsbacher, 2007; Gernsbacher, Stevenson, Khandakar, & Goldsmith, 2008a, 2008b; Gernsbacher, et al. in press).

To provide a few examples, in Aldridge et al.'s (2000) study, prelinguistic autistic children "showed the expected deficits on [the conventional] imitation tasks but were *significantly better* than [pre-linguistic typically developing children on the intentionality" tasks (p. 294, emphasis added); in Hamilton et al.'s (2007) study, autistic grade-school-age children "performed *significantly better* than the control" children in "interpreting the meaning of gestures" (p. 1866, emphasis added); in Sebanz et al.'s (2005) study, using a complex spatial compatibility reaction time task, autistic adults were deemed "far from action blind," when they capably represented a coactor's task, showing the same pattern of results as the matched control group" (p. 433).

Not one of seven studies (Aldridge et al., 2000; Carpenter et al., 2001; Falck-Ytter, 2010; Hamilton et al., 2007; McAleer et al., 2010; Russell & Hill, 2001; Sebanz et al., 2005) that directly assess autistic individuals' understanding of the intentions of theirs and other persons' actions support VG's claim (in this issue and elsewhere) that autistic persons are incapable of such understanding or that autistic individuals have

"defective intentional attunement" (Gallese, 2006a, 2006b; Gallese, Eagle, & Migone, 2007). But these studies were not cited by VG. Instead, he cited only one study that directly assessed autistic individuals' understanding of action.

In that study (Boria et al., 2009), grade-school-age autistic and nonautistic children didn't differ when the task was to explain why a photographed hand was touching an object (e.g., "to touch" it), and the two participant groups didn't differ when the task was to explain why a hand was grasping an object in such a way as to use it (e.g., "to make a telephone call"). However, both groups performed significantly worse when the task was to explain why a hand was grasping an object not to use it but "to place" it—and the autistic children performed even worse than the nonautistic children. When cues such as containers in which to place the objects were shown, both groups improved significantly, and the two groups didn't differ.

Thus, these data from Boria et al. (2009) don't provide a very strong counterweight to the multiple other data sets that have repeatedly demonstrated that autistic individuals of all ages do not differ from nonautistic individuals in understanding the intentions of actions, contra to VG's proposal otherwise.

When answering the question of whether "abnormal" MN function contributes to the autistic phenotype, MI reports that in the neuroimaging literature, "there are 20 published papers that support the idea," and only "four studies [that] do not." However, MI's tally appears to be based on a rather incomplete survey of the existing literature.

fMRI: Imitation. MI identifies only eight fMRI studies relevant to the question of MN function and the autistic phenotype. Three of those studies, Dapretto et al. (2006), Martineau et al. (2010), and Williams et al. (2006), are imitation studies that I discussed in my initial response when I stated that neither Martineau et al. (2010) nor Williams et al. (2006) replicate Dapretto et al. (2006). Indeed, as illustrated in Table 2, the three autism-imitation fMRI studies MI cites not only fail to replicate each other, they fail to provide consistent evidence concerning the putative function of MNs during imitation.

fMRI/PET: Face processing. In addition to the three fMRI studies of imitation MI cites as evidence that "abnormal" MN function contributes to the autistic phenotype, he cites three fMRI studies of face/emotion processing (Bookheimer et al., 2008; Hadjikhani et al., 2007; Schulte-Rüther et al., 2010). However, these three studies that MI cites comprise less than 10% of the published autism-face-processing literature, and the results of the studies MI cites are unreflective of that larger literature.

As illustrated in Table 3, in contrast to Hadjikhani et al.'s (2007) report of less activation in autistic individuals' superior temporal sulcus, 36 other data sets report no differences between autistic and nonautistic participants in superior temporal activation, four data sets report more activation in autistic participants' superior temporal sulcus, and only a tiny fraction of the data sets—two—corroborate Hadjikhani et al.'s (2007) report of less activation for autistic participants in the putative MN region of the superior temporal sulcus. Similarly, while 12

Table 2. Summary of Activation Reported in Three Regions of the Putative Mirror Neuron System During Imitation Experiments

Study	Inferior frontal		Inferior parietal		Superior temporal	
	Imitation	Observation	Imitation	Observation	Imitation	Observation
Dapretto et al. (2006)	⇓	⇓	⇑	(=)	=	(=)
Williams et al. (2006)	(=)	(=)	⇓	=	⇑	(=)
Martineau et al. (2010)	(=)	⇑	(=)	=	(=)	(=)

Note. ⇓ = Autistic participants' activation significantly less than that of nonautistic participants; ⇑ = autistic participants' activation significantly greater than that of nonautistic participants; = = autistic participants' activation not significantly different from that of nonautistic participants; (=) = neither autistic nor nonautistic participants exhibit a reliable amount of activation.

data sets report less inferior frontal activation for autistic participants, three data sets report more, and the clear majority of the data sets (28) report no differences between autistic and nonautistic participants in the putative MN region of the inferior frontal cortex.

Structural MRI: Cortical thickness. MI cites two structural MRI studies as evidence that "abnormal" MN function contributes to the autistic phenotype. One of these studies measures cortical thickness (Hadjikhani et al., 2004) and reports that autistic participants exhibit thinner cortices in three regions of the putative MN system: inferior frontal, inferior parietal, and superior temporal. Presumably, the assumption is that thinner cortex means fewer MNs. But what about three other studies that MI does not cite (as illustrated in Table 4)? These three studies report that autistic participants have thicker cortices in regions of the putative MN system. Do the thicker cortices of autistic persons mean they have more MNs than nonautistic people? More likely, as Table 4 suggests and as reviewed recently by Stevenson and Kellett (2010, the whole set of cortical thickness studies are too inconsistent to allow drawing such conclusions.

Structural MRI: Grey matter density and volume. The other structural MRI study that MI cites is Yamasaki et al.'s (2010) region-of-interest-based morphometry study, which reports that autistic participants have smaller Broca's areas. However, as illustrated in Table 5, a study that MI does not cite (Knaus et al., 2009) reports that autistic participants have larger Broca's areas, and another study that MI does not cite (DeFosse et al. 2004) reports no difference between autistic and nonautistic participants in volume of Broca's area or its right-hemisphere equivalent. Virtually every voxel-based morphometry study has concluded that there is no difference between autistic and nonautistic participants in either left- or right-hemisphere pars triangularis or pars opercularis, as also illustrated in Table 5.

EEG: mu rhythm suppression. MI also cites several EEG studies, most particularly those that measure mu rhythm suppression, and claims that these studies illustrate "reduced mirroring in autism during action observation." I discussed these studies in my initial response when I noted that Oberman et al.'s (2005) original mu rhythm suppression study has not replicated (Bernier et al., 2007; Fan et al., 2010; Oberman et al., 2008; Raymaekers et al., 2009). Because MI cites some

of these studies, not as failures to replicate, which they are, but instead as evidence of "reduced mirroring in autism during action observation," let me quote directly from these studies.

Bernier et al. (2007) report: "Significant attenuation in mu from baseline was found for both groups [autistic and non-autistic] for each condition [observe, execute, and imitate]" (p. 232). There was "a main effect of condition . . . but no main effect for group or interaction effects." Oberman et al. (2008) report: "There was no significant main effect of . . . group." There was "a significant main effect of familiarity . . . [but] there was not a significant group by familiarity interaction" (p. 1562). Fan et al. (2010) report: "The mu suppression over the sensorimotor cortex was significantly affected by experimental conditions [observation of hand actions, observation of a moving dot, execution of hand actions], but not by group membership [autistic vs. non-autistic], nor by the interaction between groups and conditions" (p. 981). Raymaekers et al. (2009) report: "Both groups [autistic and non-autistic] show significant mu suppression to both self and observed hand movements. No group differences are found in either condition" (p. 113).

Conclusion. The nearly 70 studies listed in Table 5 argue against MI's assertion that only a few brain imaging studies fail to support the proposal of "mirror neuron abnormalities" in autistic persons. Rather, numerous studies—indeed, the bulk of existing brain imaging studies—fail to support that proposal. Given the extraordinary federal, private, and international funds spent on identifying the neural basis of the autistic phenotype and the large bodies of research those funds have generated, it is important to examine entire bodies of data, not selective pockets.

MI. In contrast to an implication of MAG's answer to Question 5, Ramachandran and colleagues never claimed that MNs did not evolve in people with autism. To make this suggestion misses the point: Reduced functionality does not mean lack of an evolutionary mechanism.

The claim made by MAG that "The vast majority of studies (90%) with typical participants did not report imitation-specific activity in BA44/45" is wrong when taken out of the context of that particular meta-analysis. Molenberghs et al. (2009) surveyed 129 papers but included only 16 (not 20, as incorrectly stated) in its final analysis. Less than 15% of surveyed papers were analyzed due to highly selective criteria that are not

Table 3. Summary of Activation Reported in Three Regions of the Putative Mirror Neuron System During Face Processing/Emotion Experiments

Study	Inferior frontal	Inferior parietal	Superior temporal
Hadjikhani et al. (2007)	⇓	(=)	⇓
Bookheimer, Wang, Scott, Sigman, & Dapretto (2008)	⇓	(=)	(=)
Schulte-Rüther et al. (2010; other emotions)	⇓	(=)	=
Schulte-Rüther et al. (2010; self emotions)	⇑	(=)	=
Ashwin, Baron-Cohen, Wheelwright, O'Riordan, & Bullmore (2007)	(=)	(=)	⇑
Bird, Catmur, Silani, Frith, & Frith (2006)	(=)	(=)	(=)
Bölte et al. (2006)	(=)	(=)	(=)
Corbett et al. (2009)	(=)	(=)	(=)
Critchley et al. (2000)	(=)	(=)	⇑
Dalton et al. (2005; study 1)	(=)	(=)	(=)
Dalton et al. (2005; study 2)	(=)	(=)	(=)
Dalton, Holsen, Abbeduto, & Davidson (2008)	(=)	(=)	(=)
Deeley et al. (2007)	(=)	(=)	(=)
Greimel et al. (2005; other faces)	(=)	(=)	(=)
Greimel et al. (2005; self face)	⇓	(=)	(=)
Grelotti et al. (2005)	⇓	(=)	(=)
Hadjikhani et al. (2004)	(=)	(=)	(=)
Hall, Szechtman, & Nahmias (2003)	⇓	(=)	(=)
Hubl et al. (2003)	(=)	(=)	(=)
Humphreys, Hasson, Avidan, Minshew, & Behrmann (2008)	(=)	(=)	⇓
Kleinhaus et al. (2008)	(=)	(=)	(=)
Kleinhaus et al. (2010)	⇓	(=)	(=)
Kleinhaus et al. (2011)	(=)	(=)	(=)
Koshino et al. (2008)	⇓	⇓	(=)
Loveland, Steinberg, Pearson, Mansour, & Reddoch (2008)	(=)	(=)	⇓
Ogai et al. (2003; disgust)	⇓	(=)	(=)
Ogai et al. (2003; fear)	(=)	(=)	(=)
Ogai et al. (2003; happiness)	(=)	(=)	(=)
Pelphrey, Morris, McCarthy, & LaBar (2007; dynamic)	(=)	(=)	(=)
Pelphrey et al. (2007; static)	(=)	(=)	⇑
Pierce, Müller, Ambrose, Allen, & Courchesne (2001)	(=)	(=)	(=)
Pierce & Redcay (2004)	(=)	(=)	(=)
Pierce & Redcay (2008)	⇓	(=)	(=)
Piggot et al. (2004)	(=)	(=)	(=)
Pinkham et al. (2008; trustworthiness)	⇓	(=)	(=)
Pinkham et al. (2008; age judgment)	⇑	(=)	⇑
Scherf et al. (2010; Non-autistic ROIs)	(=)	(=)	(=)
Scherf et al. (2010; autistic ROIs)	⇑	(=)	(=)
Schultz et al. (2000; sample 1)	(=)	(=)	(=)
Schultz et al. (2000; sample 2)	(=)	(=)	(=)
Uddin et al. (2008)	⇓	(=)	(=)
Wang et al. (2004; matching)	(=)	(=)	(=)
Wang et al. (2004; labeling)	(=)	(=)	(=)

Note. ⇓ = autistic participants' activation significantly less than that of nonautistic participants; ⇑ = autistic participants' activation significantly greater than that of nonautistic participants; = = autistic participants' activation not significantly different from that of nonautistic participants; (=) = neither autistic nor non-autistic participants exhibit a reliable amount of activation; ROIs = regions of interest.

representative of a large literature. A recent meta-analysis (Caspers et al., 2010) from two groups well known in the neuroimaging community for their strength in methodology includes 139 studies and shows that imitation "involved a caudo-dorsal part of BA44" (p. 1148). This finding replicates our own much smaller meta-analysis of seven studies performed in my lab (Molnar-Szakacs et al., 2005).

Williams et al. (2006) and Martineau et al. (2010) could not possibly replicate Dapretto et al. (2006) because the experiments differ. Nevertheless, they support Dapretto et al.'s conclusion of abnormal activity in the MNs in ASD. Martineau et al. (2010) write that their data "...provide strong support for the hypothesis of atypical activity of the MNs that may be at the core of the social deficits in autism" (p. 168). Williams et al. (2006) show reduced activity in the ASD group in the parietal sector of the MNs. Why did they not see differences in BA44? First, absence of evidence in one study cannot be considered evidence of absence, especially when many studies show positive results. Second, Williams et al., (2006) used a 1.5T scanner, whereas Dapretto et al. (2006) used a 3T scanner. Although there

Table 4. Summary of Cortical Thickness Reported in Three Regions of the Putative Mirror Neuron System

Study	Inferior frontal		Inferior parietal		Superior temporal	
	Left hemisphere	Right hemisphere	Left hemisphere	Right hemisphere	Left hemisphere	Right hemisphere
Hadjikhani et al. (2004)	⇓	⇓	⇓	⇓	=	⇓
Chung et al. (2005)	=	=	=	=	⇓	=
Ecker et al. (2010)	⇓	=	⇑	⇑	=	⇑
Hardan, Muddasani, Vemulapalli, Keshavan, & Minshew (2006)	=	=	⇑	⇑	⇑	⇑
Hutsler, Love, & Zhang (2006)	=	=	=	=	=	=
Hyde, Samson, Evans, & Mottron (2010)	=	⇑	=	=	⇑	⇑
Jiao et al. (2010)	⇓	⇓	⇓	⇓	=	=
Wallace et al. (2010)	=	=	⇓	⇓	=	=

Note. ⇓ = autistic participants' cortices significantly thinner than nonautistic participants'; ⇑ = autistic participants' cortices significantly thicker than nonautistic participants'; = = autistic participants' cortices not significantly different from nonautistic participants'.

Table 5. Summary of Volumetric and Grey Matter Density Reported in Inferior Frontal Gyrus

Study	Left pars triangularis	Right pars triangularis	Left pars opercularis	Right pars opercularis
	ROI-based morphometry			
Yamasaki et al. (2010)	⇓	⇓	⇓	⇓
DeFosse et al. (2004)	=	=	=	=
Knaus et al. (2009)	⇑	⇑	⇑	⇑
	Voxel-based morphometry			
Abell et al. (1999)	⇓	=	=	=
Craig et al. (2007)	=	=	=	=
Kosaka et al. (2010)	=	⇓	=	=
Kwon, Ow, Pedatella, Lotspeich, & Reiss (2004)	=	=	=	=
McAlonan et al. (2002)	=	=	=	=
McAlonan et al. (2005)	=	⇓	=	=
Rojas et al. (2006)	=	=	=	=
Toal et al. (2010)	=	=	=	=
Waiter et al. (2004)	=	=	=	=
L.B. Wilson et al. (2009)	=	=	=	=

Note. ROI = regions of interest; ⇓ = autistic participants exhibit significantly smaller volume (ROI-based)/less grey matter density (voxel-based morphology) than nonautistic participants; ⇑ = autistic participants exhibit significantly larger volume (ROI-based)/greater grey matter density (voxel-based morphology) than non-autistic participants; = = autistic and nonautistic participants exhibit significantly equivalent volume (ROI-based)/grey matter density (voxel-based morphology).

is a widespread loss of signal throughout the brain from 3T to 1.5T, the biggest loss in signal happens to be in a set of areas including inferior frontal cortex, where BA44 is located (Krasnow et al., 2003). Reduced signal equals reduced ability to see differences in signal. However, others have replicated Dapretto et al. (2006; see for instance Hadjikhani et al., 2007).

Treatments based on imitation show promising results (Ingersoll, 2010). I take this evidence to support of a role of MNs dysfunction in ASD. Furthermore, MAG's claim that the broken mirror hypothesis is based on "faulty empirical foundation and eroding empirical support" is wrong. Supporting studies vastly outnumber nonsupporting ones, with an approximate ratio of 5 to 1 (see my primary answer to this question). The nonsupporting studies either have insufficient power (for instance, Avikainen et al., 1999), are based on erroneous assumptions about brain physiology (for instance, Dinstein et al., 2010), or are based on questionable assumptions about brain–behavior relationships (for instance, Hamilton et al.,

2007) that led to uninterpretable results with regard to MN activity. A theory cannot be expected to account for all existing data, because not all studies are properly designed, analyzed, and reported. The broken mirror hypothesis is reminiscent of the case of prions, where a theory backed up by solid empirical data was lambasted because it violated dogmatic assumptions in medicine. The beauty of science is that eventually the mounting evidence prevails over biased opinions, as it happened with prions.

The best strategy to study autism is to connect the dots from different approaches. For instance, a recent study found that the gene RORA is down regulated by male hormones (Sarachana, Xu, Wu, & Hu, 2011), a finding that might explain why more men than women have autism. RORA is expressed less in autism and is important for the development of the cerebellum. The cerebellum connects with BA44 (Tamada, Miyauchi, Imamizu, Yoshioka, & Kawato, 1999) and is important for "feed-forward control" of action. In the presence of an overall

reduced long-range functional connectivity in autism (Kennedy & Courchesne, 2008), reduced expression of RORA may produce reduced cerebellar input to BA44, thereby affecting its functioning to the point that the mirror mechanism is inefficient.

Question 6: To What Extent Do Mirror Mechanisms Contribute to Social Cognition Such as Similarity Bias, Empathy, and Cultural Transmission?

Initial answers to Question 6

CH. I am going to focus on cultural transmission and look at the question both ways: how much do mirror mechanisms contribute to cultural transmission, and how much does cultural transmission contribute to mirror mechanisms? Any answers to these questions about cultural transmission are necessarily more speculative than claims about the relationship between mirror mechanisms and other social cognitive functions. We have fairly standard laboratory tests for similarity bias, empathy, imitation, speech perception, and language comprehension, but nothing resembling a standard test for cultural transmission. The term implies that a behavioral or cognitive trait is transmitted with a significant degree of fidelity through a series of social learning episodes—for example, from Person A to Person B, from B to C, and so on—and that this occurs under conditions comparable with those in which culture-specific attributes are acquired in the course of normal development. Thus, unlike other social cognitive functions, cultural transmission is something that occurs ''between heads'' rather than ''in heads,'' and it is very difficult to assess rigorously in a laboratory paradigm.

Cultural transmission depends on social learning, and imitation is one kind of social learning. Therefore, the evidence that mirror mechanisms can make a causal contribution to imitation (see Question 3) also constitutes evidence that mirror mechanisms can contribute to cultural transmission. However, two considerations put this evidence in perspective. First, imitation is one of many types of social learning—others include stimulus enhancement, observational conditioning, and emulation learning (Heyes, 2011b; Tomasello, 1996)—and, at least as yet, there is no evidence implicating mirror mechanisms in these other types of social learning. Second, there is reason to doubt that imitation plays a major role in the transmission of technological skills, which have preoccupied researchers interested in cultural evolution. The transmission of ''gestural skills,'' such as culture-specific dance movements, almost certainly requires imitation. However, it is likely that the most efficient way to learn to make a stone hand-axe, or to use a computer, is to copy the effects of actions on objects, rather than the way parts of an actor's body move relative to one another (Heyes, in press). Thus, current evidence suggests that mirror mechanisms are among those that contribute to cultural transmission but not that they play a dominant role.

Cultural processes may play a more significant role in the formation of mirror mechanisms than mirror mechanisms play in supporting cultural transmission. This intriguing possibility is raised by the evidence that MNs are forged by sensorimotor learning (see Question 4). This evidence supports the view that MNs are produced by phylogenetically ancient processes of associative learning—the same processes that produce conditioning phenomena. When, in the course of normal development, the individual experiences a contingency between observation and execution of the same action (e.g., when grasping is more often accompanied by the sight of grasping than by the sight of any other act), associative learning strengthens connections between visual neurons in the superior temporal sulcus and motor neurons in classical mirror areas that code the same actions. These connections give the motor neurons mirror properties—they are activated by observation, as well as performance, of specific actions (see Fig. 1; Heyes, 2010).

Cultural processes enter the picture when we consider the circumstances in which people (and some laboratory monkeys) experience a contingency between observation and execution of the same action. This happens during direct self-observation—when we watch our own actions—but it also occurs when we watch our movements in an optical mirror; engage in synchronous behavior to a musical beat; and, perhaps most important, when infants are being imitated by caregivers. Indeed, it is only from these sociocultural sources that we could get the contingency experience necessary to build mirror mechanisms for facial expressions and whole body movements—actions that look very different when observed and executed.

Thus, the associative account suggests that the development of MNs is fostered by cultural artifacts (optical mirrors) and practices (music and dancing), as well as by direct self-observation, and that part of the developmental process is a relatively simple transmission chain: Person A transmits a certain repertoire of MNs to Person B by imitating a certain set of B's actions in infancy, and B transmits a similar repertoire to C by imitating the same set of C's actions in infancy. If this is correct, mirror mechanisms are not at one end of a causal arrow between biology and culture—they owe at least as much as they lend to cultural processes.

VG. The mainstream view in cognitive science was, and to a certain extent still is, that action, perception, and cognition are to be considered as separate domains. The discovery of the MN mechanism shows that these domains are intimately intertwined. In virtue of the translation of others' bodily movements into something that the observer is able to grasp as being part of a given motor act accomplished with a given motor intention, the observer is immediately tuned with the witnessed motor behavior of others. This enables the observer to understand others' motor goals and motor intentions in terms of her/his own motor goals and motor intentions (Gallese et al., 1996; see also Rizzolatti & Sinigaglia, 2010).

The proposed functional relevance of the mirror mechanism in action understanding does not imply that the same mechanism is completely opaque to the issue of agentive self-reference. It has been recently shown that the intensity of the discharge of F5 MNs is significantly stronger during action

execution than during action observation (Rochat et al., 2010). This means that the mirror mechanism also likely contributes to agents' implicit sense of being the owners of their actions. In other words, there is a primitive bodily self-awareness that is before and below any reflective self-awareness and before any explicit sense of agency and sense of ownership (Gallese & Sinigaglia, 2010). On this account, the motor system provides both the common ground for understanding others and the criteria for distinguishing between self and other bodily awareness.

Shepherd, Klein, Deaner, and Platt (2009) discovered a class of neurons in the posterior parietal area LIP, involved in oculomotor control, that fired both when the monkey looked in a given direction and when it observed another monkey looking in the same direction. Shepherd et al. suggested that LIP MNs for gaze might contribute to sharing of observed attention, thus playing a role in imitative behavior.

Furthermore, in a recent experiment, Rochat, Serra, Fadiga, and Gallese (2008) showed that macaque monkeys, similarly to 9- to 12-month-old human infants, detect the goal of an observed motor act and, according to the physical characteristics of the context, construe expectancies about the most likely action the agent will execute in a given context. This, however, only occurs when observed motor acts are consonant to the observer's motor repertoire. The relevance of MNs for monkeys' social cognition is also evident from the study by Caggiano, Fogassi, Rizzolatti, Their, and Casile (2009), showing that the distance at which the observed action takes place modulates the discharge of F5 MNs. Such modulation, however, doesn't simply measure the physical distance between agent and observer. A consistent percentage of MNs not responding to the experimenter's grasping actions carried out within monkeys' peri-personal space resume their discharge when a transparent barrier is blocking the observing monkey's potentiality for action. This shows the relevance of MNs when mapping the potentialities for competition or cooperation between agent and observer. All of these results show that macaque monkeys are endowed with social cognitive abilities that only a few years ago were considered to be absent even in apes. Furthermore, they suggest that these cognitive abilities can be coherently explained at the neurophysiological level by the motor resonance mechanism instantiated by MNs.

In humans, the same logic also applies to the domain of emotions and sensations. Brain imaging evidence shows that whenever we witness the emotions or sensations experienced by others, some of our brain regions display mirror activation. The same sector of the anterior insula activated by our own first-person experience of disgust is also activated when we see the facial expression of disgust displayed by another individual (Wicker et al., 2003). Similarly, the same somatosensory-related cortical regions activated when one of our body parts is touched are also activated when observing tactile stimuli applied to the body parts of someone else (Blakemore, Bristow, Bird, Frith, & Ward, 2005; Ebisch et al., 2008; Keysers et al., 2004). Such sharing, however, is only partial. Other cortical regions are exclusively activated for one's own emotion and not for others' emotion (Jabbi, Bastiaansen, & Keysers, 2008), or are activated for one's own tactile sensation, but are actually deactivated when observing the same sensation experienced by someone else (Ebisch et al., 2010).

I proposed that mirroring could be a basic functional principle of our brain (Gallese, 2001) and that our capacity to empathize with others might be mediated by embodied simulation mechanisms, that is, by the activation of the same neural circuits underpinning our own agentive, emotional, and sensory experiences (see Gallese, 2005, 2006a, 2009; Gallese, Keysers, & Rizzolatti, 2004). Following this perspective, empathy is to be conceived as the outcome of our natural tendency to experience our interpersonal relations first and foremost at the implicit level of intercorporeity—that is, the mutual resonance of intentionally meaningful sensory-motor behaviors.

MI. Current theories of empathy suggest a multilayer functional structure, with a core layer of automatic responses to reproduce the affective state of others (Preston & de Waal, 2002) interacting with higher-level functional layers. MNs are likely cellular candidates for the core layer of empathy. They can make us empathize with others in two possible ways. MNs may simulate the observed action and then send signals to emotional brain centers like the amygdala to evoke neural activity that make us feel what others feel (Iacoboni, 2008a). Alternatively, MN activity may suffice to evoke the feeling.

The pIFG and the amygdala are active during observation, and even more so during imitation, of facial expressions (Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003), and the activity in these areas correlates with empathy (Pfeifer, Iacoboni, Mazziotta, & Dapretto, 2008). Because both areas are active, and because of what we know about their functional properties, these findings suggest that MNs may simply simulate the facial expression, whereas the amygdala evokes the emotion. Other studies, however, show that pIFG activity correlates with empathy even when subjects watch grasping actions without overt emotional content (Kaplan & Iacoboni, 2006). Thus, the MN activity is a prerequisite for experiencing empathy (in the sense of understanding others) even when there is no overt emotional content.

Many recent studies have demonstrated correlations between markers of neural mirroring and empathy and also with measures of social competence (Cheng et al., 2009; Cheng et al., 2008; Fecteau, Pascual-Leone, & Théoret, 2008; Hooker, Verosky, Germine, Knight, & D'Esposito, 2010; Lepage et al., 2010; Pfeifer et al., 2008; Zaki, Weber, Bolger, & Ochsner, 2009). Thus, neural mirroring in humans potentially represents a bio-marker of sociality.

The emerging field of cultural neuroscience is also exploring the role of neural mirroring in cultural transmission and similarity bias (Losin, Dapretto, & Iacoboni, 2009). A TMS study (Molnar-Szakacs, Wu, Robles, & Iacoboni, 2007) found greater neural mirroring when European Americans observed an ethnic ingroup member versus an ethnic outgroup member performing hand gestures, suggesting a positive relationship between MNs activity and model–observer similarity. Surprisingly, another TMS study found greater neural

mirroring when individuals viewed ethnic outgroup members (Désy & Théoret, 2007) and an MEG study found greater neural mirroring for gender outgroup members (Cheng, Tzeng, Decety, Imada, & Hsieh, 2006). These conflicting findings may result from complex interactions between many factors that are invoked by experimental designs in cultural neuroscience studies. Methodological guidelines for this new field that will likely emerge in the near future potentially will clarify the role of neural mirroring in similarity bias and cultural transmission (Losin, Dapretto, & Iacoboni, 2010).

Reply to Question 6

CH. The answers to Question 6, and to Questions 1–3 and 5, confirm that authoritative figures in the field believe that MNs play a fundamental role in a range of social cognitive functions. In answering Question 4—the only one about the origins, rather than the effects, of MNs—I surveyed evidence that MNs are forged by sensorimotor learning. Responding to Question 6, I pointed out that much of this sensorimotor learning occurs in a sociocultural context. It is tempting to assume that, if this associative account is correct, MNs could not make significant contributions to social cognition. I think that would be a mistake. If MNs are a byproduct of associative learning, in a system that evolved for motor control rather than social cognition, they're unlikely to underwrite any particular function, but they could still contribute to social behavior in a variety of ways. Personally, I'll be delighted if plausibility arguments give way to solid evidence that MNs have major effects on social cognition. Evidence of this kind would indicate the power not only of MNs, but of the associative learning and sociocultural processes that put them in our brains.

Declaration of Conflicting Interests

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