

To What Extent is the Experience of Empathy Mediated by Shared Neural Circuits?

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Abstract

This paper selectively reviews the neurophysiological evidence for shared neural circuits (supposedly implemented by mirror neurons) as the mechanism underlying empathy. I will argue that while the mirror neuron system plays a role in motor resonance, it is not possible to conclude that this system is critically involved in emotion recognition, and there is little evidence for its role in empathy and sympathy. In addition, there is modest support from neurological observations that lesion of the regions involved in the mirror neuron system leads to dysfunction in empathy, whereas damage of the ventromedial prefrontal cortex is associated with such impairment. To significantly advance our understanding of the mechanisms underlying empathy, research needs finer conceptualization, better designed paradigms, and integration with knowledge from lesion studies.

Keywords

empathy, lesion studies, mirror neuron system, shared neural circuits, social emotions, sympathy

Empathy and sympathy play crucial roles in much of human social interaction and are necessary components for healthy coexistence. Sympathy is thought to be a proxy for motivating prosocial behavior, guiding our preferences and behavioral responses, and providing the affective and motivational base for moral development (Eisenberg & Eggum, 2009). Here, I distinguish between empathy (the ability to appreciate the emotions and feelings of others with a minimal distinction between self and other) and sympathy (feelings of concern about the welfare of others). While empathy and sympathy are often conflated, the two can be dissociated, and although sympathy may stem from the apprehension about another's emotional state, it does not have to be congruent with the affective state of the other. Unfortunately, most work in affective neuroscience ignores, or at best overlooks, the complexity of the empathy construct and equates sensorimotor resonance with empathy and sympathy. This is a problem because the former does not convey insight into another's internal state, nor it can account for any other-oriented motivational state that characterizes empathic concern. Understanding how neural processes instantiate the experience

of empathy provides an avenue of future directions for emotion perception in the economy of social interaction.

An increasing number of studies on empathy (and social cognition in general) have made considerable reference to the "mirror neuron system" (MNS) following the paper by Preston and de Waal (2002) in which the authors argued for a perception-action model of empathy. According to that model, the perception of the target's state automatically activates the observer's representations of that state, and activation of these representations automatically generates the associated autonomic and somatic responses, unless inhibited. This model suggests that perception of emotion activates in the observer the neural mechanisms that are responsible for the generation of similar emotion. At first glance, this direct matching hypothesis fits neatly with the simulation model of emotion processing, which proposes that our ability to understand the intentions and emotions expressed by others relies on internally simulating the same psychological state in ourselves. Here I critically examine the contribution of mirror neurons to empathy by first briefly reviewing the properties and localization of these neurons in the monkey brain, and then discussing the evidence for the MNS in

humans during the perception and execution of action, examining their contribution to emotion processing in general and to empathy in particular. Because neuroimaging data are merely correlational, lesion studies are critical to complement our knowledge about the functions implemented in these regions, and thus to clarify their causal role in the processes studied. I conclude that while the MNS plays a role in motor resonance, it is not at present possible to conclude that it is critically involved in emotion recognition, and there is little evidence for its role in empathy and sympathy.

What Are Mirror Neurons?

Mirror neurons are a unique class of cells with sensorimotor properties that were first identified in the monkey ventral premotor cortex area F5. These neurons respond both when the monkey executes a particular action—for example, grasping, placing or manipulating—and when the monkey observes someone else performing that same action (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996). Neurons with similar visuo-motor properties were discovered in the anterior intraparietal area (Fogassi et al., 2005) and in the primary motor cortex (Tkach, Reimer, & Hatsopoulos, 2007). Many functions have been attributed to mirror neurons, including action understanding, imitation, empathy, and even mindreading (Gallese, Keysers, & Rizzolatti, 2004). However, such claims have been lessened by sharp criticism and new studies that lead to alternative explanations. With the relatively recent discovery of such cells in the primary motor cortex, mirror neurons may be best interpreted as nothing more than motor system facilitators, acting via learned associations (Hickok, 2009). It was recently argued, based on a fine conceptual analysis of empirical research on mirror neurons and their putative contribution to theory of mind, that motor resonance is neither a necessary nor a sufficient mechanism for representing another individual's intentions, especially in a social context (Jacob, 2008).

The Mirror Neuron System in Humans

Evidence for the existence of mirror neurons in humans is indirect, and principally relies on functional neuroimaging studies that demonstrate an overlap in activation between observation and action conditions in regions homologous to the areas of the monkey brain where mirror neurons have been found. These regions include the inferior frontal gyrus, the ventral premotor cortex, and anterior and posterior intraparietal sulcus (e.g., Dinstein, Hasson, Rubin, & Heeger, 2007). Transcranial magnetic stimulation studies reported changes in the excitability of the observer's motor and premotor cortices that encode the execution of observed actions (Fadiga, Fogassi, Pavesi, & Rizzolatti, 1995). Magnetoencephalography measurements have also demonstrated suppression in the *mu* rhythm (8–13 Hz) over the sensorimotor cortex during the observation of action which parallels the changes detected during action production (Cheng, Lee, Yang, Lin, & Decety, 2008). The findings of these studies strongly support the role of the MNS in motor resonance.

In the context of emotion processing, it is posited that perception of an emotion in another individual activates in the

observer the neural mechanisms that are responsible for the generation of similar emotion. It is outside the scope of this essay to assess the validity of the assumption that there are distinct categories of emotions underpinned by specific neural circuits (see Barrett, 2006), or whether it would really be adaptive for instance in response to the perception of someone threatening, to simulate the threatening movements rather than flee as quickly as possible. Nonetheless, there is evidence that observing facial expressions of emotion elicits facial electromyographic (EMG) responses in the observer. Sonnby-Borgstrom, Jonsson, and Svensson (2003) examined the relationship between facial mimicry (measured by facial EMG) and self-reported mood upon exposure to static facial expressions of anger and happiness in participants who were categorized as either high or low empathizers, and found that the high-empathy participants produced greater facial mimicry than the low-empathy participants. However, another study did not find any relation between emotion recognition performance and participants' tendency to mimic dynamic displays of emotions (Hess & Blairy, 2001).

The idea that the MNS is implicated in emotion perception mainly relies on studies that reported activation in the inferior frontal gyrus during the observation and the imitation of facial expression of emotions (happiness, sadness, anger, disgust and surprise) (e.g., Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003). One such study used a paradigm in which subjects observed and imitated hand and face actions (Leslie, Johnson-Frey, & Grafton, 2004) and found that right ventral premotor cortex was commonly activated during observation and imitation of facial expressions. Another study demonstrated that even passive viewing of facial expressions activates a network of regions that were also involved in the execution of similar expressions, including the inferior frontal gyrus (IFG) and the posterior parietal cortex (van der Gaag, Minderaa, & Keysers, 2007). In contrast, only the perception of happy expressions was associated with an activation of the left IFG and not with expressions of sadness, anger and disgust in a study conducted by Chakrabarti, Bullmore, and Baron-Cohen (2006). However, the majority of functional neuroimaging studies have not reported activation of the MNS during the perception of facial expression of emotion (see Murphy, Nimmo-Smith, & Lawrence, 2003 for a meta-analysis). More disturbing and worth mentioning is that several studies claiming to have found MNS activation did not have the appropriate experimental conditions to support such a claim (Turella, Pierno, Tubaldi, & Castiello, 2009). Unfortunately, this circular interpretation has been taken to such an extreme that many studies interpret any hemodynamic response in the IFG and anterior intra parietal sulcus as being due to mirror neuron activity, as if the regions only consisted of mirror neurons, and thus both grossly ignoring that mirror neurons in the monkey account for only a small minority of the cell population. Furthermore, areas involved in the MNS itself subserve different types of computations (not solely sensorimotor integration), and in the case of the IFG, cognitive control, attention, and task management (e.g., Swick, Ashley, & Turken, 2008). A recent meta-analysis of 20 functional magnetic resonance imaging studies of imitation reported that in the frontal lobe, the dorsal premotor cortex rather than the inferior frontal gyrus is consistently active, and in the parietal region, the superior and inferior parietal lobules are equally activated during imitation

(Molenberghs, Cunnington, & Mattingley, 2009). These results seriously question the crucial role of the MNS during imitation, and demonstrate the importance of conducting meta-analyses to look beyond the idiosyncrasies of individual experiments.

A more indirect reference to the MNS in empathy, which became popular during the past years, relies on the interpretation of any overlap in activation between the experience of an emotional state and the observation of the same state in another individual as mirror activity, or shared neural circuits (Decety & Meyer, 2008). For this argument to hold, the activated clusters need not lie in the areas that belong to the MNS. For instance, in the domain of empathy for pain, a striking overlap has been demonstrated in regions underpinning the first-hand experience of pain and its perception in others, or even its imagination (Jackson, Rainville, & Decety, 2006). These findings lend credence to the idea that empathy for pain draws upon automatic sensorimotor resonance between other and self, by which the observer simulates (and shares) the pain of the other. It is important to mention, however, that the activation of these regions (anterior insula, anterior midcingulate cortex, supplementary motor area and periaqueductal gray) may simply reflect a general aversive response coupled with motor preparation for defensive actions, which may not be specific to nociception (Yamada & Decety, 2009). In addition, and against the simulation account of emotion recognition, a study of patients suffering from congenital insensitivity to pain found pertinent results. In this rare syndrome, patients cannot rely on mirror matching mechanisms to understand the pain of others, as they never experience pain. Despite never having had the personal experience of pain they showed similar hemodynamic responses to observed pain as control subjects in anterior midcingulate cortex and anterior insula, two key regions of the so-called “shared circuits” for self and other’s pain (Danziger, Faillenot, & Peyron, 2008). Thus, while the shared neural representation account of empathy for pain is appealing, one could suggest another and perhaps more parsimonious interpretation within the fundamental biological properties such as valence, arousal or approach and withdrawal (Barrett & Wager, 2006). Such an interpretation fits well with the absence of insula and anterior cingulate cortex activation when physicians are exposed to body parts being pricked by a needle, a situation not aversive to them (Cheng et al., 2007).

The Importance of Lesion Studies

While neuroimaging data are merely correlational, studies of neurological patients are critical to determine the causal role of a given region, and give the functional weight of that region to the cognitive process studied. While there is some support for the notion of shared neural substrates between emotion experience and emotion recognition mainly with respect to the involvement of the somatosensory cortex, neuropsychological observations do not support such a role (Heberlein & Atkinson, 2009). For instance, Keillor, Barrett, Crucian, Kortenkamp, and Heilman (2002) reported the case of a patient suffering from a bilateral facial paralysis who was unable to convey emotions through facial expressions. Despite her complete facial paralysis, the person did not show deficits in the experience of emotion

or the recognition of facial expressions. Similarly, patients with Moebius syndrome, who suffer from bilateral facial and usually complete paralysis, have difficulty communicating with facial expression, but are not impaired at recognizing facial expressions of emotions in others (Calder, Keane, Cole, Campbell, & Young, 2000; Rives Bogart, 2010).

So far, neuropsychological studies have documented a critical role of the medial and orbitofrontal cortex, closely interconnected with the amygdala, in social emotions, including empathy and sympathy (e.g., Hornak et al., 2003; Shamay-Tsoory, 2009). Impairment of the medial/cingulate prefrontal cortex is commonly associated with deficits in social interaction and self-conscious emotions (Sturm, Rosen, Allison, Miller, & Levenson, 2006). Such patients may become apathetic, disinterested in the environment, and unable to concentrate their attention on behavioral and cognitive tasks. It has also been suggested that frontal damage hinders perspective-taking ability, a crucial component of empathic concern (Price, Daffner, Stowe, & Mesulam, 1990). The study of degenerative neurological diseases further supplies evidence for relatively distinct neural routes to social cognition and empathy deficits. For instance, both patients with a frontotemporal dementia (FTD), a predominantly neocortical disorder associated with deficits in frontal executive functions, as well as patients with Huntington’s disease (HD), a predominantly subcortical disorder characterized by involuntary movements, present difficulties in tasks of social cognition (Snowden et al., 2003). However, the two patient groups display qualitatively different patterns of results, suggesting that the deficits of patients with a FTD may be attributed to a breakdown in theory of mind while those of patients with HD disease appear to be associated with faulty inferences drawn from social situations. Interestingly, both patients with HD and FTD demonstrate a lack of empathy, but for different reasons. In the former group, the loss of empathy arises more at an emotional than a cognitive level, while patients with a FTD live in an egocentric world in which they do not ascribe independent mental states to others. These findings are consistent with the idea of distinct and interrelated neural systems underpinning the experience of empathy (Decety & Jackson, 2004).

Conclusions

To sum up, while the MNS provides a physiological mechanism for motor resonance and plays a role in mimicry, current neurophysiological and neurological evidence does not clearly support the idea that such a mechanism accounts for emotion understanding, empathy or sympathy. Future research in affective neuroscience needs to refine conceptually what processes are encompassed in the experimental designs and at what level (e.g., implicit vs. explicit). This is critical, because empathy belongs to self-conscious emotions, which rely on complicated and distributed brain networks including the posterior superior temporal sulcus, amygdala, insula, medial and ventral prefrontal cortices. Usually, neuroimaging studies report activation associated with empathy regardless of whether the tasks implicate conscious imitation of facial expressions, implicit recognition of distress cues in others. Further, they generally confuse having emotions, understanding of the reasons they occurred, emotional

reactivity, and emotional appraisal. Functional and effective connectivity analyses are necessary to move beyond the simplistic view of single brain areas (e.g., IFG or insula) as being the neural basis of complex constructs such as empathy, and meta-analyses are crucial to accumulate consensus across tasks that involve putatively similar processes while washing out statistical idiosyncrasies in individual studies. Finally, patient studies can shed light on the processes involved in empathy, and enrich our understanding of brain behavior functions.

References

- Barrett, L. F. (2006). Emotions as natural kinds? *Perspectives on Psychological Science*, 1, 28–58.
- Barrett, L. F., & Wager, T. D. (2006). The structure of emotion. *Current Directions in Psychological Science*, 15, 79–83.
- Calder, A. J., Keane, J., Cole, J., Campbell, R., & Young, A. W. (2000). Facial expression recognition by people with möbius syndrome. *Cognitive Neuropsychology*, 17, 73–87.
- Carr, L., Iacoboni, M., Dubeau, M. C., Mazziotta, J. C., & Lenzi, G. L. (2003). Neural mechanisms of empathy in humans: A relay from neural systems for imitation to limbic areas. *Proceedings of National Academy of Sciences USA*, 100, 5497–5502.
- Chakrabarti, B., Bullmore, E., & Baron-Cohen, S. (2006). Empathizing with basic emotions: Common and discrete neural substrates. *Social Neuroscience*, 1, 364–384.
- Cheng, Y., Lee, P., Yang, C. Y., Lin, C. P., & Decety, J. (2008). Gender differences in the *mu* rhythm of the human mirror-neuron system. *PLoS ONE*, 5. Retrieved 16 March, 2010, from [http://www.cell.com/neuron/abstract/S0896-6273\(08\)01016-7](http://www.cell.com/neuron/abstract/S0896-6273(08)01016-7).
- Cheng, Y., Lin, C., Liu, H. L., Hsu, Y., Lim, K., Hung, D., & Decety, J. (2007). Expertise modulates the perception of pain in others. *Current Biology*, 17, 1708–1713.
- Danziger, N., Faillenot, I., & Peyron, R. (2008). Can we share a pain we never felt? Neural correlates of empathy in patients with congenital insensitivity to pain. *Neuron*, 61, 203–212.
- Decety, J., & Jackson, P. L. (2004). The functional architecture of human empathy. *Behavioral and Cognitive Neuroscience Reviews*, 3, 71–100.
- Decety, J., & Meyer, M. (2008). From emotion resonance to empathic understanding: A social developmental neuroscience account. *Development and Psychopathology*, 20, 1053–1080.
- Dinstein, H., Hasson, U., Rubin, N., & Heeger, D. J. (2007). Brain areas selective for both observed and executed movements. *Journal of Neurophysiology*, 98, 1415–1427.
- Eisenberg, N., & Eggum, N. D. (2009). Empathic responding: Sympathy and personal distress. In J. Decety & W. Ickes (Eds.), *The social neuroscience of empathy* (pp. 71–83). Cambridge, MA: MIT Press.
- Fadiga, L., Fogassi, L., Pavesi, G., & Rizzolatti, G. (1995). Motor facilitation during action observation: A magnetic stimulation study. *Journal of Neurophysiology*, 73, 2608–2611.
- Fogassi, L., Ferrari, P. F., Gesierich, B., Rozzi, S., Chersi, F., & Rizzolatti, G. (2005). Parietal lobe: From action organization to intention understanding. *Science*, 308, 662–667.
- Gallese, V., Fadiga, L., Fogassi, L., & Rizzolatti, G. (1996). Action recognition in the premotor cortex. *Brain*, 119, 593–609.
- Gallese, V., Keysers, C., & Rizzolatti, G. (2004). A unifying view of the basis of social cognition. *Trends in Cognitive Science*, 8, 396–403.
- Heberlein, A. S., & Atkinson, A. P. (2009). Neuroscientific evidence for simulation and shared substrates in emotion recognition: Beyond faces. *Emotion Review*, 1(2), 162–177.
- Hess, U., & Blairy, S. (2001). Facial mimicry and emotional contagion to dynamic emotional facial expressions and their influence on decoding accuracy. *International Journal of Psychophysiology*, 40, 129–141.
- Hickok, G. (2009). Eight problems for the mirror neuron theory of action understanding in monkeys and human. *Journal of Cognitive Neuroscience*, 7, 1229–1243.
- Hornak, J., Bramham, J., Rolls, E. T., Morris, R. J., O'Doherty, J. O., Bullock, P. R., & Polkey, C. E. (2003). Changes in emotion after circumscribed surgical lesions of the orbitofrontal and cingulate cortices. *Brain*, 126, 1691–1712.
- Jackson, P. L., Rainville, P., & Decety, J. (2006). To what extent do we share the pain of others? Insight from the neural bases of pain empathy. *Pain*, 125, 5–9.
- Jacob, P. (2008). What do mirror neurons contribute to human social cognition? *Mind and Language*, 23, 190–223.
- Keillor, J. M., Barrett, A. M., Crucian, G. P., Kortenkamp, S., & Heilman, K. M. (2002). Emotional experience and perception in the absence of facial feedback. *Journal of the International Neuropsychological Society*, 8, 130–135.
- Leslie, K. R., Johnson-Frey, S. H., & Grafton, S. T. (2004). Functional imaging of face and hand imitation: Towards a motor theory of empathy. *NeuroImage*, 21, 601–607.
- Molenberghs, P., Cunnington, P., & Mattingley, J. B. (2009). Is the mirror neuron system involved in imitation? A short review and meta-analysis. *Neuroscience and Biobehavioral Reviews*, 33, 975–980.
- Murphy, F. C., Nimmo-Smith, I., & Lawrence, A. D. (2003). Functional neuroanatomy of emotions: A meta-analysis. *Cognitive, Affective, & Behavioral Neuroscience*, 3, 207–233.
- Preston, S. D., & de Waal, F. B. M. (2002). Empathy: Its ultimate and proximate bases. *Behavioral and Brain Sciences*, 25, 1–72.
- Price, B. H., Daffner, K. R., Stowe, R. M., & Mesulam, M. M. (1990). The compartmental learning-disabilities of early frontal lobe damage. *Brain*, 113, 1383–1393.
- Rives Bogart, K. (2010). Facial mimicry is not necessary to recognize emotion: Facial expression recognition by people with Moebius syndrome. *Social Neuroscience*. DOI:10.1080/17470910903395692.
- Shamay-Tsoory, S. (2009). Empathic processing: Its cognitive and affective dimensions and neuroanatomical basis. In J. Decety & W. Ickes (Eds.), *The social neuroscience of empathy* (pp. 215–232). Cambridge, MA: MIT Press.
- Snowden, J. S., Gibbons, Z. C., Blackshaw, A., Doubleday, E., Thompson, J., Craufurd, D., et al. (2003). Social cognition in frontotemporal dementia and Huntington's disease. *Neuropsychologia*, 41, 688–701.
- Sonnby-Borgstrom, M., Jonsson, P., & Svensson, O. (2003). Emotional empathy as related to mimicry reactions at different levels of information processing. *Journal of Nonverbal Behavior*, 27, 3–23.
- Sturm, V. E., Rosen, H. J., Allison, S., Miller, B. L., & Levenson, R. W. (2006). Self-conscious emotions deficits in frontotemporal lobar degeneration. *Brain*, 129, 2508–2516.
- Swick, D., Ashley, V., & Turken, A. U. (2008). Left inferior frontal gyrus is critical for response inhibition. *BMC Neuroscience*, 9. Retrieved 16 March, 2010, from <http://www.biomedcentral.com/1471-2202/9/102>.
- Tkach, D., Reimer, J., & Hatsopoulos, N. G. (2007). Congruent activity during action and action observation in motor cortex. *Journal of Neuroscience*, 27, 13,241–13,250.
- Turella, L., Pierno, A. C., Tubaldi, F., & Castiello, U. (2009). Mirror neurons in humans: Consisting or confounding evidence? *Brain and Language*, 108, 10–20.
- van der Gaag, C., Minderaa, R. B., & Keysers, C. (2007). Facial expressions: What the mirror neuron system can and cannot tell us. *Social Neuroscience*, 2, 179–222.
- Yamada, M., & Decety, J. (2009). Unconscious affective processing and empathy: An investigation of subliminal priming on the detection of painful facial expressions. *Pain*, 143, 71–75.