Chapter 9

The Sensorimotor Side of Empathy for Pain

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Introduction

This chapter revolves around the phenomena and neural mechanisms underlying the human capability to empathize with the actions, emotions, and feelings of other individuals. Special attention is paid to the neural activity induced by observation and imagination of others' pain. It will be shown that representing others' pain brings about the activation of neural structures largely overlapping with those activated during the experience of pain on oneself and that neural structures involved in both emotional and sensorimotor processing may be recruited during empathy for pain.

Pain is an unpleasant subjective, sensory, and emotional experience associated with actual or potential tissue damage [1– 4]; and has a protective function related to the implementation of escape reactions. Thus, it is closely linked to the motor system [2, 5]. Sensory-discriminative components (e.g., evaluation of locus, duration, and intensity of a noxious stimulus) and affective-motivational components (e.g., unpleasantness of the noxious stimulus) contribute to the experience of pain [1–3]. Sensory and emotional components are represented in separate nodes of a complex neural network referred to as the "pain matrix" [6–9]. While the neural processing of pain perception has been widely studied, much less is known about the neural underpinnings of empathy for pain.

The term "empathy" is the English translation of the German word *Einfühlung*. This term was introduced into the psychology of aesthetics by Lipps [10] to indicate the experiences of individuals while contemplating pieces of artwork. The extension of this concept to the domain of intersubjectivity supported the notion that empathy is inherently linked to an inner imitation process [10].

Empathy plays a fundamental social role insofar as it allows the interindividual sharing of experiences, beliefs, aims, and inner states. It is widely held that empathy plays an important role in psychoanalysis and psychoanalytic therapy [11–13]. For instance, in *Jokes and Their Relation to the Unconscious* [14], Freud used this concept (influenced by the work of Lipps, which he profoundly admired) to designate the process of putting oneself into another's position, either consciously or unconsciously. According to Kohut [15, 16], empathy allows the therapist to understand what is going on inside the patient's mental life in an "experience-near" way. This would imply that the therapist had to place himself/herself into the mental life of the patient through a process of "vicarious introspection" [15]. A basic aspect of empathy in the psychoanalytic tradition appears thus to be related to the experiential understanding of others' mental states. This view of empathy seems tightly related to the accounts of empathy developed in the philosophy of mind, psychology, and neuroscience, and based on the notion of "simulation." According to simulation theories (ST) we understand others' behavior and mental states by putting ourselves in the "mental shoes" of others and thus covertly replicating their inner states in our own mind [17–27]. According to the neuroscientific formulation of this notion, empathy implies that perceptual, motor, or emotional states of a given individual activate the corresponding neural representations in another individual who observes that state [24–27].

Neural Representation of Physical Pain

Pain is a complex and enigmatic feeling that typically signals actual or potential tissue damage. The experience of pain can be described along two main phenomenological axes: (i) the sensory-discriminative dimension, comprising spatial, temporal, and intensity properties of pain; and (ii) the affective-motivational dimension, related to the unpleasantness of the stimulus, as well as the behavioral and autonomic reactions it evokes [1–3].

Consistent with the multidimensional view of pain, neuroimaging techniques (such as positron emission tomography, PET; and functional magnetic resonance, fMRI) demonstrate that a complex neural network, referred to as the "pain matrix," is involved in the experience of pain [6–9]. Sensory and affective components of pain are mapped in two major separate nodes of the pain matrix, namely the sensorimotor and the affective

node. The sensorimotor node of the pain matrix includes the primary (S1) and secondary (S2) somatosensory cortices (and also sensorimotor structures, such as cerebellum, premotor, and motor areas). Animal studies demonstrate that somatosensory cortices receive noxious and innocuous somatosensory input from the somatosensory thalamus [2–30] and contain nociceptive neurons that code key features of the sensory-discriminative dimension of stimulus processing such as spatial, temporal, and intensive aspects of innocuous and noxious somatosensory stimuli [30–34]. Accordingly, neuroimaging studies in humans indicate that S1 and S2 process sensory features of pain and display a somatotopical organization [6, 8, 35–39]. Lesions to these areas may induce deficits of pain sensation in brain-damaged patients [40, 41]. For example, Ploner et al. [41] observed that a patient who had suffered a stroke that encompassed S1 and S2 did not experience a painful sensation when a hot laser stimulus was applied to the affected arm, indicating that intact somatosensory cortices are necessary for the normal experience of pain sensation. However, the patient reported an ill-localized and ill-defined unpleasant feeling in the absence of a clear pain sensation, suggesting that pain affect was present in the absence of pain sensation.

The affective node of the pain matrix includes at least the anterior cingulate cortex (ACC) and the insular cortex (IC) [42–50], which are phylogenetically old regions and are considered to be components of the classical limbic system [51] and of MacLean's "visceral brain" [52].

In primates, ACC receives input from medial thalamic nuclei that contain nociceptive neurons, including nucleus parafascicularis and the ventrocaudal part of nucleus medialis dorsalis [53, 54]. Direct pain input to the ACC is further suggested by the observations that painful stimuli evoke potentials over the human anterior cingulate gyrus and that single nociceptive neurons are present in the ACC of humans [55, 56], monkeys [57], and rabbits [58]. Neuroimaging studies have emphasized the role of the ACC in the perceived unpleasantness of physical pain [6–8]. Rainville et al. [49] used hypnotic suggestion to modulate the perception of unpleasantness during noxious stimulations. When the experimental subjects were influenced to perceive the noxious stimulations as highly unpleasant there was a concomitant increase in the activity in the ACC compared with when the subjects were influenced to perceive the same stimulation as less unpleasant [49]. However, the activity in the somatosensory areas was unaltered. In a similar vein, studies further indicate that increasing levels of ACC activity correspond with increasing levels of self-reported pain unpleasantness and distress [59, 60]. Thus, indi-

viduals who are dispositionally pain-sensitive show more ACC activity and report greater levels of perceived distress to painful stimulation [61].

The IC also receives direct thalamocortical nociceptive input in the primate [54], and has been implicated in autonomic regulation [62, 63]. The implication of IC in the subjective experience of pain is consistent with a function of the IC in higher-order processes relevant to homeostatic regulation [54, 64] and awareness of internal bodily processes [65]. Lesions to the IC may produce a clinical condition called "asymbolia for pain," or "Schilder–Stengel syndrome," in which patients show deficits in the affective-motivational component of pain but preserve their sensory discrimination. Such patients perceive painful stimuli but do not display the appropriate emotional responses to painful stimulation [66]. Neuroimaging studies indicate that the affective dimension of pain is mainly encoded in the anterior sector of the IC (anterior insula, AI) [6–8].

Beyond Nociception

Various physical painful experiences, ranging from being pricked with a pin to feeling an aching phantom pain [67], are represented in the different nodes of the pain matrix. However, pain does not have only a physical dimension related to tissue injury. Pain is also conceived of as a universal human experience that is commonly generalized to psychic suffering of any sort [54].

Interestingly, numerous languages characterize "social pain" (the pain resulting from social injury, e.g., in cases where social relationships are threatened, damaged, or lost) using words typically reserved for describing physical pain ("broken heart," "broken bones"). Animal lesion and human neuroimaging studies indicate that the neural circuitry and the computational processes underlying physical and social pain largely overlap [68, 69]. Interestingly, a recent fMRI study in humans demonstrates that the same sectors of the ACC that are involved in the perception of painful stimuli are also activated during the experience of social loss [70].[1]

[1] In this fMRI study of social exclusion, participants were scanned while playing a virtual ball-tossing game from which they were ultimately excluded. Paralleling results from physical pain studies, the anterior cingulate cortex (ACC) was more active during exclusion than during inclusion, and correlated positively with self-reported distress [70].

Another indication of pain-related neural activity in the absence of physical noxious stimulations comes from a recent fMRI study in which Japanese participants listened to Japanese pain-evoking onomatopoeic words and nonsense syllables [71]. Listening to these sounds induced an increase in the fMRI BOLD signal in ACC, suggesting an activation of affective pain representation (see also Chapter 10 in this volume).

Although pain has been described as an essentially private subjective experience by some philosophers [72, 73], neuroscience studies support the view that pain processing has a fundamental social dimension that may extend to basic levels of neural processing.

Studies on empathy for pain further support and expand this view. In the following paragraphs we show that both affective and sensorimotor representations of the pain supposedly felt by a model are mapped in the observer's neural circuitry dedicated to processing the pain felt by oneself. We also discuss the important role of the motor system in the personal experience of pain and in some aspects of social cognition. These notions may allow us to construct a neuroscientifically based concept of empathy.

Pain and the Motor System

Pain is closely linked to action systems that can be considered as the division of the pain matrix (part of the sensorimotor node) involved in the implementation of appropriate reactions to actual or potential noxious stimuli. Nociceptive stimuli can elicit a series of defensive or reactive responses, such as withdrawal reflexes, avoidance behaviors, and emotional-motor reactions [2, 74, 75]. Moreover, chronic pain affects motor control by limiting and impairing not only actual movements [76] but also their covert mental simulation [77, 78]. Furthermore, electrical [79–81] or magnetic stimulation [82] of the primary motor cortex (M1) in patients suffering from intractable chronic pain (e.g., phantom pain) can attenuate their symptoms; in addition, pain severity in amputees increases with the shrinkage of the limb representation in M1 [67, 83]. Although the physiological basis of these phenomena is largely unknown, they hint at bidirectional influences between pain and motor systems; thus, specific activity in the motor systems influences activity in nociceptive systems and is influenced by it. Accordingly, some neuroimaging studies have shown that changes of metabolic activity in M1 and other motor-related

structures can be induced by the delivery [8, 50, 84, 85] or even anticipation [86, 87] of painful stimuli. More reliable results have been observed by means of a neurophysiological assessment, such as transcranial magnetic stimulation (TMS).[2] TMS studies in humans show that a strong reduction in the excitability of corticospinal motor systems occurs in association with different types of nociceptive stimulation [88–91]. This motor inhibition is likely to represent the electrophysiological correlate of a defensive, withdrawal reflex.

All these studies demonstrate the important link between pain and motor systems. In the section entitled "The sensorimotor side of empathy for pain" we will show that this link may also occur at a social level. But first we will focus on the role of the motor system in social cognition and empathy.

Motor System and Mirror Neurons

Recent research in nonhuman and human primates has pointed at the role of motor systems in higher-order cognitive processes [92–94]. Particularly relevant to the present discussion is the discovery in the mon-

[2] Transcranial magnetic stimulation (TMS) is a powerful, noninvasive neurophysiological technique based on Faraday's principles of electromagnetic induction. A brief pulse of current flowing through a coil of wire generates a magnetic field. If the magnitude of this magnetic field changes over time, then it will induce a secondary current in any nearby conductor. For brain stimulation, a pulse is produced in a coil held over a subject's head. As a brief pulse of current is passed through it, a magnetic field is generated that passes through the subject's scalp and skull with negligible attenuation. This time-varying magnetic field induces an electric current in the subject's brain, causing depolarization of cellular membranes and thereby neuronal activation. In many experiments, single pulses of TMS are applied over the motor cortex. The stimulation of the motor cortex is able to transsynaptically activate the corticospinal system and to produce a response in controlateral extremity muscles, the motor-evoked potential (MEP), which can be recorded by means of electrodes. The amplitude of MEPs is used as a measure of corticospinal excitability. The amplitude of these potentials is modulated by the behavioral context. Thus, the modulation of the amplitude of MEPs can be used to assess the central effects of various experimental manipulations. This approach has been used in basic neuroscience to study the effect of actual nociceptive stimulation on corticospinal excitability [5], and in cognitive neuroscience to study the modulation of the motor system during the observation of painful events delivered to others [133] or during the observation of actions performed by others [98, 99].

key premotor and parietal cortices of a particular population of visuomotor bimodal cells called "mirror neurons." The most remarkable functional characteristic of these neurons is the increase in their firing rate both when the monkey performs an action, and when he observes a similar action made by another human or monkey agent [95–97].

Evidence in support of a motor mirror system (MMS) in humans comes from a single-pulse TMS study showing that the mere observation of a given movement brings about a specific increase in amplitude of MEPs recorded from the muscles that would be recruited during actual execution of the observed movements [98, 99].

The link between perception and execution of actions is further supported by behavioral studies showing that execution of a given action is positively or negatively modulated by observation of the same or a different action [100, 101]. Importantly, neuroimaging and neurophysiological studies in humans indicate that frontoparietal structures known to be involved in action execution become active during action observation [102–111]. Moreover, the observation of actions made with different effectors activates different regions of premotor and parietal areas, thus suggesting that the MMS may be organized according to somatotopic rules [112]. These studies suggest that humans have a MMS similar to that originally discovered in monkeys. When we observe an action performed by others, our motor system becomes active *as if* we were executing the very same action. This covert mimicking can be conceived as an inner simulation of the action. It has been proposed that simulating others' actions may be crucial for action understanding [21– 23, 25, 26, 93, 113–118]. Indeed these inner motor simulations lead to shared states between self and others and may allow us to directly understand the meaning of others' action without any explicit reflective mediation [23, 26]. Thus, MMS might constitute a basic system for coding and understanding observed actions which can be of fundamental importance, not only for motor learning and imitation but also for other social aspects of cognition, such as an understanding of others' intentions and beliefs [97, 119–121].

From Mirror Neurons to Empathy

There is a rapidly growing neuroscientific literature supporting the idea that we understand other people's behavior and thoughts, in part, by putting ourselves in the "mental shoes" of others [21–26]. Indeed, several

authors have proposed that the simulative processes originally discovered and described in the domain of actions could constitute a basic characteristic of our social brain and of our ability to understand and empathize with others [23–26].

Empathy is the ability to have a direct experiential understanding of others' feelings and inner states [22, 23, 26]. Empathy is deeply grounded in the experience of our living body [122], and it is this experience that enables us to directly recognize others as *persons* like us [23, 123]. As a body-owner, we can easily grasp, through a process of sharing, the meaning of actions, sensations, or emotions displayed by others [23].

Current neuroscientific models of empathy postulate that a given motor, perceptual, or emotional state of an individual activates corresponding representations and neural processes in another individual observing that state [22–24, 27]. Thus, a basic mechanism in the empathic experience may involve the transformation of third-person visual information about others into first-person, bodily representations [27, 124, 125].

This feature may be a basic attribute of sophisticated forms of empathy and may be neurally implemented through several types of mirror simulative mechanisms [22, 23]. In keeping with this notion, a number of brain systems with mirror properties have also been recently described in the domain of emotion [126–129] and sensory processing [50, 125, 130–134]. Thus, the MMS may be only one of the systems of our social brain that allow us to have a direct experiential knowledge about others.

Evidence supporting the idea of mirror activity in a purely sensory domain has been provided by two recent studies on "empathy for touch" [130, 131]. Blakemore et al. [131], for example, found common S1 activations with a topographic organization during the personal experience of touching stimuli delivered to the face or the neck and the observation of such stimulations in others.

According to ST, there is evidence to suggest that perception of emotion automatically activates mechanisms that are responsible for the generation of emotion [25]. For instance, viewing facial expressions triggers expressions on one's own face (as measured by electromyography), even in the absence of conscious recognition of the stimulus [135, 136]. Moreover, fMRI studies indicate that similar networks of motor and emotional brain areas are activated by the perception of emotional expressions and the overt imitation of similar emotions [126, 128, 137]. Lesion studies indicate that right frontoparietal cortex is necessary for the correct recognition of emotions from prosody [25, 138].

Somatosensory-related structures are also crucial for emotion recognition [139–142]. These areas may become active in tasks involving facial expression judgment [141]. Importantly, lesions to somatosensory areas in brain-damaged patients [25, 139], or interference with the activity of these structures obtained by means of magnetic brain stimulation [140], impairs facial emotion recognition. According to ST, during the recognition of another's emotion, specific sensorimotor structures could provide a somatic description of the experience derived from actually feeling the same emotion. This may help us to learn about others' emotional states [25, 140, 142].

Some direct evidence of a sharing of emotional representations between self and others comes from studies on the emotion of disgust. Calder et al. [143] reported the case of the patient N.K., with left IC and putamen damage, who was selectively impaired in detecting social signals of disgust from facial expressions, nonverbal sounds, and emotional prosody. Interestingly, this perceptual deficit for disgust expressions was mirrored by an equivalent deficit in the phenomenological experience of the same emotion. Indeed, patient N.K. was less disgusted than controls by disgust-provoking scenes. The involvement of insula in the recognition of disgust has also been supported in healthy subjects using fMRI [144]. Consistent with these findings is the fMRI study showing that viewing another person's facial emotional reactions to unpleasant odorants activates sectors of the AI and ACC that are also activated when the subject himself inhales the same unpleasant odorants [127].

Empathic Mirroring of Others' Pain

As mentioned in the Introduction, various painful personal experiences are represented in a complex neural network referred to as the "pain matrix" [6, 7, 8, 9]. Affectively distressing components (such as unpleasantness) and sensory components (such as localization and intensity) of painful stimuli are encoded in the affective and sensorimotor node of the pain matrix, respectively. The presence of distinct sensory and affective components makes pain a particularly interesting model for testing simulative theories of empathy based on the notion of shared neural representations. Undeniably, the empathic sharing of pain representations may hold a special status in the domain of empathic processes. On the one hand, sharing affective representations of pain (distress, unpleasantness)

may represent most directly a predicate for sophisticated forms of empathy, i.e., helping or altruistic behavior and ethical and moral reasoning [75, 122, 125]. On the other hand, sharing sensorimotor representations of pain may imply that others' pain is mapped onto one's own body. This may be crucial for social learning of protective behaviors and defense reactions to potentially damaging situations [133, 145].

In this section we present findings that support the notion of shared neural representations between self and others in the domain of pain processing. According to current neuroscientific theories of empathy [22- 27], the basic simulative mirror mechanisms described in the domain of action, touch, and emotion may also be at the root of our ability to understand and empathize with the pain of others. The possibility that the human ability to recognize the pain of others is grounded in a mirror-like pain system is suggested by the anecdotal report of a patient suffering from an unusual form of allodynia (a pathological condition in which nonnoxious touching stimuli are perceived as painful) [145]. This patient apparently experienced observed pain as pain in himself. For example, when the patient's wife experienced a sudden minor injury (e.g., knocking her hand against a table), he would become very agitated, claiming that it hurt him to witness such accidents. If she merely commented that she had knocked her fingers, there was no such reaction. Although the report was anecdotal and no information about the neural circuitry involved in this type of phenomena was provided, the results have been attributed to an abnormal "mirror pain" system [145]. More direct evidence of "pain mirror neurons" comes from neurophysiology data on pain-related processing in human neurosurgical patients [55]. Using microelectrodes, Hutchison et al. [55] found several nociceptive neurons in the ACC, including cells that discharged preferentially to mechanical noxious stimuli. In that study it was noticed that a neuron responded selectively to the anticipation and delivery of noxious mechanical stimulation (pinching, pinpricks) applied to the patient's hand. Interestingly, this cell also responded during the observation of the experimenter receiving pinpricks in the hand [55].

Recent fMRI studies show that only affective components of the pain matrix are crucial for empathy for pain, thus suggesting that only emotional representations of pain are shared between self and others [50, 125, 132, 134]. In a first fMRI study by Singer et al. [50], empathy for pain was induced by means of arbitrary visual cues signaling an impending painful stimulus to the participant's romantic partner. Empathy for pain brought about an increase of fMRI signal in AI and ACC cortices, which are part of

the affective division of the pain matrix. Importantly, neural activity correlated with the subjects' emotional empathy traits scores.

Neural activity in the affective pain network was also reported in fMRI studies involving unknown human models, where subjects observed pictures [132] or movies [125] in which potentially painful stimuli were delivered to hands or other human body parts, or movies depicting facial expressions of pain [134].

Despite some activations in structures that may be involved in somatic processing, such as the thalamus, brainstem, parietal cortex, and cerebellum, found in studies when participants imaged others' pain [50], watched facial pain-related behavior [134], or observed potentially painful situations [132], the authors concluded that only the affective division of the pain matrix is crucial for empathy for pain.

The Sensorimotor Side of Empathy for Pain

The ability to understand and to experience indirectly the pain of others may be fundamental to social cohesion [24, 50, 75]. Previous fMRI studies on empathy for pain indicate that perceiving pain in others mainly involves cerebral regions known to play an important role in the affective experience of pain [50, 125, 132, 134]. This "affective resonance" may be at the base of complex forms of empathy [50], e.g., emotional concern, piety, and altruistic behavior.[3]

Do we share with others only emotional representations of pain? From a developmental and evolutionary perspective, having a detailed repre-

[3] The study of Singer et al. [50] may reflect more directly the activity of such sophisticated forms of empathy. In this study neural activity in ACC and AI correlated positively with two personality questionnaires assessing the subjects' emotional empathy trait (e.g., the tendency to experience feelings of concern and piety in response to others in distress, and the tendency to desire to help others). In the other fMRI studies involving unknown human models [125, 132, 134], activity in the affective division of the pain matrix may imply a more simple mechanism of mirroring others' unpleasant emotional state. For instance, in the study by Jackson et al. [132], neural activity in ACC correlated with the level of the pain ascribed to the model, but not with the same personality questionnaire adopted by Singer et al. [50]. Interestingly, both high and basic levels of empathic emotional mechanism are mapped in the same emotional neural structures (ACC and AI).

sentation of the source and nature of others' pain may be crucial for survival. Some authors have speculated that, during infancy, avoidance of noxious stimuli may be facilitated by early recognition of others' pain [145]. We posit that mirror mechanisms that map detailed sensory representation of others' pain (e.g., locus and intensity of a noxious stimulus) onto one's own body may be fundamental for the social learning of escape or avoidance reactions to noxious stimuli.

Why has previous fMRI on empathy for pain failed to find specific somatic activations? The simplest explanation[4] is that previous studies may have adopted nonbiological relevant visual stimuli for evoking pain body-mapping, such as static pictures of potentially painful situations [132], very superficial injections in the hands [125], or stimuli in which the body was not directly shown [50, 134].

One may speculate that body mapping of others' pain may occur especially when the visual scene is of functional relevance for the individual (e.g., when the stimuli are shocking or very intense). Thus, in a recent study we presented subjects with "flesh and bone" painful stimulations shown on the body of a human model [133]. We used TMS[5] to record changes in corticospinal motor representations of the hand muscles of individuals observing needles penetrating the hands or feet of a human model or noncorporeal objects (Fig. 1a). Videos depicting static hands and feet and Q-tips touching the same body parts were used as control stimuli. We found a reduction in amplitude of MEPs that was specific to the hand muscle that subjects observed being deeply pricked (Fig. 1b). No inhibition of the muscles of the hand was found during observation of harmless touching stimuli or needles in feet or noncorporeal objects. Importantly, this motor inhibition was clearly related to the observer's subjective empathetic rating of the sensory, but not affective, qualities of the pain ascribed to the model. In other words, the largest motor inhibition was found in the participants who evaluated as most intense the model's pain [133] (Fig. 1c).

We interpreted the observational pain-related motor inhibition as reflecting the activity of a simulative mirror mechanism that extracts

[4] For an alternative hypothesis see Singer and Frith [149].

[5] As reported above, previous TMS studies indicate that actual nociceptive stimulations bring about a decrease in excitability of the corticospinal motor system [5, 88, 89, 90, 91]. That is, pain reduces the amplitude of the MEP induced by TMS. This motor inhibition may represent the corticospinal correlate of a defensive withdrawal reaction.

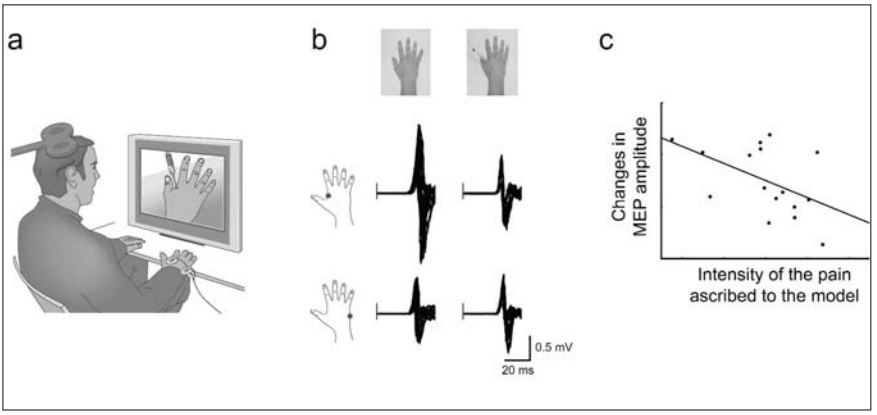

Fig. 1. a A magnetic pulse was delivered over the left primary motor cortex during the observation of different types of video-clips. Motor-evoked potentials (*MEPs*) induced by transcranial magnetic stimulation (*TMS*) were recorded from the first dorsal interosseus (*FDI*, in the region of the index finger) and the abductor digiti minimi (in the region of the little finger) of the observer's right hand. **b** Examples of MEPs recorded from the FDI (*top*) and the ADM (*bottom*) muscles during the observation of the model's static hand (*right*) and of a needle deeply penetrating the FDI region of the model's hand. Note the specific reduction of amplitude of MEPs recorded from FDI muscle during the observation of a syringe penetrating the model's FDI region. **c** Amplitude changes of MEPs recorded from the FDI during the observation of pain with respect to the static hand correlated with the intensity of the pain ascribed to the model

basic sensory qualities of the model's painful experience (location and intensity of the noxious stimulus) and maps them onto the observer's motor system according to topographical rules [133].[6] This hypothesis was strongly supported by the muscle specificity and by the link between MEP inhibition and the intensity of the pain attributed to the model.

Motor responses to one's own pain allow freezing or escape reactions, and ultimately survival. The observational pain-related motor inhibition indicates that similar motor responses occur as a result of observation of "flesh and bone" painful events in others [133].

It is worth noting that neuroimaging studies indicate that anticipation

6 Alternative interpretations, such as the shift of attention to the target body part, or the predictive motor imitation of the model's behaviour, were not likely based on the proprieties of the neurophysiological results (inhibition rather than facilitation, and muscle-specificity). A discussion of these alternative hypotheses is provided by Avenanti et al. [133].

of painful stimuli being administered to one's own body increases the hemodynamic signal in several regions of the pain matrix [46, 87, 146, 147]. These activations triggered by pain anticipation may also include somatotopical organized spots in the primary sensorimotor cortices (M1, S1) [86, 148]. According to shared representation models [24, 27], it is possible that the simulative mirror responses triggered by the observation of "flesh and bone" painful stimulations in others may reflect anticipation of pain in oneself [132, 133, 149]. The selective embodiment of others' pain in the observer's corticospinal system, sensitively more than emotionally denoted, may thus be crucial for the social learning of reactions to painful stimuli in that it may help the observer's corticospinal system to implement specific escape or freezing reactions before painful stimuli are actually experienced [133].

By means of neurophysiological techniques such as somatosensory-evoked potentials (SEPs) and laser-evoked potentials (LEPs) (that allow direct testing of activity within S1 and S2, respectively), we recently found support to the idea of pain sensorimotor representation sharing [150, 151]. In those studies, we found that some brain potentials evoked by somatosensory [151] and nociceptive stimulations [150] that originated from somatosensory cortices were selectively modulated by the observation of "flesh and bone" painful stimulations in others. In keeping with our TMS study, such potentials were highly linked with sensory, but not affective, components of pain. Preliminary fMRI data from our laboratories indicate that premotor and multisensory parietal structures may participate in such a mapping of sensory components of others' pain.

Conclusions

In humans, empathy for pain may rely not only on affective-motivational [50, 125, 132, 134] but also on fine-grained somatic representations [133]. This supports the notion that empathy is based on different types of sensory, motor, and emotional simulative mechanisms [22–24, 27].

It may be possible to think of at least two forms of empathy linked to one another in an evolutionary and developmental perspective. A comparatively simple form of empathy, based on somatic resonance, may be primarily concerned with mapping external stimuli onto one's own body [133]. This mapping may be important for learning of reactions to pain [133, 145]. A more complex form of empathy, based on affective reso-

nance, may deal with emotional sharing [50, 125, 132, 134] and with the evaluation of social bonds and interpersonal relations [50].

All in all, studies on empathy for pain indicate that the affective and sensorimotor divisions of the pain matrix are important nodes in the complex neural network recruited not only during the personal experience of pain [5–9, 50, 84–91] but also during empathy for others' pain [50, 125, 132–134].

A direct matching of specific sensory aspects of others' pain occurs in sensorimotor structures of the pain matrix [133], whereas emotional components of others' painful experiences (along with other-oriented compassion feelings) are coded in the affective division of the network [50, 125, 132, 134]. Such a sensorimotor and affective mapping of others' pain components may allow us to have a direct experiential empathic understanding of others' painful experiences that does not necessitate any explicit reflective mediation. In order to understand others, we use our internal body representations.

Hence, empathy for pain may take different forms in different nodes of the complex neural network that represent sensations, feelings, and emotions linked to the experience of pain. Philosophers have emphasized that our bodily sensations are intrinsically private [72, 73]. However, cognitive neuroscience suggests that, at least in humans, the social dimension of pain extends even to the very basic, sensorimotor levels of neural processing.

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