# Dissecting the Neural Mechanisms Mediating Empathy

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### Abstract

Empathy is thought to play a key role in motivating prosocial behavior, guiding our preferences and behavioral responses, and providing the affective and motivational base for moral development. While these abilities have traditionally been examined using behavioral methods, recent work in evolutionary biology, developmental and cognitive neuroscience has begun to shed light on the neural circuitry that instantiate them. The purpose of this article is to critically examine the current knowledge in the field of affective neuroscience and provide an integrative and comprehensive view of the computational mechanisms that underlie empathy. This framework is of general interest and relevance for theory as well as for assisting future research in the domains of affective developmental neuroscience and psychopathology.

#### Keywords

affective neuroscience, amygdala, empathy, orbitofrontal cortex, shared neural circuits, sympathy, theory of mind

Suppose you are on a plane during a long red-eye flight. Just seated next to you a father is holding his 6-month-old baby, and while you're trying to relax and get some sleep, the baby is screaming almost continuously. What your reaction would be? You may be compassionate or concerned for the baby girl and her dad, upset and annoyed because you cannot sleep, or maybe angry at yourself because you're aware of being irritated and you are thinking that babies do not belong on airplanes. If only you had noise-canceling headphones! You may benefit by appraising the situation, taking the perspective of that baby, imagining what is like to have earache from changes in cabin pressure, and down-regulating your negative and aversive feelings towards that baby, or imagining if you were in the shoes of her father, how concerned you would be for the baby and embarrassed for troubling other passengers. Would your reaction be different if you were a parent of a baby? This example illustrates the complexity of the emotions and the range of reactions one may experience when exposed to another's distress depending on various intrapersonal (e.g., moods, goals, dispositions) and situational factors. It also shows that our ability to appreciate and understand the emotions of others does not automatically lead to prosocial behavior, caring and concern, and does not necessarily ensure the benevolence that characterizes

the lay concept of empathy. Empathy is something that needs to be regulated. People who never show empathy as well as people who are too sensitive to the feelings and thoughts of others cannot be socially adapted.

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). Empathy is not unique to humans as many of the biological mechanisms are shared with other mammalian species, including the processes involved in intergroup relations that modulate its expression. However, humans are special in the sense that high-level cognitive abilities such as executive function, language and theory of mind are layered on top of phylogenetically older social and emotional capacities (Stone, 2006). These evolutionarily newer aspects of information processing expand the range of behaviors that can be driven by empathy for the best (like caring for and helping outgroup members or even individuals from different species) but also for the worst (such as cruelty and dehumanization). Furthermore, various psychopathologies are marked by empathy deficits. For instance, a key feature that distinguishes

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psychopaths from other criminals is their marked lack of concern for their victims, referred to as a lack of empathy, guilt or remorse. Thus a better knowledge of the neural circuits that instantiate the experience of empathy will not only advance our understanding of interpersonal sensitivity but also shed light onto the basic neural and cognitive mechanisms of emotion processing, their relation with cognition and motivation (i.e., empathic concern), individual differences in personality traits, and mental health.

The goal of this article is to critically examine our current knowledge about the neurophysiological underpinnings of empathy in humans. After clarifying some definitional issues of empathy and associated phenomena, and arguing that the construct of empathy needs to be decomposed in a model that includes bottom-up processing of affective communication and top-down processing in which the perceiver's motivation, intentions, and self-regulation influence the extent of an empathic experience, I will discuss how empathy develops and what are its evolutionary origins focusing on the biology of autonomic, endocrine, and other homeostatic processes functions of the autonomic nervous system that have developed to support the needs of mammalian communication and selective sociality. Next, I will critically review the empirical evidence that supports the notion of shared neural circuits for the generation of behavior, including emotions in oneself and their perception from others. I emphasize recent functional neuroimaging studies of pain empathy showing a partial overlap in the neural circuits underlying the first-hand experience of pain and the observation of pain in others, and how some interpersonal variables moderate empathy and sympathetic concern. Lesion studies are critical to complement our knowledge about the functions implemented in the regions found to be involved in empathy, and will thus be briefly reviewed. Finally, I conclude that the current data are compatible with the core affect model of emotion.

#### **Empathy and Associated Phenomena**

The term empathy is applied to various phenomena which cover a broad spectrum, ranging from feelings of concern for other people that create a motivation to help them, experiencing emotions that match another individual's emotions, knowing what the other is thinking or feeling, to blurring the line between self and other (Hodges & Klein, 2001). These phenomena are related to one another, but they are not elements, aspects or facets of a single thing that is empathy, as one might say that an attitude has cognitive, affective, and behavioral components (Batson, 2009). Given this variety of phenomena, it is not surprising that philosophers and psychologists have long debated the nature of empathy, and whether the capacity to share, appreciate and respond to other people's emotions sets humans apart from other species (de Waal & Thompson, 2005). In developmental psychology and social psychology (the two academic disciplines that have produced most of the research on the subject), empathy is generally defined as an affective response stemming from the understanding of another's emotional state or condition similar to what the other person is feeling or would be expected to feel in the given situation (Eisenberg, Shea, Carlo, & Knight, 1991). Other theorists more narrowly characterize empathy as one specific set of congruent emotions, those feelings that are more other-focused than self-focused (Batson, Fultz, & Schoenrade, 1987). Very often, empathy and sympathy are conflated. 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 confused, the two can be dissociated, and although sympathy may stem from the apprehension of another's emotional state, it does not have to be congruent with the affective state of the other. The experience of empathy can lead to sympathy (which includes an other-oriented motivation), or personal distress, an egoistic motivation to reduce stress by withdrawing from the stressor, thereby decreasing the likelihood of prosocial behavior. Another valuable definition of empathy-because it is based on processes-comes from psychoanalysis, in which empathy consists of two acts: (1) an identification with the other person; and (2) an awareness of one's own feelings after the identification, and in this way an awareness of the object's feeling (Fenichel, 1945).

# The Components of Empathy

Given the complexity of what the phenomenological experience empathy encompasses, investigation of its neurobiological underpinnings would be worthless without breaking down this construct into component processes. In spite of reports in the popular press that give the appealing, yet wrong notion, that the organization of psychological phenomena maps in a 1:1 fashion into the organization of the underlying neural substrate, in reality empathy, like other social cognitive processes, draws on a large array of brain structures and systems which are not limited to the cortex, but also include the autonomic nervous system (ANS), hypothalamic-pituitary-adrenal axis (HPA), and endocrine systems that regulate bodily states, emotion and reactivity.

Several scholars have argued that empathy includes both cognitive and affective components (Eisenberg & Eggum, 2009; Goubert, Craig, & Buysse, 2009; Hodges & Wegner, 1997). Based on theories and data from cognitive neuroscience, behavioral neurology and developmental psychology, Decety and colleagues (Decety, 2005, 2007; Decety & Jackson, 2004; Decety & Meyer, 2008; Decety & Moriguchi, 2007) proposed a model that includes bottom-up processing of affective sharing and top-down processing in which the perceiver's motivation, intentions, and self-regulation influence the extent of an empathic experience, and the likelihood of prosocial behavior. Under that working model, a number of distinct and interacting components contribute to the experience of empathy: (1) affective arousal (or sharing), a bottom-up process grounded in perception-action coupling in which the amygdala, hypothalamus and orbitofrontal cortex play a critical role; (2) emotion awareness and understanding which involves the AIC, mPFC, and vmPFC; and (3) emotion regulation which depends on executive

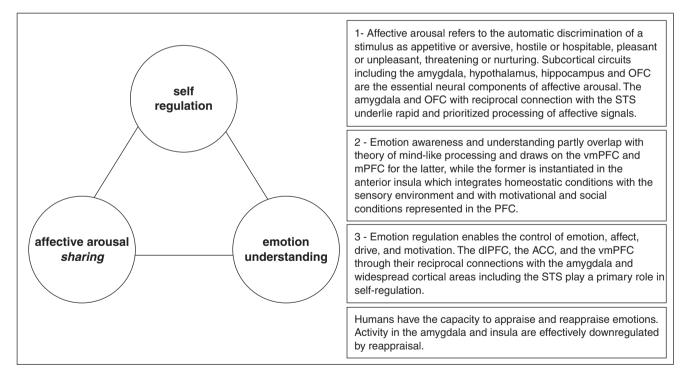
functions instantiated in the intrinsic cortico-cortical connections of the OFC, mPFC and dlPFC. These networks operate as top-down mediators, crucial in regulating emotions and thereby enhancing flexible and appropriate responses (Figure 1; for expansions of all abbreviations used in this article, see Appendix). While this model is helpful and has exploratory and predictive values, it remains to be functionally connected with other biological systems that are implicated in interpersonal sensitivity, such as the ANS and endocrine systems, in order to fully account for a comprehensive model of the experience of empathy. For instance, oxytocin is a putative mediator of empathy, especially if the behavioral reactions involve immobilization without fear, whereas vasopressin might be implicated in situations where a more active strategy is required for an affective response (Carter, Harris, & Porges, 2009).

#### The Neurodevelopment of Empathy

The focus of studying subcomponents of more complex behaviors is also particularly useful from a developmental perspective, when it is the case that only some components of, or precursors to, more complex behaviors are observable. In addition, developmental studies can provide unique opportunities to see how the components of the system interact in ways not possible in

adults, where all the components are fully mature and operational (De Haan & Gunnar, 2009). Behavioral manifestations of empathy occur very early during development. For instance, 6-month-old infants show a preference for characters that help others over characters that are not cooperative or are hindering (Hamlin, Wynn, & Bloom, 2007). Children aged 18-25 month's inclination to sympathize with others in strife has been demonstrated even in the absence of overt emotion cues (Vaish, Carpenter, & Tomasello, 2009), which suggests some early form of affective perspective-taking that does not rely on emotion contagion or mimicry. There is also compelling evidence that prosocial behaviors, such as altruistic helping, emerge early in childhood. Infants as young as 12 months of age begin to comfort victims of distress, and children aged 14-18 months display spontaneous and unrewarded helping behaviors (Warneken & Tomasello, 2009).

The affective component of empathy develops earlier than the cognitive and regulatory aspects. Affective responsiveness is known to be present at an early age, is involuntary, and relies on mimicry and somato-sensorimotor resonance between other and self. For instance, newborns and infants become vigorously distressed shortly after another infant begins to cry (Dondi, Simion, & Caltran, 1999; Martin & Clark, 1987). Facial mimicry of basic emotional expressions also contributes to affective



**Figure 1.** Component processes and neural architecture underpinning the experience of empathy. Empathy is a molar construct developed by behavioral and social scientists which, like other concepts used in social cognition, provides a means of understanding highly-complex activity without needing to specify each individual action by its simplest components, and thereby providing an efficient approach to describing complex systems. From this model, it is clear that empathy is implemented by a complex network of distributed, often recursively connected, interacting neural regions including the STS, AIC, mPFC and vmPFC, amygdala and ACC, as well as autonomic and neuroendocrine processes implicated in social behaviors and emotional states. In addition, empathy is not a passive affective resonance phenomenon with the emotions of others. Rather, goals, intentions, dispositions, context and motivations play feed-forward functions in how emotions are perceived and experienced.

sharing, and this phenomenon starts very early in life, by approximately 10 weeks of age (Field, Woodson, Greenberg, & Cohen, 1982; Haviland & Lewica, 1987). The cognitive aspects of empathy are closely related to processes involved in theory of mind (ToM, the capacity to infer the explicit content of others' mental states such as intentions and beliefs), executive function (attention, working memory and inhibitory control), and self-regulation. The regulation of internal emotional states and processes is particularly relevant to the modulation of vicarious emotion and the experience of empathy and sympathy. Both theory of mind and emotion regulation tap into executive function resources implemented in the prefrontal cortex (Zelazo, Carlson, & Kesek, 2008), with different regions (medial and dorsolateral respectively) through their connections with subcortical limbic structures subserving distinct functions. The prefrontal cortex develops more slowly than other brain areas, reaching maturation only late in adolescence (Bunge, Dudukovic, Thomasson, Vaidya, & Gabrieli, 2002). It is well documented that the prefrontal cortex and its functions follow an extremely protracted developmental course, and age-related changes continue well into adolescence (Toga, Thompson, & Sowell, 2006). The maturation of the prefrontal cortex allows children to use verbalizations to achieve self-regulation of their feelings and exercise inhibitory control over their thoughts, attention, and action (Diamond, 2002).

Recent developmental neuroscience research indicates that the affective, cognitive, and regulatory aspects of empathy involve interacting, yet partially nonoverlapping, neural circuits with distinct developmental trajectories. Functional MRI measures reveal age-related changes in the patterns of activation and functional connectivity in individuals (aged between 7 and 38 years) when they are exposed to empathy-eliciting stimuli, reflecting a shift from a visceral emotional response critical for the analysis of the affective significance of stimuli and mediated by the amygdala, posterior insula and OFC to a more evaluative function which critically involved the mPFC and vmPFC (Decety & Michalska, 2010).

### The Evolutionary Origins of Empathy

Human beings are intrinsically social and their survival critically depends on social interactions with others, the formation of alliances, and accurate social judgments (Cacioppo, 2002). It is therefore logical that dedicated neurobiological mechanisms have evolved to perceive, understand, predict, and respond to the internal states (subjective in nature) of other individuals. While one needs to be cautious regarding the forms of behaviors in the animal kingdom that have been interpreted as evidence of empathy and sympathy (see Silk, 2007, for a critical review), basic affective feelings-and the neural mechanisms to support them-are shared by all mammals. In the case of social species, the care for offspring sufficiently long that they too reproduced thereby ensuring their genetic legacy is associated with the ability to perceive and respond to emotional expressions of hunger, pain, distress or fear. MacLean (1985) proposed that empathy emerged in relation with the evolution of mammals (180 million years ago). In the

evolutionary transition from reptiles to mammals, three key developments were: (1) nursing, in conjunction with maternal care; (2) audio-vocal communication for maintaining maternaloffspring contact; and (3) play. The development of this behavioral triad may have depended on the evolution of the thalamocingulate division of the limbic system, a derivative from early mammals. This division (which has no distinctive counterpart in the reptilian brain) is, in turn, geared in with the prefrontal cortex that, in human beings, may be inferred to play a key role in familial acculturation. When mammals developed parenting behavior, the stage was set for increased exposure and responsiveness to emotional signals of others, including signals of pain, separation, and distress. Indeed, parenting involves the protection and transfer of energy, information, and social relations to offspring. African hominoids, including chimpanzees, gorillas, and humans, share a number of parenting mechanisms with other placental mammals, including internal gestation, lactation, and attachment mechanisms involving neuropeptides such as oxytocin (Geary & Flinn, 2001).

The phylogenic origin of behaviors associated with social engagement has been linked to the evolution of the autonomic nervous system and how it relates to emotion. Social approach or withdrawal stems from the implicit computation of feelings of safety, discomfort, or potential danger. Porges (2001) proposed that the evolution of the autonomic nervous system (sympathetic and parasympathetic systems) provides a means to understand the adaptive significance of mammalian affective processes, including empathy and the establishment of lasting social bonds. These basic evaluative systems are associated with motor responses that aid the adaptive responding of the organism. At a primitive level, appetitive and aversive behavioral responses are modulated by specific neural circuits in the brain that share common neuroarchitectures among mammals (Parr & Waller, 2007). These brain systems are genetically hardwired to enable animals to respond unconditionally to threatening, or appetitive, stimuli using specific response patterns that are most adaptive to the particular species and environmental condition. The limbic system, which includes the hypothalamus, the parahippocampal cortex, the amygdala, and several interconnected areas (septum, basal ganglia, nucleus accumbens, AIC, retrospenial cingulate cortex, and prefrontal cortex) is primarily responsible for emotion processing. What unite these regions are their roles in motivation and emotion, mediated by connections with the autonomic system. The limbic system also projects to the OFC and ACC which are involved in the evaluation and regulation of emotion.

At the behavioral level, it is apparent from the descriptions of ethologists that behaviors homologous to empathy and sympathy can be observed in other mammalian species. Notably, reports on ape empathic reactions suggest that they have an explicit appreciation of the other's situation. A good example is consolation, defined as reassurance behavior by an uninvolved bystander toward one of the combatants in a previous aggressive incident (de Waal & van Roosmalen, 1979). Without doubt, some aspects of empathy are present in other species, such as motor mimicry and emotion contagion (see de Waal & Thompson, 2005). An experiment in which peripheral skin temperature (indicating greater negative arousal) was measured in chimpanzees while they were exposed to emotionally negative video scenes demonstrated that skin temperature decreased, indicative of negative sympathetic arousal, when subjects viewed videos of conspecifics injected with needles or videos of the needles themselves, but not videos of a conspecific chasing the veterinarian (Parr, 2001). Thus, when chimpanzees are exposed to meaningful emotional stimuli, they are subject to physiological changes similar to those observed during fear in humans, which is similar to the dispositional effects of emotional contagion (Hatfield, Rapson, & Le, 2009).

In humans, the construct of empathy accounts for a more complex psychological state than the one associated with the automatic sharing of emotions. As in other species, emotions and feelings may be shared between individuals, but humans also can intentionally "feel for" and act on behalf of other people whose experiences may differ greatly from their own (Batson et al., 1991; Decety & Hodges, 2006). This phenomenon, called empathic concern or sympathy, is often associated with prosocial behaviors such as helping kin, and has been considered as a chief enabling process for altruism. According to Wilson (1988), empathic helping behavior has evolved because of its contribution to genetic fitness (kin selection). In humans and other mammals, an impulse to care for offspring is almost certainly genetically hardwired. Once the empathic capacity evolved, following the principle of motivational autonomy (i.e., motivation for a given behavior becomes disconnected from its ultimate goals), it could be applied outside the parental-care context. When people send money to distant earthquake victims, or when a dolphin rescues a drowning individual, empathy reaches beyond its context of evolutionary origins.

The emotional and social aspects associated with empathy in humans rely on ancient systems for intersubjectivity that are shared with other primates. However, layered on top of this, higher level (in the sense of newer) abilities for understanding others' mental states, language, executive functions and, more generally, metacognition expanded the range of behaviors that can be driven by empathy. In addition, as emphasized by Harris (2000), humans can put their emotions into words, allowing them not only to express emotion but also to report on current as well as past emotions. These reports provide an opportunity to share, explain, and regulate emotional experience that is not found in other species. Conversation helps to develop empathy, for it is often here that people learn of shared experiences and feelings.

Interestingly, two key regions, the ACC and the AIC, involved in affective processing in general and empathy in particular, have singularly evolved in apes and humans. The structural and functional organization of the ACC positions it ideally to participate in the regulation of behavior. This is based on three key elements: motor channels, which provide access to skeletomotor and oculomotor output systems; extensive connections with the lateral prefrontal cortex, which provide access to the cognitive apparatus of this neocortical area; and afferents from the midline thalamus and the brainstem, which provide a strong modulatory influence reflecting the arousal state of the organism

(Paus, 2001). The AIC receives inputs from the ventromedial nucleus of the thalamus that convey emotional and homeostatic information, connects reciprocally with the secondary primary sensory cortex, and plays a critical role in the subjective awareness of emotional states (Craig, 2002). Both the ACC and AIC are strongly interconnected with the amygdala, hypothalamus, OFC, and brainstem homeostatic regions. Cytoarchitectonic studies indicate that a population of large spindle neurons is uniquely found in the AIC and ACC of humanoid primates (Allman, Watson, Tetreault, & Hakeem, 2005). They reported a trenchant phylogenetic correlation, in that spindle cells are most numerous in aged humans, but progressively less numerous in children, gorillas, bonobos and chimpanzees, and nonexistent in macaque monkeys. These spindle neurons interconnect the most advanced portions of limbic sensory (AIC) and ACC (Craig, 2007). This is in sharp contrast to the tightly interconnected and contiguous sensorimotor cortices, which are situated physically far apart as a consequence of the pattern of evolutionary development of limbic cortices. Thus, the spindle neurons could enable fast, complex, and highly-integrated emotional behaviors. In support of this view, convergent functional imaging findings reveal that the AIC and the ACC are conjointly activated during all human emotions. According to Craig (2002), this indicates that the limbic sensory representation of subjective feelings (in AIC) and the limbic motor representation of volitional agency (in ACC) together form the fundamental neuroanatomical basis for all human emotions, consistent with the definition of an emotion in humans as both a feeling and a motivation with concomitant autonomic sequelae (Rolls, 1999).

Overall, this evolutionary conceptual view is compatible with the hypothesis that advanced levels of social cognition may have arisen as an emergent property of powerful executive functioning assisted by the representational properties of language (Barrett, Henzi, & Dunbar, 2003). These higher levels operate on previous levels of organization and should not be seen as independent of, or conflicting with, one another. Evolution has constructed layers of increasing complexity, from nonrepresentational (e.g., emotion contagion) to representational and metarepresentational mechanisms, which need to be taken into account for a full understanding of human empathy.

#### Shared Neural Circuits between Self and Other

It has long been suggested that empathy involves resonating with another person's unconscious affect. For instance, Ax in 1964 proposed that empathy can be thought of as an autonomic nervous system state that tends to simulate the state of another person. Similarly, Basch (1983) speculated that, because their respective autonomic nervous systems are genetically programmed to respond in a similar fashion, a given affective expression by a member of a particular species can trigger similar responses in other members of that species. This idea fits neatly with the notion of embodiment, which refers both to actual bodily states and to simulations of experience in the brain's modality-specific systems for perception, action, and the introspective systems that underlie conscious experiences of emotion, motivation, and cognitive operations (Niedenthal, Barsalou, Winkielman, Krauth-Gruber, & Ric, 2005).

The view that unconscious automatic mimicry of a target generates in the observer the autonomic response associated with that bodily state and facial expression subsequently received empirical support from behavioral and physiological studies marshaled under the perception–action coupling account of empathy (Preston & de Waal, 2002). The core assumption of the perception–action model of empathy is that perceiving a target's state automatically activates the corresponding representations of that state in the observer, which in turn activates somatic and autonomic responses. Further, this direct-matching hypothesis, at first glance, fits neatly with the simulation models of emotion processing, which propose that our ability to understand the intentions and emotions expressed by others relies on internally simulating the same psychological state in ourselves (Goldman & Sripada, 2005).

The discovery of sensorimotor neurons, called mirror neurons, provides a physiological mechanism for this direct link between perception and action. Mirror neurons are a unique class of cells with sensorimotor properties that were first identified in the monkey ventral premotor cortex area F5. In one of the seminal papers, Gallese and colleagues (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996) reported that approximately 17% of neurons recorded in ventral premotor area F5 of the macaque monkey responded both when the monkey executed a particular movement-for example, grasping, placing or manipulating-and when the monkey observed someone else performing that same movement. Later, neurons with similar visuo-motor properties were discovered in the anterior intraparietal area (Fogassi et al., 2005), and recently in the primary motor cortex (Tkach, Reimer, & Hatsopoulos, 2007). Many functions have been attributed to mirror neurons, including action understanding, imitation, empathy, and even mind-reading. However, with the relatively recent discovery of such cells in the primary motor cortex, mirror neurons may be best interpreted as motor system facilitators, acting via learned associations (Hickok, 2009). Further, it was recently argued, from a fine conceptual analysis of empirical research on mirror neurons and their putative contribution to theory of mind, that motor resonance is neither necessary nor a sufficient mechanism for representing another individual's intentions, especially in a social context (Jacob, 2008).

Evidence for the existence of mirror neurons in humans is indirect and principally relies on functional neuroimaging studies that reported overlapping activation between observation and action-execution conditions in regions homologous to the areas of the monkey brain where mirror neurons have been found. These regions include the IFG (*pars triangularis*), the ventral premotor cortex (*pars opercularis*), anterior and posterior intraparietal sulcus, and an area in the lateral occipital cortex (e.g., Dinstein, Hasson, Rubin, & Heeger, 2007). In addition, TMS studies have demonstrated changes in the excitability of the observer's brain motor and premotor cortices that encode the execution of observed actions (e.g., Fadiga, Fogassi, Pavesi, & Rizzolatti, 1995). Applying repetitive TMS on the premotor or somatosensory cortices impairs the motor facilitation effects (Catmur, Walsh, & Heyes, 2007). Similarly, magnetoencephalography and electroencephalographic measurements have demonstrated suppression in the mu rhythm (8–13 Hz) over the sensorimotor cortex during the observation of action that parallels the changes detected during action production (Cheng, Yang, Lin, Lee, & Decety, 2008). It has been hypothesized that this mu rhythm reflects downstream modulation of primary sensorimotor areas by mirror-neuron activity, representing a critical information-processing function, translating perception into action (Pineda, 2005).

The somatosensory cortex seems to be endowed with selfother sharing properties. In one study, similar activations in the secondary somatosensory cortex were detected when participants were exposed to videos of someone else's legs being touched with a stick and when they were being actually touched on their legs (Keysers et al., 2004). Another study found that the visual perception of touch was associated with increased activity in the primary somatosensory cortex, and this activity was somatotopically organized (i.e., different areas react to the observation of someone being touched on the neck and the face) (Blakemore, Bristow, Bird, Frith, & Ward, 2005).

The precise location of mirror neurons in the human brain is still a matter of debate. Two meta-analyses concluded that the ventral precentral gyrus-and not the IFG-shares the visual properties of mirror neurons found in area F5 of the macaque brain (Grèzes & Decety, 2001; Morin & Grèzes, 2008). Another recent meta-analysis of 20 functional MRI studies of imitation of hand and finger movements reported that in the frontal lobe, the dorsal premotor cortex rather than the IFG 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 frontal mirror neuron area, the pars opercularis of the inferior frontal gyrus.<sup>1</sup> Finally, a study using the asymmetric fMRI adaptation paradigm failed to reveal evidence for mirror neurons in humans (Lingnau, Gesierich, & Caramazza, 2009). Thus, it is not very clear what function mirror neurons are subserving, and to what extent they play a role in affect sharing.

The sharing of affective states may simply rely on the activation of the core affect which refers to the automatic discrimination of a stimulus—or features of a stimulus—as appetitive or aversive, hostile or hospitable, pleasant or unpleasant, threatening or nurturing (Barrett, Mesquita, Ochsner, & Gross, 2007). Subcortical circuits, including the amygdala, hypothalamus, hippocampus and OFC, are the essential neural components of affective arousal. The amygdala and OFC with reciprocal connection with the STS underlie rapid and prioritized processing of emotion signal.

#### To What Extent Do We Share the Emotions of Others

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.

In line with the perception-action matching mechanism, a number of behavioral and electromygraphic studies demonstrated that viewing facial expressions triggers similar expressions on the viewer's own face, even in the absence of conscious recognition of the stimulus (Hatfield et al., 2009). While watching someone smile, the observer activates the same facial muscles involved in producing a smile at a subthreshold level, and this would create the corresponding feeling of happiness in the observer. There is evidence for such a mechanism in the recognition of emotion from facial expression. Viewing facial expressions triggers distinctive patterns of facial muscle activity, even in the absence of conscious recognition of the stimulus (Dimberg, Thunberg, & Elmehed, 2000). One study 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 (Sonnby-Borgstrom, Jonsson, & Svensson, 2003). However, another study did not find any relation between emotion recognition performance and participants' tendency to mimic dynamic displays of emotions (Hess & Blairy, 2001). Selective facial EMG responses were detected in participants presented with movie clips of morphed (nonnatural) happy and angry facial expressions, but no correlation between the intensity of facial mimicry and dispositional empathy level was found (Achaibou, Pourtois, Schwartz, & Vuilleumier, 2008). However, signal change in EMG was detected in subjects when they were exposed to videos of facial expression of pain only when they adopted an imagine-self perspective but not during an imagine-other perspective (Lamm, Porges, Cacioppo, & Decety, 2008).

Making a facial expression generates changes in the autonomic nervous system and is associated with feeling the corresponding emotion. In a series of experiments, participants were instructed to produce facial configurations for anger, disgust, fear, happiness, sadness, and surprise while heart rate, skin conductance, finger temperature, and somatic activity were monitored (Levenson, Ekman, & Friesen, 1990). Results showed that such a voluntary facial activity produced significant levels of subjective experience of the associated emotions as well as specific and reliable autonomic measures. Unfortunately, these results have never been reproduced, and a series of meta-analyses conducted by Cacioppo, Berntson, Larsen, Poehlmann, and Ito (2000) indicated that even a limited set of discrete emotions such as happiness, sadness, anger, and disgust cannot be fully differentiated by visceral activity alone. The only consistent result is that negative emotions are associated with stronger ANS responses than are the positive emotions.

A number of functional neuroimaging studies have demonstrated that imagining emotional experiences from one's own and from someone else's perspective produce similar patterns of brain activation as well as psychophysiological reactions (Decety & Grèzes, 2006). For instance, Ruby and Decety

(2004) presented participants with short written sentences that depicted real-life situations (e.g., someone opens the toilet door that you have forgotten to lock) which induce social emotions (e.g., shame, guilt, pride) as well as emotionally neutral situations, and asked them to imagine how they would feel if they were in those situations, and how their mother would feel in those situations. Cortical regions that are involved in emotional processing were found similarly activated in the conditions that integrated emotional-laden situations for both self and other's perspectives, including the amygdala and the temporal poles. Interestingly the interaction between the emotional content and perspective-taking factors led to a cluster in the right somatosensory cortex, which was stronger for the self-perspective. Another study combined psychophysiology (heart rate and skin conductance) and neuroimaging measurements in participants who were required to imagine: (1) a personal experience of fear or anger from their own past; (2) an equivalent experience from another person as if it were happening to them; and (3) a nonemotional experience from their own past (Preston et al., 2007). When participants could relate to the scenario of the other, they produced patterns of psychophysiological and neuroimaging activation equivalent to those of personal emotional imagery. The somatosensory cortex (though not on the right) was specifically involved in the first-person imagery.

Single-pulse TMS applied to the right somatosensory cortex during emotion-judgment tasks selectively disrupts the recognition of facial expression of fear, but not happy expressions (Pourtois et al., 2004). Additional support for a role of the somatosensory cortex in emotion recognition comes from a study designed to test whether recognizing facial expressions requires visual processing followed by simulation of the somatovisceral responses associated with the perceived expression. In this study, Pitcher, Garrigo, Walsh, and Duchaine (2008) targeted the right occipital face area and the face region of right somatosensory cortex with repetitive transcranial magnetic stimulation (rTMS) while participants discriminated facial expressions. Results demonstrated that rTMS selectively impaired discrimination of facial expressions at both sites but had no effect on a matched-face-identity task.

A more indirect reference to the mirror neurons systems in empathy, which became popular during the past five 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. This has led to an unfortunate confusion between mirror neurons and shared neural substrates.

The idea that the mirror neuron system is implicated in emotion perception is based on studies that have reported activation in the inferior frontal gyrus (IFG: an area homologue to the monkey ventral premotor cortex) during the observation and the imitation of facial expression of emotions (e.g., happiness, sadness, anger, disgust and surprise) and during the imitation of these emotions (see, e.g., Carr, Iacoboni, Dubeau, Mazziotta, & Lenzi, 2003). One study used a paradigm in which subjects had

to observe and imitate hand and face actions (smile and frown condition) using film clips instead of static displays (Leslie, Johnson-Frey, & Grafton, 2004). The right IFG was commonly activated during observation and imitation of facial expressions. A more recent study demonstrated that even passive viewing of facial expressions activates a wide network of brain regions that were also involved in the execution of similar expressions, including the IFG and the posterior parietal cortex (van der Gaag, Minderaa, & Keysers, 2007). However, it is important to note that the majority of functional neuroimaging studies have not reported activation of the IFG or other mirror neurons areas during the perception of facial expression of emotion (see Murphy, Nimmo-Smith, & Lawrence, 2003; Phan, Wager, Taylor, & Liberzon, 2002 for meta-analyses). For instance, Chakrabarti, Bullmore, & Baron-Cohen (2006) presented participants with video clips depicting happy, sad, angry and disgusted facial expressions. Only the perception of happy expressions was associated with an activation of the left pars opercularis. Finally, it is worth mentioning that many studies claiming to have found mirror-neuron-system activation during action and emotion tasks do not have the appropriate experimental conditions to support such a claim (Turella, Pierno, Tubaldi, & Castiello, 2009). Most studies have simply disregarded activity in all cortical areas except for the IFG and anterior intraparietal sulcus (aIPS), because these two areas are assumed to be homologous to monkey areas F5 and PF and are therefore expected to contain mirror neurons. Using such circular reasoning, these studies have sidestepped the most important issue, which is to examine whether human mirror neurons actually exist and to characterize their physiology (Dinstein, Thomas, Behrmann, & Heeger, 2008). This circular interpretation has been taken to such an extreme that many studies interpret any hemodynamic response in the IFG and aIPS as being due to mirror neuron activity, as if the regions only consisted of mirror neurons, thus both grossly ignoring that mirror neurons in the monkey account for only a small minority of cells and that these areas subserve different computations, such as cognitive control and task management in the case of the right IFG (e.g., Kawashima et al., 1996; Swick, Ashley, & Turken, 2008).

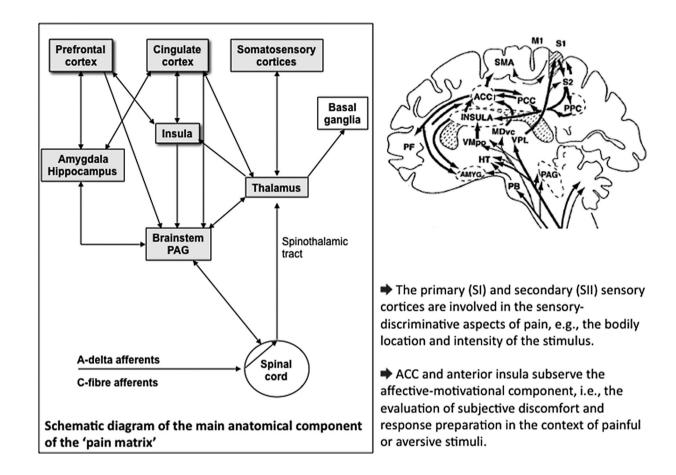
### Perceiving Others in Pain

Pain serves evolved protective functions not only by warning the suffering person, but also by impelling expressive behaviors that attract the attention of others (Craig, 2009). It has been argued that the long history of mammalian evolution has shaped maternal brains to be sensitive to signs of suffering in one's own offspring (Haidt & Graham, 2007). In many primates as well as many social animals, this sensitivity has extended beyond the mother–child relationship, so all normally developed individuals dislike seeing others suffering. For instance, rats that had learned to press a lever to obtain food would stop doing so if their action is paired with the delivery of an electrical shock to a visible neighboring rat (Church, 1959). This example illustrates the functional connection between the first-hand experience of pain, its perception in others, and empathic concern, which draws on the encephalization of pain evaluation (Tucker, Luu & Derryberry, 2005). Developmentally, empathic distress (i.e., aversive feeling contingent on another's physical, emotional, or economic distress) plays a crucial role in the building blocks of morality (Hoffman, 1990).

Pain is conceived as a subjective experience triggered by the activation of a mental/neural representation of actual or potential tissue damage. This representation involves somatic sensory features, as well as affective-motivational reactions associated with the promotion of protective or recuperative visceromotor and behavioral responses. It is the affective experience of pain that signals an aversive state and motivates behavior to terminate, reduce, or escape exposure to the source of noxious stimulation (Price, 2000). The expression of pain also provides a crucial signal that can motivate soothing and caring behaviors in others. It is therefore a valuable and ecologically-valid means to investigate the mechanisms underlying the experience of empathy.

A growing body of research demonstrates shared physiological mechanisms between the first-hand experience of pain and the perception of pain in others (Figure 2). In the first functional MRI experiment, study participants were scanned during a condition of feeling a moderately-painful pinprick stimulus to the fingertips and another condition in which they watched another person's hand undergo similar stimulation (Morrison, Lloyd, di Pellegrino, & Roberts, 2004). Both conditions resulted in common hemodynamic activity in a pain-related area in the right dorsal ACC. In contrast, the primary somatosensory cortex showed significant activations in response to noxious tactile, but not visual, stimuli. Another fMRI study demonstrated that the dACC, AIC, cerebellum, and brain stem were activated when healthy participants experienced a painful stimulus, as well as when they observed a signal indicating that another person was receiving a similar stimulus. However, only the actual experience of pain resulted in activation in the somatosensory cortex and a more ventral region of the ACC (Singer et al., 2004). These findings were supported by two fMRI studies in which participants were shown still photographs depicting right hands and feet in painful or neutral everyday-life situations, and asked to imagine the level of pain that these situations would produce (Jackson, Meltzoff, & Decety, 2005; Jackson, Rainville, & Decety, 2006). Significant activation in regions involved in the affective aspects of pain processing, notably the dACC, the thalamus, and AIC was detected. Unlike the first neuroimaging studies of pain empathy mentioned above, more recent functional MRI and MEG investigations reported significant signal change in the somatosensory cortex/posterior insula, a region involved in the sensory discriminative dimension of pain (Akitsuki & Decety, 2009; Benuzzi, Lui, Duzzi, Nichelli, & Porro, 2008; Cheng et al., 2007, 2008; Jackson, Brunet, Meltzoff, & Decety, 2006; Lamm & Decety, 2008; Lamm, Meltzoff, & Decety, 2009; Lamm, Nusbaum, Meltzoff, & Decety, 2007; Moriguchi et al., 2007).

Facial expressions of pain constitute an important category of facial expression that is readily understood by observers. One study investigated the neural response to pain expressions by performing fMRI as participants viewed short video sequences showing faces expressing either moderate pain or, for comparison,



**Figure 2.** Neurophysiological research on pain processing points out a distinction between the sensory-discriminative and the affective-motivational domains. The former domain engages stimulus localization and is assessed with ratings of intensity while the latter one involves the affective component of pain and is measured with ratings of unpleasantness. This duality is also framed in terms of medial and lateral thalamic processing and extent for cortical structures including somatosensory and ACC, respectively based on thalamic afferents. These two dimensions of pain processing are underpinned by discrete yet interacting neural networks. A growing number of neuroimaging studies recently demonstrated that the observation of pain in others recruits brain areas chiefly involved in the affective and motivational processing of direct pain perception (areas colored in gray). The AIC lies between the lateral and medial systems and is involved in processing associated with each system including sensory coding, body-state assessment, and autonomic regulations as well as emotional valence-coding of sensory events. The ACC mediates the three aspects of pain processing that may use affect but are explicitly involved in avoidance/nocifensive behaviors.

no pain (Botvinick et al., 2005). In alternate blocks, the same subjects received both painful and nonpainful thermal stimulation. Facial expressions of pain were found to engage cortical areas also engaged by the first-hand experience of pain, including the ACC and AIC. Similarly, Lamm, Baston and Decety (2007), exposed study participants to videos of individuals expressing pain due to listening to painful sounds, and also exposed the participants to the same painful sounds in the scanner. Overlapping activation between first-hand experience of pain and second-hand perception of pain in others was found in the aMCC, SMA, AIC, amygdala, and PAG.

Most neuroimaging studies that have explored the overlap in brain response between the observation of behavior performed by others and the generation of the same behavior in self have used simple subtraction methods and generally highlight the commonalities between self and other processing, and ignore the differences. This is particularly true for the recent series of fMRI studies that have reported shared neural circuits for the first-hand experience of pain and the perception of pain in others (see Jackson, Rainville, et al., 2006). It is possible, as argued by Zaki, Ochsner, Hanelin, Wager, and Mackey (2007), that common activity in the ACC and AIC reflect the operation of distinct but overlapping networks of regions that support perception of self or other pain. To address this issue, the authors scanned participants while they received noxious thermal stimulation (self-pain condition) or watched short videos of other people sustaining painful injuries (other-pain condition). Analyses identified areas

whose activity covaried with ACC and AIC activity during self or other pain either across time (intra-individual connectivity) or across participants (inter-individual connectivity). Both connectivity analyses identified clusters in the midbrain and periaqueductal gray with greater connectivity to the AIC during self-pain as opposed to other pain. The opposite pattern was found in the dorsal mPFC, which showed greater connectivity to the ACC and AIC during other pain than during self-pain using both types of analysis. Intra-individual connectivity analyses also revealed regions in the superior temporal sulcus, posterior cingulate, and precuneus that became more connected to the ACC during other pain compared with self-pain. The results of this experiment document distinct neural networks associated with ACC and AIC in response to first-hand experience of pain and response to seeing other people in pain. These networks could not have been detected in prior work that examined overlap between self and other pain in terms of average activity, but not connectivity. Individual subject analyses in generic space similarly indicate that distinct neural networks in anterior and aMCC are activated during the first-hand versus second-hand experience of pain (Morrison & Downing, 2007). This is in line with a quantitative meta-analysis of studies on empathy for pain versus pain in the self, using activation likelihood estimation (Decety & Lamm, 2009a). This analysis revealed distinct subclusters in both aMCC and the AIC. While activation in aMCC seems to be more left-lateralized, caudal, and dorsal during empathy for pain, a rostro-caudal activation gradient is evident in the insular cortex. These distinct activation patterns suggest the involvement of only partially overlapping neural subpopulations and indicate the involvement of distinct cognitive and affective processes. It should also be kept in mind that the effective spatial resolution of fMRI, the different experimental paradigms as well as the inherently complex mapping from cognitive to neural/hemodynamic processes make it difficult to achieve a definite conclusion about how much of the activation during empathy for pain can be attributed to shared neural and mental representations.

Summing up, neuroimaging evidence indicates that perceiving or imagining another individual in pain is associated with hemodynamic responses in the neural network processing the motivational-affective and the sensory dimensions of pain in oneself. Recent studies have documented that this network is modulated by various social and interpersonal factors, such as by perceived agency (Akitsuki & Decety, 2009), social context (Cheng et al., 2007), attitudes such as attribution of responsibility and stigma (Decety, Echols, & Correll, 2009), and racial bias (Xu, Zuo, Wang, & Han, 2009). Thus, incoming sensory information is constrained by appraisal and reappraisal processing that shapes the emergence of the experience of empathy and behavioral outcomes. It is worth noting that vicariously instigated activations in the pain matrix are not necessarily specific to the emotional experience of pain, but to other processes such as negative stimulus evaluation, attention to noxious stimuli, somatic monitoring, and the selection of appropriate skeletomuscular defensive movements. Thus, the shared neural representations in the affective-motivational part of the pain matrix may not be specific to the sensory qualities of pain, but instead might be associated with more general survival mechanisms such as aversion and withdrawal when exposed to danger and threat (Yamada & Decety, 2009).

#### Impact of Brain Lesions on Empathy

While neuroimaging data are merely correlational, studies of neurological patients are critical to infer the role of a given area, and give the functional weight of that region to the cognitive process studied. Considering the multifaceted nature of empathy, it is to be expected that there may be distinct disorders related to empathy rather than a unique deficit.

Supporting evidence for a role of the somatosensory cortex in emotion processing is provided by a lesion study which reported that damage of the right somatosensory cortex (including the anterior supramarginal gyrus) was associated with impaired recognition of facial expressions (Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000). Such a finding is consistent with an account of emotion recognition involving shared neural circuits (i.e., we recognize the emotional expressions of others by relying on somatosensory representations). However, whether this mechanism is always necessary is open to debate and more research. Indeed, several neuropsychological observations speak against shared circuits between emotion experience and emotion recognition (Heberlein & Atkinson, 2009). For instance, Keillor, Barrett, Crucian, Kortenkamp, & 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 or mental imagery 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 the emotions signified by facial expressions of others (Calder, Keane, Cole, Campbell, & Young, 2000; Rives Bogart & Matsumoto, 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—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 aMCC and AIC, two key regions of the so-called "shared circuits" for self and other pain (Danziger, Faillenot, & Peyron, 2009).

Behavioral variants of frontotemporal dementia (FTD), a group of related conditions resulting from the progressive degeneration of the temporal and frontal lobes and dramatic social cognition impairments, constitute an important source of knowledge in the relationship between empathy and cognition. In one study, first-degree relatives were asked to use the interpersonal reactivity index to rate 18 patients with FTD, 19 patients with semantic dementia. 16 patients with Alzheimer's disease, and 10 age-matched healthy control subjects (Rankin, Kramer, & Miller, 2005). Both, groups with FTD and semanticdementia showed significantly lower levels of empathy than either the group with Alzheimer's disease or the normal control one. Patients with semantic dementia showed disruption of both emotional and cognitive empathy, whereas FTD patients showed only disruption of cognitive empathy. In a second study conducted by Rankin et al. (2006), the neuroanatomic basis of empathy was investigated in 123 patients with FTD, Alzheimer's disease, corticobasal degeneration and progressive supranuclear palsy also using the IRI. The subscales of empathic concern and perspective-taking were correlated with structural MRI brain volume using voxelbased morphometry (i.e., a technique used for comparison of brain volume and detection of regional brain atrophy). Voxels in the right temporal pole, the right fusiform gyrus, the right caudate and right subcallosal gyrus correlated significantly with the total empathy score. Empathy scores correlated positively with the volume of right temporal structures in semantic dementia, and with subcallosal gyrus volume in frontotemporal dementia. These findings suggest that the right anterior temporal and medial frontal regions are essential for real-life empathic behavior. The study of degenerative neurological diseases has also supplied evidence for relatively-distinct routes to social cognition and empathy deficits. For instance, Snowden et al. (2003) have shown that both patients with FTD as well as patients with Huntington's disease (HD), characterized by involuntary movements, present difficulties in tasks of social cognition. However, the two patient groups display qualitatively different patterns of results. This suggests that the deficits of patients with 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 lack empathy, but for different reasons. In the former group, the loss of empathy arises more at an emotional than a cognitive level, while FTD patients live in an egocentric world in which they do not ascribe independent mental states to others. A compatible finding from a voxel-based morphometry analysis on FTD patients revealed that atrophy in bilateral temporal lobe and medial orbitofrontal structures correlated with loss of cognitive empathy and that atrophy to the temporal pole correlated significantly with loss of emotional empathy (Rankin et al., 2005). These findings are consistent with the idea of distinct neural underpinnings for the cognitive and affective aspects of empathy (Decety & Jackson, 2004).

Clinical reports have consistently indicated that acquired damage to the prefrontal cortex may result in severe impairment in interpersonal behavior, including empathy and sympathy. For instance, a series of studies using the IRI in patients with neurological lesions showed reduced empathy following right hemisphere damage. Empathy was most severely impaired following lesions within the right frontal structures and, most notably, the ventromedial region, suggesting a greater role for right PFC structures in the mediation of empathy (Shamay-Tsoory, Tomer, Berger, & Aharon-Peretz, 2003; Shamay-Tsoory, Tomer, Berger, Goldsher, & Aharon-Peretz, 2005).

Another study by the same group recently found a behavioral and anatomic double dissociation between deficits in cognitive empathy (associated with ventromedial lesions) and emotional empathy (associated with lesion of the left inferior frontal gyrus (Shamay-Tsoory, Aharon-Peretz, & Perry, 2009). The pattern of empathy deficits among these patients represents a first direct evidence of a double dissociation between emotional and cognitive empathy using the lesion method. However, the fact that the lesion of the inferior frontal gyrus was in the left hemisphere is at odds with the neuroimaging studies with healthy volunteers that reported right-sided activation of the IFG in recognition of emotion.

Impairment of the medial/cingulate prefrontal cortex is commonly associated with deficits in social interaction and selfconscious 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).

In sum, neurological studies indicate a critical role of the medial and orbitofrontal cortex in social emotions, including empathy and sympathy (e.g., Shamay-Tsoory, 2009). In addition, there is little evidence from neurological studies that lesion of the regions involved in the mirror neuron system (ventral premotor, motor cortex and anterior IPS) leads to any dysfunction in empathy, sympathy or moral reasoning, whereas lesions of the ventromedial prefrontal cortex are associated with such socio-cognitive disturbances (e.g., Hornak et al., 2003). Additional work is necessary to determine the specific role of these frontal regions in the experience of empathy and new tasks need to be developed to evaluate empathy and sympathy more reliably than the simple use of questionnaires such as the IRI (see Box 1).

# The Regulation of Empathy

A fundamental human capacity is the ability to regulate and control emotions, thoughts and behavior. The regulation of internal emotional states and processes is particularly relevant to the modulation of vicarious emotion and the experience of empathy and sympathy. In humans, fear and personal distress are usually not associated with empathic concern or prosocial behavior, but with self-directed efforts and avoidance behavior.

As illustrated in the opening paragraph of this article, when we are exposed to another person in distress, our reaction can range from concern for personal safety, including feelings of alarm, fear and avoidance, to concern for the other person, including compassion, sympathy, and care-giving. In the case of perceiving others suffering, the somatic sensorimotor resonance in pain-processing areas between other and self may

#### Box 1. Correlations between empathy disposition and brain activation

A number of self-report questionnaires are available to assess individual differences in empathic disposition. The most widely used are the BEES (Mehrabian, 1997), the IRI (Davis, 1983), and the EQ (Baron-Cohen and Wheelwright, 2004).

Among these measures, the IRI has been particularly used in neuroimaging studies. The IRI consists of four subscales; each measuring a different aspect of empathy: perspective taking, empathic concern, fantasy, and personal distress.

A selective review of the studies that used the IRI in search of correlation with brain response to the perception of others in pain does not support any reliable nor meaningful relationship between empathy disposition and neural processing. One study reported strong correlation between activation in the ACC (r = 0.62) and left insula (r = 0.52) and the IRI subscale of empathic concern (Singer et al., 2004). Another one found significant correlations between the IRI empathic concern subscale and activation in the dorsal premotor cortex, left ventral premotor cortex, and left somatosensory cortex, but no significant clusters were detected in the insula or ACC (Lamm, Nusbaum, et al., 2007). However, the vast majority of studies did not find any correlation between subscales of the IRI and hemodynamic activation (Akitsuki & Decety, 2009; Cheng et al., 2007; Danziger et al., 2009; Decety, Echols, et al., 2009; Jackson et al., 2005; Lamm, Batson, & Decety, 2007; Lamm & Decety, 2008; Lamm et al., 2009; Moriguchi et al., 2007).

It is unclear as to whether this inconsistency is due to a lack of power from the small number of participants typically found in fMRI designs, or due to the broader issue of whether such generalized disposition should or should not predict specific neural responses in context-specific situations.

trigger empathic concern and feelings of sympathy. But the same signals may also constitute a threat to the individual that can lead to personal distress (Yamada & Decety, 2009). If not regulated, this distress can be costly, both physiologically and cognitively, and can eventually conflict with the observer's capacity to be of assistance to the other (Decety & Lamm, 2009b). Difficulty inhibiting or reducing an emotional response and excessive attention towards negative emotional information may deplete the resources available for other aspects of selfregulation (Muraven & Baumeister, 2000). Lacking the ability to regulate emotions can result in deleterious emotional arousal and the mis-identification of emotions, hindering the ability to function adaptively and appropriately.

Empathy is particularly important in patient-physician communication, and is associated with improved patient satisfaction and compliance with recommended treatment (Epstein et al., 2007). However, as Hodges and Biswas-Diener (2007) argued, there are costs to being too empathic. For instance, paying attention to an other's suffering in the course of caring for patients experiencing trauma or pain can exhort a cost for medical practitioners, exhausting their emotional resources and ironically reducing their capacity for or their interest in bearing the suffering of others. Empathy may thus be viewed as a double-edged sword, facilitating caring and compassion but at the same time leaving the physician vulnerable (Figley, 2002; Sabo, 2006). It is therefore critical that physicians develop effective emotion appraisal and regulation processes in the context of providing care to their patients. Indeed, in order to cope with repeated exposure to the suffering of others and minimize negative arousal, which would deplete executive functioning, physicians as well as other emergency-service personnel learn to regulate their interpersonal sensitivity. However, active (conscious) regulation of negative emotions has physiological and sociopsychological costs too. For instance, research has shown that it can disrupt communication, reduce rapport and increase blood pressure (Butler et al., 2003). Thus, without some powerful

regulatory mechanisms, it is very likely that medical practitioners would experience personal distress and anxiety when facing other people in pain, and this negative arousal would interfere with their ability to heal.

Another important aspect to be elucidated is whether the down-regulation is the outcome of conscious inhibitory or unconscious inhibitory processing. A number of studies have shown that the former mode of emotional regulation (also called expressive suppression) may be particularly costly and disrupts multiple aspects of social exchange, creating stress for both the regulator and the interaction partner alike (Butler et al., 2003). Such suppression is accompanied by increased sympathetic and cardiovascular responding and reduces memory for social information (Gross & Levenson, 1993). Physicians face the challenge of devoting the right balance of cognitive and emotional resources to their patients' pain experience. They must try to resonate and understand the patient without becoming emotionally overinvolved in a way that can preclude effective medical management.

One functional MRI study from our group provided the first evidence of the impact of medical expertise on the neural response to witnessing painful situations being experienced by another person (Cheng et al., 2007). This study compared the brain hemodynamic response in a group of physicians and a group of matched control participants when they were exposed to short video clips depicting hands and feet being pricked by a needle (painful situations) or being touched by a cotton bud (nonpainful situations). Unlike control participants, physicians showed a significantly reduced neurohemodynamic empathic response in the AIC and ACC, and no activation of the PAG (a mediator of the flight-or-fight response) when shown video clips of body parts being pricked by a needle. Instead, cortical regions underpinning executive functions and self-regulation (dorsolateral and medial prefrontal cortex) and executive attention (precentral, superior parietal and temporo-parietal junction) were found

to be activated. Connectivity analysis further demonstrated that activation in the medial and dorsolateral prefrontal cortices subserving executive control and self-regulation (Ochsner & Gross, 2005) was inversely correlated with activity in the AIC in the physicians, indicating an executive suppression of the emotional response to the other's pain.

To determine at what stage of information processing this regulation occurs, Decety, Yang, and Cheng (2010) recorded event-related potentials (ERP) from physicians and matched controls as they were exposed to the same visual stimuli. The results in control participants showed an early N100 differentiation between pain and no pain over the frontal area as well as a late-positive potential around 300–800 ms over the centroparietal regions. In contrast, no such early and late ERP responses were detected in the physicians has very early effects, inhibiting the bottom–up processing of the perception of pain in others. One may suggest that physicians' down-regulation of the pain empathy dampens their negative arousal in response to the pain of others and thus may have beneficial consequences in the efficacy of being of assistance.

#### **Conclusion and Caveats**

For a very long time, empathy has been a focus of speculation in philosophy and in the empirical investigations of social psychology and developmental science. But in the past decade, empathy research has blossomed into a vibrant and multidisciplinary field of study, appealing to scholars in economics, evolutionary biology, and affective neuroscience. Much of this new work relies on functional neuroimaging studies that investigated the perception of a restricted number of primary facial expressions of emotions such as disgust or fear, and pain, mainly outside of any social context. The combined results of these studies demonstrate a partial overlap between the neural regions involved in recognizing emotion and emotional experience. In the case of pain, when individuals perceive or imagine others in painful or distressful situations, the pain matrix is activated to a great extent, and this activation includes the somatosensory cortex.

Such a shared neural mechanism between other and self offers an interesting foundation for intersubjectivity because it provides a functional bridge between first-person information and third-person information grounded in self-other equivalence (Decety & Sommerville, 2003; Sommerville & Decety, 2006), allows analogical reasoning and offering a possible route, yet is partial to understanding others. However, while the capacity for two people to resonate with each other emotionally, prior to any cognitive understanding, is the basis for developing shared emotional meaning, it is not enough for mature empathic understanding and sympathetic concern. Such an understanding requires forming an explicit representation of the feelings of another person, an intentional agent, which necessitates additional computational mechanisms beyond the emotion-sharing level, as well as self-regulation to modulate negative arousal in the observer (Decety,

Michalska, & Akitsuki, 2008; Decety & Moriguchi, 2007). In order to appreciate the emotions and feelings of others in relation to oneself, second-order representations of the other need to be available to awareness without confusion between self and other. This necessitates a decoupling computational mechanism between first-person information and secondperson information, for which the medial and ventromedial prefrontal cortices play crucial roles (Decety & Jackson, 2004). These representations become more abstract as we move forward, such that the most anterior region of the medial prefrontal cortex is associated with metacognitive representations that enable us to reflect on the values linked to outcomes and actions (Amodio & Frith, 2006). This idea is strongly supported by lesion studies that consistently document the critical role of the orbitofrontal cortex in empathy.

Whether the sharing of neural circuits really supports a simulationist model of emotion recognition remains an open question. There is little evidence for brain circuits that are selectively implicated in particular emotions, a key element for the theory of simulation. The current neurophysiological data are more compatible with the model of core affects than distinct categories of emotions (Barrett, 2006; Barrett & Wager, 2006), which conceptualizes emotion states as the interaction of two orthogonal dimensions (valence and arousal). For instance, the neural circuit associated with the perception of pain in others (ACC, AIC, PAG, and somatosensory cortex) may have more to do with the activation of the processing of threat-related (i.e., negative) information, which may trigger aversive or even defensive behavior. Part of the ACC may work together with the SMA during pain observation to recognize the aversive nature of the event, to mount an appropriate motor response, and to modulate this response according to prevailing task constraints (Morrison, Peelen, & Downing, 2007). The SMA as a result of feedback from the limbic system represents one of the anatomical substrate for activating the motor response associated with danger and threats (Oliveri et al., 2003).

There is no doubt that the construct of empathy is useful at the phenomenological level-we need words to navigate the social world. Yet it may be too complex to be both meaningful and useful for sound research in affective and social neuroscience. Certainly, the behaviors associated with, or triggered by, what social psychologists and biologists call "empathy" are heterogeneous in the extreme, ranging from motor mimicry, emotional contagion, and imagination of others' feelings, to altruism, sympathy, cruelty and so on. While the different disciplines' ideas of empathy clearly have something in common, one could justifiably question the heuristic or conceptual advantages of one monolithic concept of empathy. This serious reservation highlights the need for a more careful conceptualization of empathy. Our future understanding of empathy, whether derived from the social sciences, biological sciences, or economics, will benefit from such a fine conceptual analysis.

Breaking down empathy and related phenomena into component processes will also be beneficial in the exploration of psychiatric conditions of disordered or abnormal empathy such as psychopathy, narcissistic personality disorder, or conduct disorder in children (Blair & Blair, 2009; Decety, Michalska, Akitsuki, & Lahey, 2009; Decety & Moriguchi, 2007). Trying to understand impairments of empathy and their related behaviors requires an examination of development, hormones and physiology, brain structure and function, behavior, personality, and social context. By focusing on fundamental mechanisms of the brain and behavior rather than on discrete psychiatric disorders, we can gain insights into therapeutic interventions that may be applied to a range of disorders. In this way, the fundamental approach offers the biggest pay-off in the final analysis.

#### Note

1 Meta-analyses are crucial to accumulate consensus across tasks that involve putatively similar processes while washing out statistical idiosyncrasies in individual studies.

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#### Appendix

Abbreviations used in the article

ACC	anterior cingulate cortex
AIC	anterior insular cortex
dACC	dorsal anterior cingulate cortex
aMCC	anterior medial cingulate cortex
dlPFC	dorsolateral prefrontal cortex
EMG	electromyography
fMRI	functional magnetic resonance imaging
IFG	inferior frontal gyrus
IPS	intraparietal sulcus
mPFC	medial prefrontal cortex
OFC	orbitofrontal cortex
PAG	periaqueductal gray
SMA	supplementary motor area
SPL	superior parietal lobule
STS	superior temporal sulcus
ToM	theory of mind
TMS	transcranial magnetic stimulation
vmPFC	ventromedial prefrontal cortex