The Neural Bases for Empathy

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Abstract

Human empathy relies on the ability to share emotions as well as the ability to understand the other's thoughts, desires, and feelings. Recent evidence points to 2 separate systems for empathy: an emotional system that supports our ability to empathize emotionally and a cognitive system that involves cognitive understanding of the other's perspective. Converging evidence from neuroimaging and lesion studies shows that a neural network that includes the inferior frontal gyrus and the inferior parietal lobule is necessary for emotion recognition and emotional contagion. On the other hand, the involvement of the ventromedial prefrontal cortex, temporoparietal junction, and the medial temporal lobe in self-reflection and autobiographical memory places these key regions as necessary for cognitive empathy. The proposed dissociation between these systems is supported by recent neurochemical experiments involving administration of oxytocin as well as by ethological, psychiatric, and developmental studies. Finally, although the emotional and cognitive systems appear to work independently, every empathic response may still evoke both components to some extent, depending on the social context.

Keywords

empathy, theory of mind, emotion, ventromedial prefrontal, inferior frontal gyrus, mirror neurons

Empathy is a broad concept that refers to the cognitive as well as the emotional reactions of one individual to the observed experiences of another. There are several well-acknowledged controversies in psychology, biology, and ethology over whether empathy is unique to humans and whether it is an emotional (sensing another's feelings) or cognitive (understanding another's perspective) construct. Neuroimaging, lesion, and behavioral studies with humans and animals have been increasingly capable of characterizing the neural basis of empathy, thus providing new insights into these questions. Recent evidence supports a model of 2 separate systems for empathy: an emotional system and a cognitive system. The capacity to experience affective reactions to the observed experiences of others or share a "fellow feeling" has been described as emotional empathy. As shown in Figure 1, emotional empathy may involve several related underlying processes, including, among others, emotional contagion, emotion recognition, and shared pain. On the other hand, as shown in Figure 1, the term *cognitive empathy* describes empathy as a cognitive role-taking ability, or the capacity to engage in the cognitive process of adopting another's psychological point of view (Frith and Singer 2008). This ability may involve making inferences regarding the other's affective and cognitive mental states (Shamay-Tsoory and others 2009) .

Current evolutionary evidence supports the existence of several systems mediating empathy. De Waal (2008) suggests that the phylogenetically earliest system is the emotional contagion system, in which one is affected by another's emotional or arousal state. On the other hand, the cognitive empathic perspective-taking system is a more advanced system and involves higher cognitive functions including mental state attribution. Indeed, emotional contagion has been reported in rodents (Langford and others 2006), whereas only the closest living relatives of humans, the chimpanzees, possess rudimentary traits of cognitive aspects of empathy such as theory of mind (Call and Tomasello 2008).

In accordance with this ethological evidence, developmental studies also indicate that emotional contagion (e.g., contagious crying) is observed earlier in young babies than cognitive perspective–taking abilities, which are acquired during cognitive development (de Waal 2008).

Furthermore, recent evidence suggests a dissociation between cognitive and emotional empathy in psychiatric disorders such as autism (Dziobek and others 2008) and borderline personality disorder (Harari and others 2010), supporting the possibility that some individuals may show

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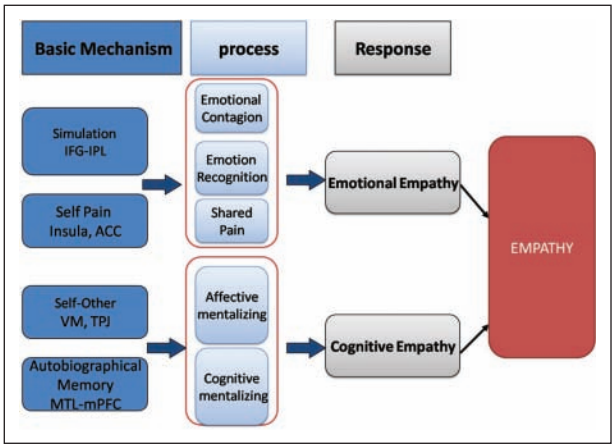

Figure 1. The components that participate in the cognitive and the emotional empathy networks. Each component is associated with distinct functions that comprise the empathic response.

impairment in one system and intact ability in the other system and vice versa (Dziobek and others 2008).

The distinction between the emotional and cognitive empathic subprocesses may relate also to different neurochemichal systems. Although it is likely that the empathic response is modulated by several neurotransmitters and neuromodulators, in a recent study, intranasal administration of the neuropeptide oxytocin increased emotional, but not cognitive, empathy (Hurlemann and others 2010), suggesting that the oxytocingeric system, which has been associated with attachment and pair bonding, may modulate emotional but not cognitive empathy. On the other hand, it has been recently suggested that dopaminergic functioning is associated with cognitive aspects of empathy in preschool students (Lackner and others 2010).

This suggests that although the 2 systems may work together, they may be behaviorally, developmentally, neurochemically, and neuroanatomically dissociable. Based on recent findings, a neuroanatomical model for empathy is proposed here. Table 1 depicts the possible underlying evolutionally, neurochemical, developmental, and neural mechanisms of emotional and cognitive empathy.

It seems likely that each component in the empathy network is associated with distinct functions that comprise the empathic response. To fully characterize the empathy network, it is necessary to identify the roles of each contributing brain region to the processes that support the 2 systems (Figure 1).

Emotional Empathy

Because emotional empathy is essentially the elicitation of corresponding emotions and respective related behaviors in the observer, it may be suggested that the mere perception of emotion in others will activate the same neural mechanisms that are responsible for the first-hand emotional experience and that the motor response corresponding to the particular emotion will be automatically activated.

Emotional Contagion and Simulation

The basic emotional contagion system is thought to support our ability to empathize emotionally. According to Preston and de Waal's (2002) perception-action hypothesis, perception of a behavior in another automatically activates one's own representations for the behavior, and output from this shared representation automatically proceeds to motor areas of the brain where responses are prepared and executed. This state-matching reaction has been related to the simulation theory, which suggests that processing of social information involves activating neural states during observation that match those that the observer experiences in a similar situation (Gallese 2007). Simulation theories were greatly reinforced by the discovery of the mirror neurons, a set of neurons that fire both when a monkey acts and when it observes the same action performed by another monkey (Rizzolatti and others 2009). Given its observation-execution properties, it was suggested that the mirror neuron system (MNS) is particularly well suited to provide the appropriate mechanism for motor empathy, imitation, and emotional contagion. As shown in Figure 2, in humans, the MNS has been identified in the inferior frontal gyrus (IFG; Brodmann's Area [BA] 45/44/6) and in the inferior parietal lobule (IPL; BA 39,40). It has been suggested that the IFG identifies the goals or intentions of actions by their resemblance to stored representations for these actions (Rizzolatti and others 2009). Nonetheless, although many authors believe that the MNS has a central role in empathy, others question its role in empathy and suggest that the MNS is positioned to support primarily motor goals (Thioux and others 2008).

Although it is reasonable to question the various applications of the MNS in all types of empathic responses, there is consistent and strong evidence for the involvement of the IFG in emotional contagion and emotion recognition. Indeed, it has been suggested that overt facial mimicry (as measured by an electromyograph or through observation) is related to emotional contagion and emotion understanding (Niedenthal 2007). The existence of mirror neurons related to emotional facial expressions in the human IFG suggests that the human MNS may be used to convert observed facial expressions into a pattern of neural activity that would be suitable for producing similar facial expressions and provide the neural basis for emotional contagion (Keysers and Gazzola 2006). Jabbi

Table 1. Comparison of Emotional and Cognitive Empathy

	Emotional Empathy	Cognitive Empathy
Behavior	Emotion Recognition, Emotional Contagion, Motor Empathy, Shared Pain	Cognitive ToM, Affective ToM, Perspective Taking
Neuroanatomical networks	IFG, IPL, ACC, AI	vmPFC, dmPFC, TPJ, MTL
Phylogenetic	Rodents	Primates
Developmental	Infants	Adolescents
Neurochemical mechanism	Oxytocin	Dopamine

ToM = theory of mind; IFG = inferior frontal gyrus; IPL = inferior parietal lobe; ACC = anterior cingulated; AI = anterior insula; vmPFC = ventromedial prefrontal cortex; dmPFC = dorsomedial prefrontal cortex; TPJ = temporoparietal junction; MTL = medial temporal lobe.

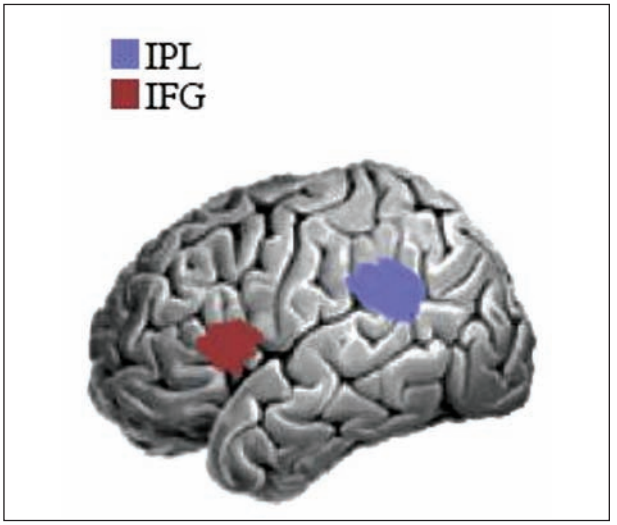

Figure 2. The emotional contagion network.

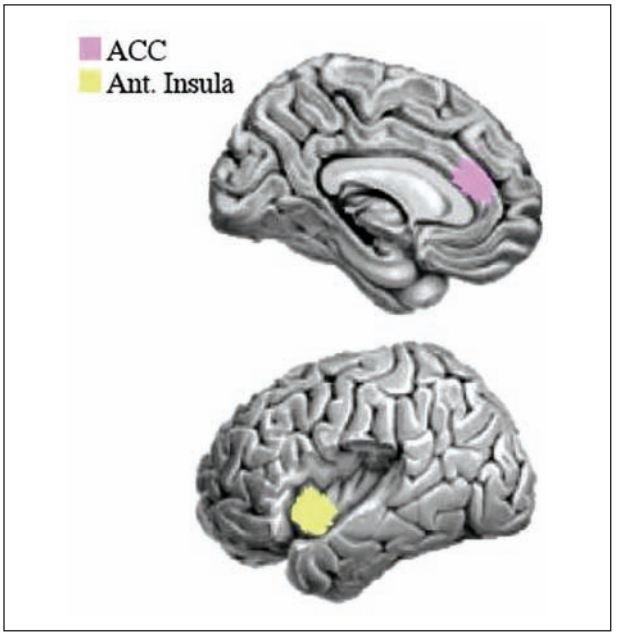

Figure 3. The empathy to pain network.

and others (2007) have reported that observing positive and disgust facial expressions activated parts of the IFG and that participants' empathy scores were predictive of their IFG activation while witnessing facial expressions. In addition, 2 neuroimaging studies, one that involved emotion recognition (Schulte-Ruther and others 2007) and one that involved empathizing with people suffering serious threat or harm (Nummenmaa and others 2008), have further emphasized the specific role of the IFG in emotional empathy. Finally, it has been recently reported that cortical lesions involving the IFG, particularly in BA 44, are associated with impaired emotional contagion and deficits in emotion recognition, whereas lesions in the ventromedial prefrontal cortex result in impaired cognitive empathy (Shamay-Tsoory and others 2009), suggesting that the IFG not only participates in tasks that involve emotional empathy but is also necessary for emotional empathy.

Empathy to Pain

Whereas emotion recognition and emotional contagion appear to involve the IFG, shared pain appears to involve regions related to the first-hand experience of pain, such as parts of the pain matrix. Specifically, a network including the anterior cingulate cortex (ACC) and the insula (see Figure 3) was reported to respond to both felt and observed pain (Decety and Echols 2010). Activation in the ACC and insula has been found also to correlate with the participant's judgments of the subjective severity of pain experienced by others on the basis of the other's facial pain expression (Saarela and others 2007). This indicates that empathizing with people in pain is associated with hemodynamic activity in the brain that is similar to the activity that occurs when people feel pain themselves.

The underlying possible mechanism of empathic response to pain may be evolutionarily adaptive on several levels. On one level, empathizing with the pain of others may aid in the immediate perception and avoidance of a threat to oneself (Yamada and Decety 2009). On another

level, shared pain may also serve a prosocial function, by facilitating cooperation between family members who share a similar genetic makeup (Preston and de Waal 2002). This suggests that empathy to pain is, at least in part, an automatic, bottom-up process and perhaps an evolved adaptation. Yet, research also strongly suggests that empathy to pain is also mediated by top-down processing. In fact, recent neuroimaging studies have demonstrated that the empathic response to pain is either strengthened or weakened when contextual and interpersonal variables are manipulated. For example, Xu and others (2009) have demonstrated that empathic neural response in the ACC decreased when participants viewed pain applied to faces of other races. Furthermore, in a recent event-related potentials study, Decety and others (2010) have demonstrated that the emotion regulation of empathy to pain in physicians (who need to deal with the pain of their patients on a daily basis) has a very early effect, inhibiting the bottom-up processing of the perception of pain in others. This indicates that the inhibition of empathy to pain response may be rapid and early.

To conclude, the crux of emotional empathy appears to be the generation of corresponding (to the target) emotional response (e.g., the insula in shared pain) and the corresponding motor representation (IFG) related to the emotion. The neural networks that participate in this system are detailed in Figures 2 and 3. Although this system appears to be bottom-up, some top-down processes may modulate this automatic system, and perhaps aspects of higher-order cognitive process as well as cognitive empathy interact with emotional empathy in such cases.

Cognitive Empathy

Although emotional contagion appears to be the common denominator of all forms of empathy, some complex forms of empathy involve the ability to create a theory about the other's mental state and cognitively take the perspective of others. This process of understanding another person's perspective, termed *cognitive empathy*, appears to involve theory of mind.

Theory of Mind

Theory of mind (ToM) may be defined as the ability to put oneself into someone else's shoes, imagine their thoughts and feelings (Baron-Cohen 2009). ToM, also known as mentalizing, enables one to extract and understand the goals of others by drawing on the capacity to understand the other's thoughts, intentions, emotions, and beliefs and predict their behavior (Amodio and Frith 2006). Consistent with this possibility that mentalizing comprises several distinct processes that meet different cognitive demands, recent studies have identified a set of brain regions involved in ToM: the medial prefrontal cortex (mPFC), the superior temporal sulcus (STS), the temporoparietal junction (TPJ), and the temporal poles (TP; Frith and Singer 2008; Van Overwalle and Baetens 2009). A recent review of imaging studies of ToM (Carrington and Bailey 2009) found that 93% of the 40 studies reviewed report activation in the mPFC. The TPJ region was active in 58% of the studies reviewed and the STS (including the IPL) in 50% of the studies. Based on a separate meta-analysis, Van Overwalle and Baetens (2009) proposed that the TPJ is mainly responsible for transient mental inferences about other people (e.g., their goals, desires, and beliefs), whereas the mPFC subserves the attribution of more enduring traits and qualities about the self and other people.

Although many studies have considered the mPFC as one unit that mediates ToM, recent studies have proposed a neuroanatomical and behavioral dissociation within the mPFC between dorsomedial and ventromedial (vmPFC) regions (Mitchell 2009). Particularly, it has been suggested that the vmPFC is necessary for the affective aspects of ToM (Shamay-Tsoory and Aharon-Peretz 2007). Indeed, it has been repeatedly demonstrated that ToM is not a monolithic process and that it involves cognitive as well as affective aspects of mentalizing. Affective ToM is not equivalent to emotional empathy; it is an emotional form of mentalizing. Although *cognitive ToM* refers to our ability to make inferences regarding other people's beliefs, *affective ToM* refers to inferences one makes regarding others' emotions. Although lesions in the vmPFC have been associated with impaired affective ToM, Kalbe and others (2010) have recently reported that 1-Hz repetitive transcranial magnetic stimulation, which interferes with cortical activity of the dorsolateral PFC, impaired cognitive ToM.

One of the elementary prerequisites for mentalizing is the basic distinction between actions generated by the self versus others (Mitchell 2009). Although the self-other distinction is also required in emotional empathy, it appears that during higher-level inference-based processes, a network involving the vmPFC and to some extent the TPJ is responsible for shared representations of self and other (Zaki and others 2009). Mitchell and others (2009) have recently suggested that the involvement of the vmPFC in self-reflection places it as a key region necessary for evaluating the similarities and differences distinguishing the mental states of oneself from others. It is possible that situations that involve affective ToM entail more self-reflection as compared with situations involving cognitive ToM, which are more detached. Therefore, the vmPFC, which is highly connected to the amygdala, appears to be particularly necessary

for affective mentalizing as opposed to neutral or cognitive forms of mentalzing.

Impairments in self-other distinction were reported in patients with ToM impairment such as individuals with autism. Recently, Lombardo and others (2010) have demonstrated that whereas healthy individuals recruit the ACC and the vmPFC in response to self as compared with others (referential processing), in autism, the vmPFC responds equally to self and other. The authors concluded that this atypical activation of the vmPFC in self-reflection may account for the mentalizing impairments reported in autism.

Taken together, these studies suggest that the vmPFC forms a core region within the larger mentalizing network (which includes the mPFC, STS, and TP) that is involved in self-other distinction and affective ToM. Indeed, a recent meta-analysis (van der Meer and others 2010) proposed that the connections of the vmPFC with the limbic system place it in a position of a key region for emotional self-reflection. Moreover, the authors propose that although the vmPFC is responsible for emotional self-reflection, a network involving the mPFC and the medial temporal lobes (MTL) is responsible for integrating self-referential representation and autobiographical memory. In agreement with this, the same network reported to participate in mentalizing has been reported to participate in autobiographical memory (Mitchell 2009).

Autobiographical memory, our ability to recall knowledge of our past, has been shown to involve a widespread cerebral network incorporating the MTL and the mPFC. Accumulating data suggest that self-projection, remembering the past, and mentalizing abilities are based on the same core brain networks (Buckner and Carroll 2007), suggesting that these processes share analogous mechanisms.

One plausible hypothesis that emerges from this line of studies is that autobiographical memory and ToM rely on a common set of processes by which past experiences are used to understand events happening to the self as well as to others. Findings from a lesion study, however, have put this hypothesis in question by showing that performance on tasks that involve ToM is not affected by impairments in autobiographical memory (Rosenbaum and others 2007). The authors demonstrated that despite losing the ability to consciously recollect personal history, amnesic patients exhibit intact ToM abilities. Yet, Rabin and others (2010) have recently reported that left MTL structures including the hippocampus have a role in the modulation of ToM with respect of the vividness of the event. This study demonstrates that recollection of autobiographical memories is involved in making inferences regarding other people's mental states.

To conclude, it appears that cognitive empathy involves higher-order cognitive functions that require self-other differentiation, cognitive and affective ToM, and autobiographical memory. As depicted in Figure 4, the self-other distinction and affective ToM involve a network in which the vmPFC (and the TPJ to some extent) is a core region. A network that includes the mPFC and the MTL appears to modulate mentalizing by tracking similar past autobiographical memories.

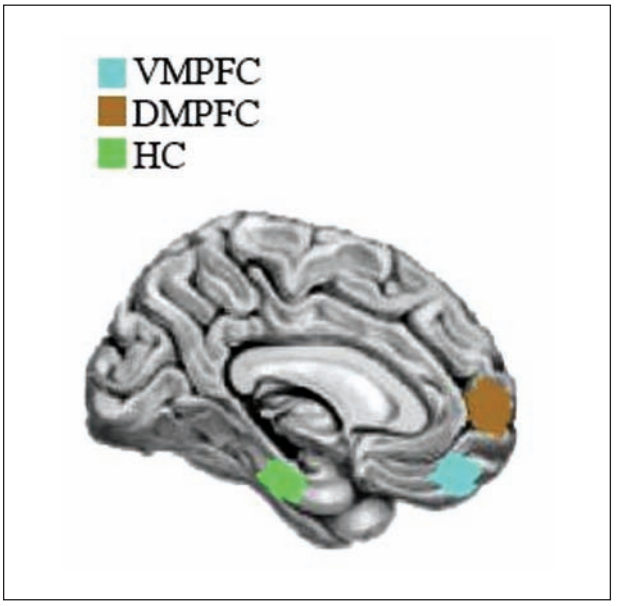

Figure 4. The cognitive empathy network.

Possible Interactions between the 2 Empathy Systems

According to the proposed model (Figure 1), under normal circumstances, every interaction with a protagonist may trigger independently both an emotional response (emotional empathy) and a cognitive evaluation of his or her state of mind and perspectives (cognitive empathy). Although both emotional and cognitive components of empathy may operate partly autonomously, it is likely that every empathic response will evoke both components to some extent, depending on the social context. The protagonist's emotions are shared, activating brain areas involved in simulation and mirroring, including the IFG. Independently, the ability to accurately infer the other's perspective and imagine the protagonist perspective and state of mind is also activated. This process may require self-other decoding and autobiographical memory. Both functional neuroimaging and lesion studies in humans indicate that the vmPFC may play a crucial role in the network performing cognitive empathic function. This system is phylogenetically younger and is unique to primates and human adults.

Nonetheless, although these 2 systems work independently, it is possible that they also interact. Because the MNS appears to be a more basic system, it may be speculated that activation of this system is a prerequisite for the complex of ToM. Van Overwalle and Baetens (2009) proposed that because the MNS and the mentalizing network are rarely concurrently active, it is possible that MNS provides a rapid and intuitive input to the mentalizing system. The author further suggested that the TPJ is the most likely candidate for such a mentalizing area that interacts with the MNS because of its proximity to the IPL.

Future studies may focus on the interactions between these systems and the different conditions that may affect the activation of each system. Different variables such as the level of emotions involved, the past experiences of the empathizer, gender, relationship with the protagonist, and the perceived similarity between the individual and the protagonist may differentially activate the emotional and the cognitive systems. Exploring these questions using a combination of several research tools such as neuroimaging, neurostimulation, and electrophysiology may prove to be essential in characterizing the relationship between these 2 systems and the conditions in which each system is activated.

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