THE NEUROPSYCHOLOGY OF EMPATHY: EVIDENCE FROM LESION STUDIES

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Review

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The neuropsychology of empathy: evidence from lesion studies

Neuropsychologie de l'apathie : apports des études lésionnelles

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Abstract

Empathy is a multi-component process consisting of at least two systems: one which involves state-matching or affective sharing referred to as emotional empathy, and a more deliberate and controlled cognitive component called cognitive empathy [1]. Recent neuropsychological evidence indicates that emotional empathy is supported by a neural network that includes the inferior frontal gyrus and the inferior parietal lobule. This system also involves the empathy for pain network including the anterior insula and anterior cingulate. Cognitive empathy, on the other hand, involves a neural network that includes the ventromedial prefrontal cortex, temporoparietal junction and the medial temporal lobe. The two empathy systems appear to be dissociable and differentially affected in various neuropsychological and psychiatric syndromes. Nonetheless, despite the independence of each system, it appears that every empathic response may still involve to some degree both components. In addition, we show that empathy deficits may affect not only patient's behavior but also the wellbeing of his/her surrounding.

Key words: affective empathy • cognitive empathy • brain lesions

Résumé

L'empathie est un processus psychologique à composantes multiples comprenant au moins deux systèmes : un système dit d'empathie émotionnelle, permettant la correspondance d'états affectifs ou le partage affectif et un système dit d'empathie cognitive dans lequel les états affectifs sont plus délibérés et plus contrôlés [1]. Les données neuropsychologiques récentes indiquent que l'empathie émotionnelle est sous-tendue par un réseau neuronal incluant le gyrus frontal inférieur et le lobule pariétal inférieur. Ce système engage aussi le réseau cérébral pour l'empathie à la douleur qui comprend les régions insulaires et cingulaires antérieures. L'empathie cognitive est sous-tendue par le cortex préfrontal ventro-médian, la jonction temporo-pariétale et le lobe temporal médian. Ces deux systèmes d'empathie semblent être dissociables et susceptibles d'être sélectivement perturbés dans les différents syndromes neuropsychologiques et psychiatriques. Néanmoins, malgré l'indépendance de chaque système, il semble que chaque réponse empathique puisse impliquer, à un certain degré, les deux composantes empathiques. De plus, nous montrons que les déficits d'empathie peuvent affecter, non seulement le comportement du patient, mais aussi son bien-être et son environnement.

Mots clés : empathie affective • empathie cognitive • lésions cérébrales

Introduction

Social interactions depend, to a great measure, on our ability to react empathically to the emotions of the people around us. Empathy is a central mechanism of understanding the other, which helps us sense and understand the

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other's feelings and emotions [2]. Recent evidence indicates that empathy involves two dissociable systems:
– one developmentally and phylogenetically 'early' system for emotional empathy;
– one developmentally and phylogenetically 'later' system for cognitive empathy [1].

Our fundamental affective reaction to the observed experiences of others or share a "fellow feeling" has been described as "*emotional empathy*". This ability supports our tendency to react emotionally to the pain and distress of others and to recognize their emotions. While emotional empathy, involves an emotional reaction towards the suffering of a target, "*cognitive empathy*" involves the ability to engage in the cognitive process of adopting another's psychological point of view [3]. This ability may involve making inference regarding the other's affective and cognitive mental states [4].

Consistent with its multidimensional nature, empathy appears to be supported by a number of neural networks involving several regions including frontal regions, the insula and temporal regions [5]. Neuropsychological studies of individuals with localized lesions and degenerative disorders have pointed to critical regions which are necessary for emotional and cognitive empathy. Lesion studies are particularly essential to the growing study of empathy because it is crucial to demonstrate that regions activated in neuroimaging studies during a task are critical to that task, and not only correlated with the task.

Nonetheless, despite the importance of investigating empathy using a neuropsychological framework, few lesion studies have examined empathy deficits in patients with localized lesions. To fully characterize the emotional and cognitive empathy network, it is necessary to identify the roles of each contributing brain region to the processes that support the two systems.

The emotional empathy system

Emotional empathy involves vicarious sharing of emotions as well as the elicitation of similar emotions experienced by a target in the observer.

According to Preston and de Waal's perception-action hypothesis [6], perception of a behaviour in another automatically activates one's own representations for the behaviour, and output from this "shared" representation automatically proceeds to motor areas of the brain where responses are prepared and executed. Underlying this shared representation is a state-matching reaction (affective resonance component of empathy), which represents the elicitation of corresponding emotions and respective related behaviours in the observer [1]. Indeed, brain imaging studies have confirmed that observing an emotion in others is often sufficient to produce changes in cerebral response that are similar to the changes observed when individuals are actually feeling the emotion [7].

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 [8]. 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 [9]. 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 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 has a major role in identifying the goals or intentions of actions by their resemblance to stored representations for these actions [9]. Indeed, a recent meta-analysis of emotional empathy, which examined 112 experiments, reported that the IFG as well as bilateral anterior insula, anterior and posterior cingulate, bilateral temporoparietal junction, right amygdala, are core regions of emotional empathy [10]. Another meta-analysis which focused on emotion recognition reported that anterior insula and anterior cingulate cortex were the areas most commonly activated [11]. Indeed, the relationship between emotion recognition and empathy has been demonstrated before. It has been suggested that overt facial mimicry (as measured by an electro-myograph or through observation) is related to emotional contagion and emotion understanding [12]. 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 [13]. Jabbi *et al.* [14] 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. Additionally, two neuroimaging studies, one which involved emotion recognition [15] and one that involved empathizing with people suffering serious threat or harm [16] have further emphasized the specific role of the IFG in emotional empathy. Finally, it has been reported that cortical lesions involving the IFG, particularly in BA 44, are associated with impaired emotional contagion and deficits in emotion recognition, while lesions in the ventromedial prefrontal cortex result in impaired cognitive empathy [4], suggesting that the IFG not only participates in tasks that involve emotional empathy but is also *necessary* for emotional empathy.

In addition to the IFG, recent studies point to the role of the right hemisphere to emotional empathy. A voxel-based morphometry study of 123 patients with Alzheimer's disease, progressive supranuclear palsy, corticobasal degeneration and frontotemporal dementia using caregivers' ratings on emotional contagion significantly correlated

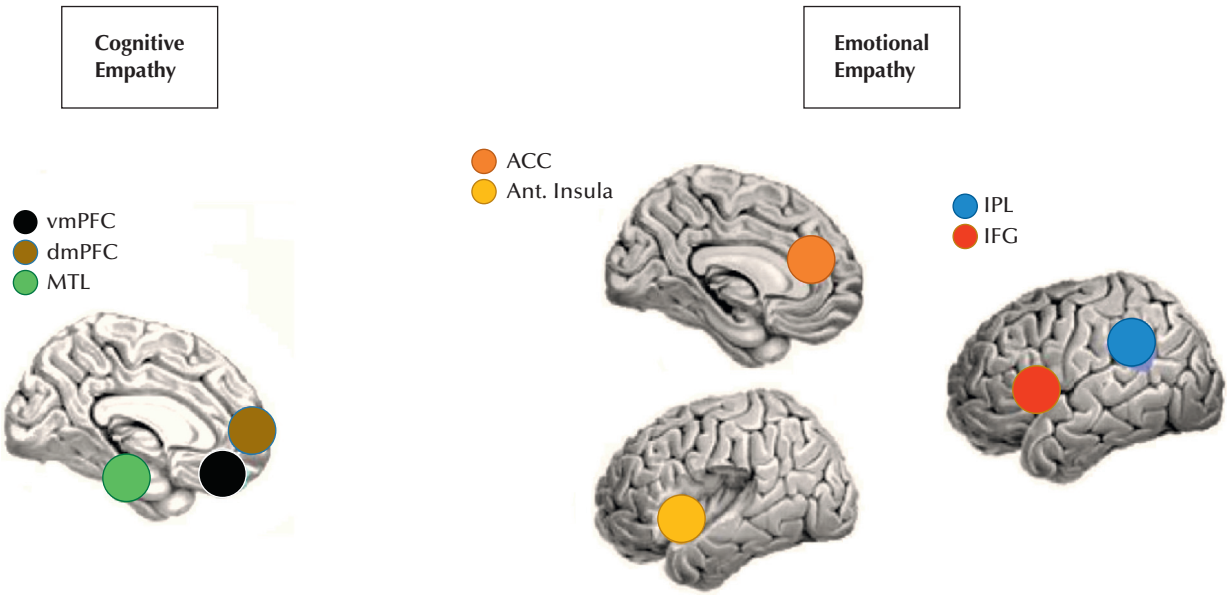

Figure 1. The components of cognitive and emotional empathie.

with the volume of grey matter in right temporal pole, fusiform gyrus and medial inferior frontal region [17]. In line with this, studies of behavioural variant frontotemporal dementia, a neurodegenerative disease characterized by impaired social behavior, have also reported a role of right temporal pole and/or orbitofrontal atrophy in empathy deficits [18-20]. Furthermore, a case study of a patient with hereditary multiple exostoses and frontotemporal dementia revealed severely impaired affective empathy associated with atrophy in right anterior temporal lobe and orbitofrontal gyrus [21]. Leigh *et al.* [22] have recently studied 27 patients with acute right hemisphere ischemic stroke and 24 neurologically intact inpatients on a test of affective empathy. The authors report that impairments in emotional empathy were observed in patients with infarcts in the right temporal pole and right anterior insula. In line with this, Herbet *et al.* [23] have recently examined empathy in a large-sized cohort of 107 patients who had undergone surgery for a diffuse low-grade glioma. The authors found positive association between emotional empathy and the volumes of residual lesion infiltration in the right hemisphere.

Although studies on emotional empathy have focused on emotion recognition and vicarious feelings, another important aspect of emotional empathy is that of empathy for physical pain.

Indeed, state-matching is clearly evident in the case of empathy for pain where the observer shares to some level the experience of pain of the target. While 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. Neuroimaging studies consistently show that nociceptive stimuli commonly elicit activity in a target in a neural network termed the Pain Matrix [24], a system which involves a very wide array of subcortical and cortical brain structures [25, 26] that includes the primary (S1) and secondary (S2) somatosensory cortices, the thalamus, anterior cingulate cortex (ACC) and the anterior and posterior insula (AI, PI). This set of regions has been further subdivided into at least two partly dissociable circuits coding for the sensory (S1, S2, thalamus) and affective (ACC, AI) dimensions of pain [24].

Accumulating evidence point to a network including the anterior cingulate cortex (ACC) and the insula (see *figure 1*) which responds to both felt and observed pain [27]. 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 [28]. 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 role of the AI was recently extended beyond empathy for pain showing that the AI is also critical for emotion recognition. Driscoll *et al.* [29] have studied 192 male Vietnam combat veterans who had sustained focal penetrating traumatic brain injuries, and matched controls. Using voxel-based lesion-symptom, the authors show that the ventrolateral prefrontal cortex, left and right posterior temporal lobes, and insula, were associated with diminished emotional empathy.

Indeed, the role of the AI in empathy has been recently documented in a study by Boucher *et al.* [30] who examined a group of fifteen patients for whom the insula was removed as part of their epilepsy surgery. The authors report that patients who underwent insular resection showed poorer ability to recognize facial expressions of emotions. Specific emotion analyses revealed impairments in fear recognition in both groups of patients, whereas happiness and surprise recognition was only impaired in patients with insular resection. There was no evidence for a deficit in disgust recognition. The findings suggest that unilateral damage to the operculo-insular region may be associated with subtle impairments in emotion recognition, and provide further clinical evidence of a role of the insula in emotional empathy.

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 emotion recognition), and the corresponding motor representation (IFG) related to the emotion.

The cognitive empathy system

Cognitive empathy involves the ability to create a theory about the other's mental state, and cognitively take the perspective of others. Cognitive empathy appears to involve theory of mind.

Theory of mind [31] may be defined as the ability to put oneself into someone else's shoes, imagine their thoughts and feelings [32]. 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 [33]. The processes that comprise theory of mind involves the abilities to represent cognitive and affective mental states, attribute these mental states to self and other, and deploy these mental states in a manner that allows one to correctly understand and predict behavior [34, 35]. Thus, cognitive empathy reflects the representation of the internal mental state of others, which is in effect ToM. 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) [3, 36]. A recent review of imaging studies of ToM [37] 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 et Baetens [36] proposed that the TPJ is mainly responsible for transient mental inferences about other people (*e.g.* their goals, desires and beliefs), while the mPFC subserves the attribution of more enduring traits and qualities about the self and other people.

While 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 (dmPFC) and ventromedial (vmPFC) regions [38]. Particularly, it has been suggested that the vmPFC is necessary for the affective aspects of ToM [39]. 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. While "cognitive ToM" refers to our ability to make inferences regarding other people's beliefs, "affective ToM" refers to inferences one makes regarding others' emotions. While lesions in the vmPFC have been associated with impaired "affective ToM", Kalbe *et al.* [40] have recently reported that 1 Hz repetitive transcranial magnetic stimulation which interferes with cortical activity of the dorsolateral PFC impaired cognitive ToM. On the other hand, Adjeroud *et al.* [41] found that the manifestation of Huntingon's disease was associated with impaired of both cognitive and affective ToM.

One of the elementary prerequisites for mentalizing is the basic distinction between actions generated by the self *versus* others [42]. Although 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 [43]. Mitchell *et al.* [38] 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 to 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 mentalizing.

Impairments in self-other distinction were reported in patients with ToM impairment, such as individuals with autism. Lombardo *et al.* [44] have demonstrated that while 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 (that includes the mPFC, STS and TP), that is involved in self-other distinction and affective ToM. Indeed, a recent meta-analysis [45] proposed that the connections of the vmPFC with the limbic system places it in a position of a key region for emotional self reflection. Moreover, the authors propose that while the vmPFC is responsible for emotional self reflection, a network involving the mPFC and the medial temporal lobes (MTL) is responsible for inte-

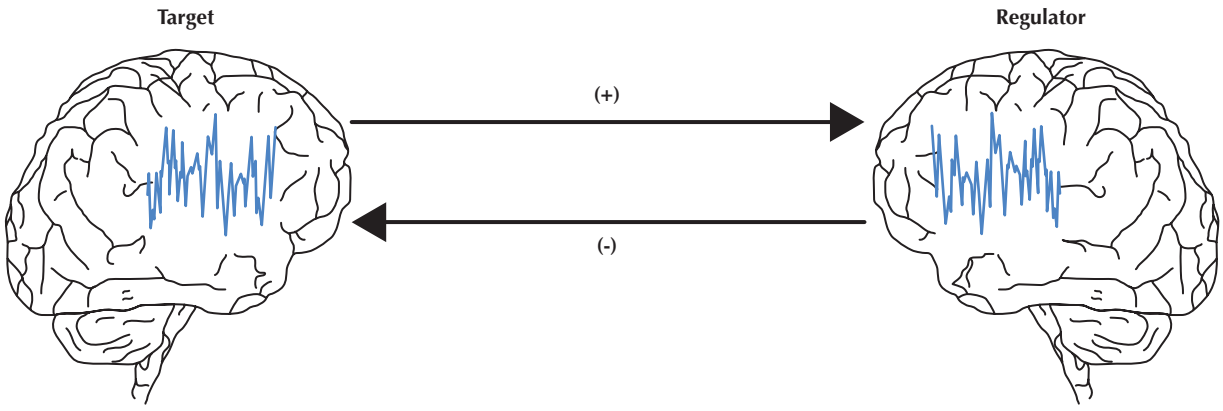

Figure 2. The effects of empathy on the target.
Interpersonal emotion regulation occurs when a target experiences distress, which triggers (+) empathy in the regulator. The activation of empathy circuits in the regulator may initiate an empathic response, which then may diminish (-) levels of distress in the target.

grating 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 [42].

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 [46], 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. Recently, it has been suggested that the "default network" is activated both during mentalizing and episodic retrieval, as well as future simulation.

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 [47]. The authors demonstrated that despite losing the ability to consciously recollect personal history, amnesic patients exhibit intact ToM abilities. Yet, Rabin *et al.* [48] have recently reported that left MTL structures, including the hippocampus, have a role in modulation of ToM with respect of the vividness of the event. In addition, Perry *et al.* [49] have recently suggested that mentalizing is modulated by memories of similar past events and depends on the extent of similarity we feel towards that person. This study demonstrates that recollection of autobiographical memories is involved in making inferences regarding other people's mental states. Andrews-Hanna *et al.* [50] have argued that while mentalizing tasks are preferentially linked to the dorsal medial subsystem, autobiographical tasks engage both the dorsomedial prefrontal cortex as well as the medial temporal lobe, suggesting that the components of the default support both mentalizing autobiographical memory.

To conclude, it appears that cognitive empathy involves higher order cognitive functions that require self-other differentiation, cognitive and affective ToM and autobiographical memory. 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.

Translating the empathy model to every day interactions

In many everyday situations, it is likely that both emotional and cognitive processing will be necessary for social cognition. However, the question remains how empathy deficits may affect patients' ability to provide social support during social interactions.

Indeed, it has been suggested that empathy may support human ability to provide help and support for targets in distress. A new point of view which focuses on the regulation of an individual's emotion through interaction with another person (referred to as "interpersonal emotion regulation") examines how the empathy of the observer affects the emotional state of the sufferer (see *figure 2*). Emotion regulation refers to the processes by which we monitor, evaluate and modify our emotional reactions [51]. While an abundance of research has examined emotional self-regulation [52], few studies have recently suggested that emotion regulation is also affected by interpersonal factors [53].

To understand interpersonal emotion regulation, we must understand not only how the emotions of a target

affect the regulator, but also the way the in which the response of the regulator affects the target. In line with the distinction between emotional and cognitive aspects of empathy proposed above, two types of interpersonal regulatory strategies may be used during interpersonal down-regulation of negative emotions: **emotional strategies** (*e.g.* touch), and **cognitive strategies** (*e.g.* reappraisal). Thus, it is possible that patients with impaired emotional empathy may have difficulties in providing emotional support to other individuals, whereas patients with cognitive empathy deficits may have difficulties in understanding and providing cognitive support to others. It may be speculated that the ability of the regulator to select the best strategy for the target requires taking the target's perspective and therefore may depend upon cognitive empathy.

As described above, the IFG is a core region of the emotional empathy network, and therefore, patients with IFG lesions may show difficulties in providing emotional support to others. On the other hand, the vmPFC is a core region for cognitive empathy and may, therefore, play a crucial role in mediating interpersonal regulatory choice made by the regulator. Indeed, Janowski *et al.* [54] have recently shown that the vmPFC is a central region for empathic choice, which means selecting the best option for a target. Furthermore, Hallam *et al.* [55] have recently examined the neural underpinnings of interpersonal emotion regulation by focusing on the role of the regulator and found that parts of the vmPFC participated in a selection of strategies for someone else, along with other frontal and temporal regions. Taken together, it appears that deficits in empathy may not only affect the patient's ability to understand the social world, but also dampen their ability to provide social support. Lesion studies contribute to the understanding of emotional and cognitive empathy, and how empathy contributes to patients' own social behavior as well as the wellbeing of their surroundings. ■

Conflict of interest

There are no conflicts of interest.

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