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The neuroscience of empathy

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In the fifty years following the first mention of empathy in the neuroscience literature, significant gains in understanding the neural basis of empathy have been made. Converging strands of evidence support simulation-based models of empathy, such that, for example, empathy for pain recruits networks involved in the first-hand experience of pain. Similarly, empathy for other distinct sensory and affective states (e.g. tactile pleasure, fear) leverages the networks involved in the firsthand experience of those states. Such empathic simulations are not unique to humans but can be observed across social species. Both emotional empathy and mentalizing (cognitive empathy) may promote empathic concern or compassion, an outcome variable of particular interest to researchers and practitioners. Although individuals vary in their baseline empathic capacities and proclivities, empathy and concern can be modulated by interpersonal and contextual variables and with training.

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The neuroscience of empathy

The word *empathy* first appeared in the neuroscience literature fifty years ago in a 1967 article by Paul MacLean [1], who defined it as 'the capacity to identify one's own feelings and needs with those of another person.' He viewed empathy as the basis of caring for and desiring to help others, and therefore a topic of critical importance for solving pressing problems of the modern era, including interpersonal callousness and aggression. He concluded with a plea that physicians and scientists of the future not neglect the study of empathy and the brain. One can only imagine the pleasure that the profusion of neuroscience research on empathy in recent decades would have brought him. It might have brought him surprises as well. MacLean described empathy as a holistic and cognitively complex phenomenon that was primarily the endowment of humans, and he hypothesized that it was heavily reliant on the prefrontal cortex. Recent empirical evidence suggests otherwise. Empathy instead appears to reflect multiple dissociable processes, many of which rely on ancient, subcortical structures that function similarly to promote empathy across a variety of social species.

Evidence for simulation-based accounts of empathy

MacLean's definition of empathy anticipated empirical findings that empathically representing others' sensory and emotional states may require leveraging the networks involved in experiencing those states firsthand. Perhaps the clearest such example is observed in the literature on empathy for pain. Early evidence for empathic simulation emerged nearly two decades ago, when co-activation in a single neuron in dorsal anterior cingulate cortex was observed both during experienced and observed pain [2]. Since that time, a large body of functional neuroimaging research has demonstrated that experiencing pain firsthand and observing or inferring others' pain are both associated with increased activation in this and other cortical and subcortical structures collectively described as the 'pain matrix' [3], a network of second-order pain processing regions that include somatosensory cortex, posterior insula, and periaqueductal gray as well as integrative regions involved in affective and motivational aspect of pain, such as the mid-anterior cingulate cortex (which includes the cingulate and paracingulate gyri) and the anterior insula [4,5]. Direct sensory exposure to others' pain is not necessary for empathic activity in this network; reading about others' pain, for example, results in comparable patterns of activity [6]. That overlapping patterns of activity are recruited during experienced and observed or imagined pain echoes MacLean's conjectures about the neural basis of empathy.

However, until recently, a major limitation of this interpretation has been the correlational nature of most investigations of empathic neural responses. Large integrative regions such as the mid-anterior cingulate cortex and anterior insula subserve a wide array of processes not directly related to pain, and each measured voxel within them incorporates activation in thousands of neurons, so fMRI-based observations of shared activation patterns cannot conclusively demonstrate true empathic simulation. But recent experimental approaches to understanding empathy for pain lend further support to simulationist models. Multi-voxel pattern analysis confirms common

neural coding of experienced and empathic pain in anterior insula and mid-anterior cingulate cortex [4]. And placebo analgesia reduces — to comparable degrees — reports of pain during electrical stimulation and descriptions of others' pain during identical stimulation (whereas responses during non-pain control conditions are unaffected) [7••,8,9]. These subjective changes are accompanied by nearly identical reductions in activity in mid-anterior cingulate cortex and bilateral anterior insula during experienced and empathic pain. More, placebo effects for both experienced and empathic pain are blocked by the opioid antagonist naltrexone, indicating that both processes are subserved by comparable neurochemical processes [7••]. Empathy for pain can also be reduced by administration of non-opioid analgesics, including acetaminophen and oxytocin [10,11], or increased using real-time fMRI-based neurofeedback. Using this approach to increase activity in anterior insula and functional coupling between this region and frontal cortex results in increased reported empathy for pain in the absence of changes in general arousal [12]. Collectively, these findings provide strong support for the causal role of empathic simulation in representing others' internal states.

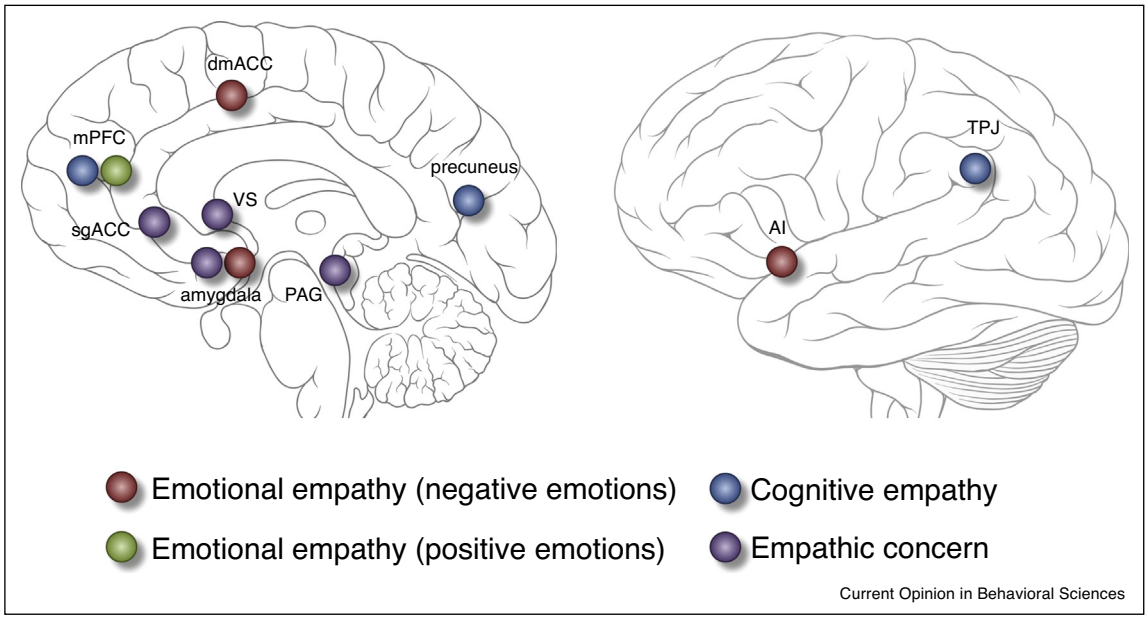
In terms of the specific functions served by regions within the pain matrix, the anterior insula appears to play an interpretive role, as lesions to this region interfere with the ability to simply perceive and recognize others' pain [13]. But debates about the specific role of the mid-anterior cingulate cortex in pain are ongoing. Although it has been suggested that activity in this region encodes pain specifically [14], other theoretical models argue instead that it is primarily involved in allocating control [15] or in threat appraisal [16], such that neither subjective nor empathic pain can be accurately described as localized to this region [17]. But the mid-anterior cingulate cortex is clearly causally implicated in affective and motivational aspects of first-hand pain. Ablations or deep-brain stimulation of this region provide significant pain relief, which appears to reflect reductions in the motivational significance of pain [18–20]. It may therefore be the case that these procedures also decrease the motivational significance of others' pain.

Empathy as multiple distinct processes

Empathic pain is the most robustly supported form of empathy in the neuroscience literature. But it should not be viewed as synonymous with the construct of empathy, which represents a collection of dissociable processes [21] (Figure 1). Indeed, simulation-based accounts of empathy *require* that empathic pain be subserved by different processes than empathy for other sensory and affective states, as empathy for any given state would rely on the recruitment of systems that support the first-hand experience of that state.

Thus, for example, although empathy for physical pain recruits some overlapping neurocircuitry as empathy for

Figure 1



Overview of key regions associated with four forms of empathy. Emotional empathy for negative emotion is associated with activity in dorsal mid-anterior cingulate cortex (dmACC) and amygdala; emotional empathy for positive emotion is associate with activity in medial prefrontal cortex (mPFC); cognitive empathy (mentalizing) is associated with activity in mPFC, temporo-parietal junction (TPJ) and precuneus; and empathic concern is associated with activity in amygdala, ventral striatum (VS), subgenual anterior cingulate cortex (sgACC), and periaqueductal gray (PAG).