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Some considerations on de Waal and Preston review

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In their interesting Review, de Waal and Preston propose that the emotional states of others are "understood through personal, embodied representations that allow empathy" (*Nat. Rev. Neurosci.* **18**, 498–509 (2017))[1]. One of the key pieces of neuroscientific evidence for such a proposal has been the discovery of mirror neurons in the monkey parietal and premotor areas. Despite recognizing that the mirror mechanism plays an important role in various aspects of affective empathy[1], the authors suggest that this mechanism is only a part of a "bigger picture" of empathy, with the mirror mechanism being restricted to the motor system. Other aspects of empathy are linked to the mirror mechanism indirectly, and are better explained by the perception–action model (PAM).

The ability to perform intracranial recordings in humans and then to stimulate the recorded sites in the same patients demonstrated the presence of the mirror mechanism in centres responsible for emotions, thus extending its role outside of the parieto-frontal motor centres. Criteria for claiming that an area is endowed with the mirror mechanism for empathy are the following: electrical stimulation must produce a specific emotion; presentation of social stimuli that show this same emotion should produce a modulation of neural activity in these sites; and, in addition, lesions to the same area should produce impairment in feeling the emotion and in processing the same emotion in others. If these criteria are satisfied, we have three brain areas endowed with the mirror mechanism that automatically transforms sensory representation of a given emotional state, both in terms of feeling that emotion and in terms of the action tendencies associated with this state.

The first is the anterior insula (AI). Stimulation of the ventral AI produces the feeling of disgust in humans[2] and monkeys[3]; administration of disgusting stimuli (natural stimuli), as well as observation of facial expression of disgust (social s timuli), activate AI[2,4]; permanent or temporary lesions to AI impair the capacity of feeling disgust and of recognizing it in others, while the capacity to recognize other emotions remains intact[5,6].

The second is the amygdala (AM), which produces fear when stimulated in humans[7,8]; the presentation of fearful facial expressions triggers faster and stronger responses relative to other facial expressions[9,10]; selective bilateral destruction of AM (Urbach–Wiethe disease) produces an impaired, abnormal experience of fear[11,12]. These patients do not feel fear but are able to describe cognitively what fear is[13].

The third area is the perigenual sector of anterior cingulate cortex (pACC). Stimulation of pACC elicits smiling and laughter often with mirth[14], and the presentation of movies showing laughing, but not crying or neutral expressions, modulates the gamma band activity in the same site[15].

These data strongly support the contention that empathy is based on personal, embodied representations of emotions that are mediated by the mirror mechanism. We argue that this mechanism is not complementary to the PAM, nor a subpart of it; rather, in the light of new emerging studies in the field of human electrophysiology, the mirror mechanism appears to be the principal component responsible for the "overlapping neural signature for experiencing and observing affective states".

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Competing interests statement
The authors declare no competing interests.