Empathy is one of the traits that make us human. In exploring the origins of empathy disorders, however, we can learn a lot by studying animals.
in which their own receipt of a reward (food) was linked to an unpleasant stimulus being delivered to another animal in a neighboring cage refrained from performing the rewarded action.

Under an alternative definition, Frans de Waal’s perception-action model, empathy is a complex behavior that corresponds to three levels of imitation. The most basic level is motor mimicry, which corresponds to emotional contagion. The next level comprises coordination and shared goals, which are reflected in sympathetic concern and consolation. The highest level of complexity comprises perspective-taking and targeted helping, which correlate with true imitation and emulation on the imitation side. According to de Waal, the first two levels of empathy can be found in different animals, whereas the highest level is limited to humans, apes, dolphins, and elephants – in other words, to species whose brains, more specifically the anterior cingulate cortex and the anterior insular cortex, are characterized by the presence of spindle neurons, which are responsible for complex emotions, cooperation, and deception.

Another hierarchical model of empathy has been proposed by Jaak and Jules Panksepp, who linked de Waal’s perception-action model to a broader model of empathy that includes a third dimension: the emotional aspect of empathy. In this model, empathy is a complex behavior that is divided into three levels: cognitive empathy, emotional empathy, and social empathy.

Cognitive empathy is responsible for understanding other people’s thoughts and intentions. Emotional empathy is responsible for feeling what the other person is feeling. Social empathy is responsible for responding to the other person’s emotional state, even if it is different from our own.

Human imperfections

Studies that use neuroimaging techniques show that the aforementioned parts of the brain, namely the anterior insular cortex as well as the anterior cingulate and midcingulate cortices, are the main structures involved in the sharing of both pain and positive emotions in humans. When subjects were shown abstract cues suggesting the infliction of pain to a loved one, their brains exhibited strong activity in the structures responsible for dealing with representations of other people’s emotions, such as the prefrontal cortex, the superior temporal cortex, and the temporo-parietal junction.

Empathy disorders in humans can have a range of causes and should be considered in four categories: cognitive empathy deficit disorder (CEDD), emotional empathy deficit disorder (EEDD), general empathy deficit disorder (GEDD), and general empathy surfeit disorder (GESD). The first, cognitive empathy deficit disorder, is characteristic of neurodevelopmental disorders such as autism spectrum disorders, Asperger’s syndrome, or the agenesis of the corpus callosum, whereas emotional empathy deficit disorder is typical of psychopathy and behavioral disorders of an asocial nature (e.g. aggression). Individuals that show deficits in cognitive empathy lack the capacity to fully identify other people’s emotional states, despite declaring that they share their feelings. By contrast, individuals with emotional empathy deficit disorder are aware of other people’s feelings, but these do not affect them or cause them to feel and behave in a compassionate manner. If also characterized by a lack of scruples, such individuals may have a tendency to abuse and hurt others. The third category, general empathy deficit disorder occurs, in patients who suffer from schizophrenia. Such people have a tendency to isolate themselves from society, which additionally intensifies their symptoms. Lastly, general empathy surfeit disorder is exhibited by patients with Williams syndrome, in whose case it is difficult to determine to what extent they understand other people’s emotions due to coexisting cognitive disorders.

Cognitive and emotional empathy disorders are reflected in low activity of the brain structures that are responsible for imagining other people’s feelings. In children and adults with diagnosed autism spectrum disorders, the insular cortex is characterized by decreased activity and is functionally less connected to the limbic structures, which may cause insufficient stimulation. Curiously enough, autistic individuals show greater empathy towards other autistics, which is attributable to increased activity of the ventral prefrontal cortex (responsible for the detection of similarities). In addition to lower empathy levels related to the inability to differentiate between fear and disgust, individuals with congenital agenesis of the corpus callosum (which is involved in the exchange of information between the left and right hemispheres) are more likely to display empathy.

The argument that empathy is a uniquely human trait is contradicted by research seeking the sources of empathy in the parental care commonly displayed by birds and mammals.
Confirming the occurrence of emotional contagion in mice opened up new avenues of research into the underpinnings of empathy disorders in humans.

For several years, the Laboratory of Emotions Neurobiology at the Nencki Institute has been studying the exchange of emotional information between subjects of the same species. Initially, we looked at pairs of rats. In each pair, one rat (the demonstrator) was subjected to fear conditioning, while the other (the observer) was waiting in the homecage. After the return of the demonstrator, the interaction between the rats was recorded and compared to the behavior of pairs of rats in which the demonstrator had not undergone aversive training. After that, the brains of the rats were removed and analyzed using immunohistochemical tests. The observers paired with the demonstrators that were subjected to aversive conditioning were a lot more interested in the return of their cage mates, which was accompanied by strong activation of the amygdala, for example in the cells of the central nucleus of the amygdala. However, this part of the brain was not activated in the demonstrators that were subjected to conditioning. This part of the brain is involved not only in learning pleasant experiences but also in aversive learning. Further behavioral experiments showed that following contact with the demonstrators subjected to conditioning, the observers were more agitated (enhanced startle responses to acoustic stimuli) and were better at learning the avoidance response in the shuttlebox. Similar results were obtained in mice: the placement of a mouse subjected to conditioning in the cage led to the renewal of the fear response in animals in which the extinction of that response had been already observed.

The confirmation that emotional contagion occurs in mice opened up new avenues of research into the underpinnings of empathy disorders in humans. This is thanks to numerous mouse models of social interaction disorders. In the most recent study, we demonstrated that the transfer of fear between two mice was impaired in BTBR T^Itpr3tf/J mice, characterized by the absence of the corpus callosum. This strain is considered the most extensively studied mouse model of idiopathic autism. Moreover, these animals exhibit not only impaired reception of information from distressed individuals but also impaired neuronal plasticity in response to the same stress stimulus. Combined with data indicating the impairment of functional neuronal plasticity in the amygdala of these animals and the elevated levels of enzymes involved in synaptic plasticity in the amygdala of these mice, this model allows for the identification of a possible mechanisms responsible for impaired emotional contagion. This offers a starting point for further studies using local interventions in the level of proteins regulating neuronal plasticity. Their findings should bring answers to questions related to the molecular mechanisms of empathy disorders.

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