The neurology of disgust

Emotions form part of the fabric of our minds, and the scientific attempts to map these dimensions onto the underlying neural substrates date back to the first half of the last century. It has long been suggested that emotional functions are associated with subcortical structures (e.g. Cannon, 1927; Klüver & Bucy, 1937; Hess, 1954). James Papez (1937) was the first to postulate that emotional processing is based on an interconnected cortical-subcortical system, for which later the term ‘Papez circuit’ was coined. The idea of a ‘limbic system’, defined as a functional entity responsible for all emotional experience and emotional expressions, was subsequently put forward by Paul MacLean in the early 1950s. Neuroanatomically, the ‘limbic system’ was associated with neural structures surrounding the midbrain (MacLean, 1993). A different view on the neuroanatomy of emotions emerged during the 1970s, emphasizing the role of the right hemisphere in emotion processing (Schwartz et al., 1975). However, neither of these conceptual frameworks, dominating the clinical neurosciences well into the mid-1990s, allow for selective impairments of specific emotions.

In 1994, Adolphs and colleagues reported an intriguing finding, which changed the way we have to think about how emotions are organized within the brain (Adolphs et al., 1994). They investigated a person suffering from Urbach–Wiethe disease, a rare hereditary disorder which causes bilateral calcifications of the medial temporal lobes, affecting particularly the amygdala. In this person, a selective deficit in recognizing fearful faces was found, while recognition of other basic emotions such as happiness, surprise, sadness, disgust and anger was preserved. The prominent role of the amygdala in processing fear related information was later substantiated in a number of subsequent studies investigating emotion processing in Huntington’s disease and other basal ganglia disorders (e.g. Suzuki et al., 2006 for Parkinson’s disease; Corcoran et al., 2007 for obsessive compulsive disorder), as well as studies looking for the neural substrate of disgust processing.

Wang and colleagues reported a disproportionate impairment in disgust recognition in Chinese people suffering from Huntington’s disease (Wang et al., 2003), a finding very similar to that reported by Sprengelmeyer and colleagues in 1996. Montagne and colleagues investigated facial expression recognition in early symptomatic Huntington’s disease and found a specific impairment in recognizing anger and disgust (Montagne et al., 2006). Other studies suggest that not only the recognition of facial signals depicting disgust can be impaired in the more advanced stages of Huntington’s disease, but also the experience of this emotion. Unpleasant odours were rated as significantly less disgusting by people with Huntington’s disease than by control participants in a study by Mitchell and colleagues (Mitchell et al., 2005) and Hayes and colleagues (Hayes et al., 2007). The latter also showed that recognition of vocal stimuli depicting disgust as well as lexical and semantic aspects of disgust processing can be affected in Huntington’s disease.

However, results are not always unequivocal. For example, the attempt of Milder’s and colleagues to show a selective or disproportionate disgust recognition deficit in Huntington’s disease failed. Instead, they reported an overall impairment in facial expression recognition, with fear and not disgust as the most affected emotion (Milders et al., 2003). They also included Huntington’s disease gene carriers in the pre-clinical stages of the disorder for which they could not show a deficit in facial expression recognition at all.
These results are in stark contrast to a study by Johnson and colleagues published in this issue of Brain. The authors form part of the Predict-HD Study Group, which aims to investigate early signs of Huntington’s disease. Amongst other dimensions, the ability to recognize facial expressions of emotions was identified as a possible early marker for the disease. The sheer number of more than 500 participants included in this study is impressive. Participants were divided in four different groups (group 1 = no clinical signs, group 2 = non-specific signs, group 3 = possible signs of Huntington’s disease and group 4 = likely signs of Huntington’s disease). There was no selective deficit evident, but recognition of all negative emotions was significantly impaired, and this impairment was more pronounced in the groups in which the onset of the disease can be expected in the near future. This is an interesting finding of possible clinical importance. Follow up studies now have to establish whether deficits in emotion recognition are of prognostic value to estimate the onset of the disease in individuals.

There are other studies based on much smaller numbers, but methodologically better controlled, which report circumscribed deficits in emotion recognition in pre-clinical Huntington’s disease. Gray and colleagues used a double blind design, in which the assessment of facial expression recognition of possible Huntington’s disease gene carriers took place prior to genetic testing (Gray et al., 1997). The same design, but with a more sophisticated set of neuropsychological tests was adopted by Sprengelmeyer and colleagues (Sprengelmeyer et al., 2006). Both studies found selective deficits in facial disgust recognition.

Hennenlotter and colleagues found deficits in disgust recognition in a small sample of pre-clinical Huntington’s disease patients (Hennenlotter et al., 2004) and, in a functional imaging study, reduced responses to disgusted looking faces in the insula cortex. Other studies on healthy participants (Phillips et al., 1997; Sprengelmeyer et al., 1998) and lesion studies (Calder et al., 2000) also point to the insula cortex as the neural site most important for disgust processing. A functional imaging study by Wicker and colleagues even suggests a mirror neuron system within the insula cortex as the neural site most important for disgust processing. The common neural basis of seeing and feeling disgust (Wicker et al., 2003). Given the likely role of the insula in mediating disgust, it is not surprising that Johnson and colleagues, who performed volumetric measures of the caudate nucleus and the putamen, found no correlation between these measures and deficits in emotion recognition.

Research in the past 10 years has helped to change the way we think about how emotions are organized within the brain. Instead of assuming a functional unit handling all emotions, it is now very possible that a set of relatively independent neural systems might exist, each handling a specific basic emotion and all working together in a concerted way. This raises many exciting questions regarding the evolutionary age of these systems, as well as their biochemical, anatomical and functional architecture. A number of disorders are now known to be associated with severe impairments in recognizing facial expression of emotions. These are important clinical findings. Future studies have to establish if and how these deficits impact on the everyday life of patients and their families. If so, the ability to recognize facial expression of emotions should be incorporated into routine neuropsychological assessments of these disorders and neuropsychological compensation strategies to address this deficit must be developed.

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