

Emotions and the brain: linking affective disorders to brain regions

If you respond to the question “what is apathy?” by saying “I don’t know and I don’t care”, you’ve got the correct answer. But if you didn’t get this joke, you may be suffering from impaired humour appreciation, according to research presented at this year’s Rotman Research Institute Conference (Toronto, ON, Canada; March 25–26). Apathy is sometimes jokingly referred to as “the get-up-and-go that got up and went”, Robert van Reekum (University of Toronto, ON, Canada) told delegates. Nevertheless, apathy is important both clinically and for research purposes, he argued, because it contributes to a number of adverse outcomes across a number of ailments. The prevalence of apathy is around 60% in patients with traumatic brain injury and in outpatients with Alzheimer’s disease—it is even higher among nursing-home residents. However, somewhat surprisingly perhaps, only about 50% of depressed patients suffer from apathy.

According to van Reekum, apathy has been associated with neurological dysfunction in limbic and frontal subcortical regions, and reduced activity in the anterior cingulate has been implicated as a causative factor by three separate studies. There is some evidence that people who have a stroke or develop Alzheimer’s disease become more apathetic as they age. “But otherwise there is a weak correlation between age and apathy, so apathy is not an inevitable consequence of ageing”, he noted.

While ageing may not necessarily lead to apathy, getting old does seem to result in some impairment of humour appreciation, according to a recent study by Donald Stuss (University of Toronto, ON, Canada) and colleagues. The researchers compared the performance of 20 elderly participants (mean age 73 years) with that of 17 younger individuals (mean age 29 years) on verbal and nonverbal tests of humour. They found that the elderly participants made significantly more errors in the “joke completion test” and in the “cartoon appreciation task”, suggesting that they had some trouble

understanding humour. The errors made by the older participants were similar to errors made by patients with right frontal lobe lesions in a previous study. Based on that study, the investigators concluded that “damage to the right frontal lobe impairs the ability to appreciate humour and to demonstrate humorous reactions”.



60% of patients with Alzheimer’s are apathetic

Right hemisphere damage also appears to be detrimental to emotional communication, according to Kenneth Heilman (University of Florida, Gainesville, FL, USA). Many neurologists believe that the left hemisphere is dominant for communication, but, according to Heilman, this is only true for verbal communication. For most people, the right hemisphere is dominant for emotional communication. A large number of case studies show that “right-hemisphere-damaged patients can’t understand emotional communication”, he explained. Patients with right-hemisphere damage (especially in the temporal-parietal region) appear to lose their ability to understand facial expressions and the tone or “emotional prosody” of voices.

Another part of the brain that is very important for emotional communication is the orbitofrontal cortex. According to Edmund Rolls (University of Oxford, UK), the orbitofrontal cortex plays a crucial role in decoding reinforcers, a function which is important for understanding emotional expression. Reinforcers, both positive and negative, are represented in the orbitofrontal cortex, and facial expressions can be primary reinforcers, he said. Recent studies by Rolls and colleagues have found that patients who had part of their

orbitofrontal cortex removed because of tumours, had difficulty in understanding emotional communication. Patients who have undergone surgery show that “bilateral damage to the orbitofrontal cortex can impair face expression recognition”, Rolls told delegates. And even unilateral damage “can be sufficient in some patients to produce deficits in voice expression identification”, he added. Rolls’ team also found that damage to the anterior cingulate cortex can impair voice expression identification.

According to Helen Mayberg (University of Toronto, ON Canada), some parts of the cingulate cortex also play a key role in the development of depression. In an effort to identify “depression circuits” in the brain, Mayberg and colleagues looked at the effects of fluoxetine on depressed inpatients, following six weeks of treatment, in a double-blind, randomised, controlled trial. Functional neuroimaging of patients who responded to the treatment showed evidence of altered activity in several different brain regions. The changes included “subgenual cingulate suppression, cortical normalisation, and suppression of hippocampal metabolism”, Mayberg said. Based on her research, she questioned whether it was possible to implicate specific neurological structures as being responsible for the development of depression. Instead, she concluded that what seems to be important is the interaction of—or a balance between—different brain regions.

Nevertheless, even if neuroscience cannot yet identify distinct neurological structures and processes behind every single emotion or emotional disorder, it is becoming increasingly clear that there are physiological correlates to all behaviour—emotional or not. And in light of all the evidence presented at the conference in this regard, one must wonder whether it makes any sense at all to distinguish between so-called “mental disorders” on the one hand and “physical disorders” on the other.

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