IN THE NEWS

The truth about lies

A functional MRI study that looked at brain activity in people who were lying has been hailed as the next generation of polygraph.

Scott Faro, from the Temple University, Philadelphia, USA, presented the work at the Radiological Society of North America. According to the *Daily Telegraph* (30 November 2004), "there were more areas of the brain activated during the deception process — seven in all — compared with four activated by truth-telling."

The authors interpret these results as showing that lying takes more effort than telling the truth. But according to *ABC.net* (30 November 2004), "The study not only sheds light on what goes on when people lie, but may also provide new technology for lie detecting."

Traditional polygraphs measure physical changes, such as increased sweating, that are associated with lying. However, "the accuracy is limited because people who are telling the truth can show similar changes merely as a result of being anxious," says Faro (*BBC Online*, 30 November 2004). "Furthermore, those adept at lying can learn how to cheat the polygraph."

By contrast, it is possible that fMRI scans would be a more reliable (albeit a more expensive and perhaps less practical) way to detect liars. Richard Wiseman, from the University of Hertfordshire, UK, told the BBC, "I'm sure it would be better than the polygraph." However, the days when every police station has an MRI scanner might be a long way off yet.

Rachel Jones

INFECTIOUS DISEASES

Gaining entry to the CNS

The human JC polyomavirus (JCV) is estimated to be present in 70–80% of the adult population, and in healthy people it is generally tolerated without ill effects. However, in immunocompromised individuals — in particular, patients with AIDS — it can cause progressive multifocal leukoencephalopathy (PML), a fatal demyelinating disease. At present, this devastating condition cannot be treated, but a recent report in *Science* indicates that serotonin (5-hydroxytryptamine or 5-HT) receptor antagonists could provide a new therapeutic approach.

PML results from infection of oligodendrocytes by JCV, so how does the virus gain entry to these cells? From a series of *in vitro* experiments, Elphick and colleagues obtained several pieces of evidence to indicate that it latches on to 5-HT receptors. It has previously been shown that glia can be rendered resistant to JCV infection by certain drugs of the serotonin–dopamine inhibitor (SDI) class. Elphick *et al.* showed that dopamine-receptor-specific drugs were considerably less effective at blocking JCV infection than drugs that inhibited both dopamine and 5-HT receptors, so they focused their attention on the 5-HT receptors.

The authors found that JCV infection of glial cells could be inhibited by 5-HT_{2A}-receptor (5-HT_{2A}R) antagonists or anti-5-HT_{2A}R monoclonal antibodies. In the reverse experiment, they asked whether 5-HT_{2A}R could confer susceptibility to JCV infection on cells that do not normally express this receptor. The 5-HT_{2A}R-negative HeLa cell line is usually resistant to JCV infection, but when these cells were transfected with a 5-HT_{2A}R-expressing construct, they could be readily infected by JCV.

Further evidence that JCV uses 5-HT_{2A}R as an entry point came from the observation that the internalized viral particles co-localized with 5-HT_{2A}R in glial cells. However, 5-HT receptors are not the only factors that determine a cell's vulnerability to JCV infection. For example, 5-HT receptors are widely expressed in neurons, yet these cells do not normally become infected by JCV. It was previously shown that α2,6-linked sialic acid is an important component of the JCV receptor, and this monosaccharide is displayed on the surface of glia but not neurons. HeLa cells express α2,6-linked sialic acid but not 5-HT receptors, so they can bind JCV but cannot internalize the virus. So, a model for JCV infection can be envisaged, in which the virus is captured on the cell surface by α2,6-linked sialic acid, and is subsequently internalized through an interaction with 5-HT receptors.

5-HT-receptor antagonists are already used to treat many neurological and psychiatric disorders, so if these new *in vitro* findings can be recapitulated *in vivo*, these drugs could represent a ready-made source of therapeutic agents for treating PML. Elphick *et al.* suggest two approaches: the drugs could be used to prevent JCV from entering the CNS in HIV-infected individuals, or to limit further demyelination in patients who have already developed PML.

Heather Wood

References and links

ORIGINAL RESEARCH PAPER Elphick, G. F. *et al.* The human polyomavirus, JCV, uses serotonin receptors to infect cells. *Science* **306**, 1380–1383 (2004)

FURTHER READING Eash, S. *et al.* Differential distribution of the JC virus receptor-type sialic acid in normal human tissues. *Am. J. Pathol.* **164**, 419–428 (2004)

WEB SITE

Encyclopedia of Life Sciences: http://www.els.net/polyomaviruses