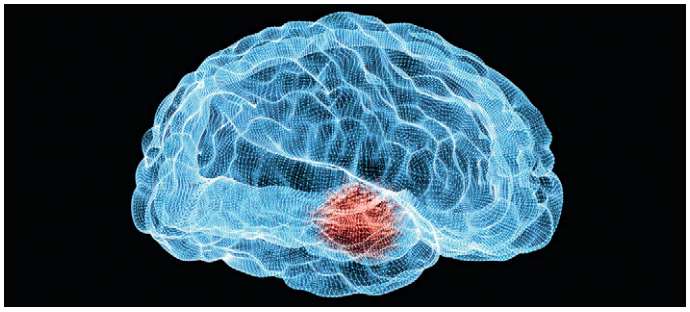
Harrison, 5, who has autism, plays with Kaspar, a child-sized humanoid robot developed at the University of Hertfordshire in the UK to interact and help improve the lives of children with autism and other communication difficulties. Read more about Kaspar at www.herts.ac.uk/kaspar.

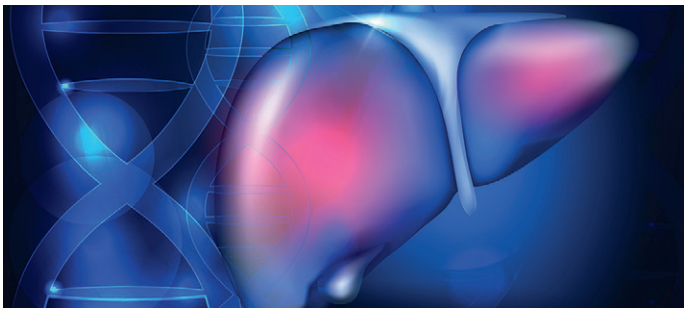
Photo: Matthew Stock/Reuters/Picture Media



Reprogramming brain cells may help Parkinson's

Cells similar to dopamine neurons can be induced by treating non-neuronal brain cells with a specific combination of DNA transcription factors, according to a study published in *Nature Biotechnology*. The new reprogramming method has been demonstrated both in cultured human cells and in a mouse model of Parkinson's disease. A defining feature of Parkinson's disease is the progressive death of a specific group of neurons that secrete dopamine. Although several treatments are available to patients, including the chemical precursor of dopamine, none change the course of the disease. A decades-long research effort has sought to develop a disease-modifying therapy in which dopamine neurons or their precursors would be generated in the laboratory and transplanted into the brain. The authors of this study described a different approach to cell replacement that does not require cell transplantation. By testing a number of genes known to be important for dopamine neuron identity, they identified three transcription factors and a microRNA that reprogrammed human brain cells called astrocytes as cells that resemble dopamine neurons. The authors used a toxin to kill dopamine neurons in mice and then delivered the genes for the four factors to the brain using a system designed to express the genes only in astrocytes. Some astrocytes were successfully reprogrammed, acquiring characteristics of dopamine neurons, correcting several behavioral symptoms caused by dopamine neuron loss. Substantial further research would be needed before this approach could be considered for human trials, the authors noted.

http://dx.doi.org/10.1038/nbt.3835



Protein that causes liver disease found

Australian scientists have published research in *Nature Genetics* which identifies that variations in the interferon lambda 3 (IFNL3) protein are responsible for tissue damage in the liver, paving the way for new treatments to be developed. The international team, led by Professor Jacob George and Dr Mohammed Eslam at the Westmead Institute, had previously identified that the common genetic variations associated with liver fibrosis were located on chromosome 19 between the IFNL3 and IFNL4 genes. The team analysed liver samples from 2000 patients with hepatitis C, using state-of-the art genetic and functional analysis, to determine the specific IFNL protein responsible for liver fibrosis. The team demonstrated that there was increased migration of inflammatory cells from blood to the liver following injury, increasing IFNL3 secretion and liver damage. Notably, this response was determined to a great extent by an individual's genetic makeup. "We have designed a diagnostic tool based on our discoveries, which is freely available for all doctors to use, to aid in predicting liver fibrosis risk," Prof Jacob said. "This test will help to determine whether an individual is at high risk of developing liver fibrosis, or whether a patient's liver disease will progress rapidly or slowly, based on their genetic makeup." The research team will now extend their work to further understand the fundamental mechanisms of how IFNL3 contributes to liver disease progression and to translate these discoveries into new therapeutic treatments.

http://dx.doi.org/10.1038/ng.3836

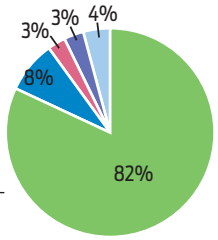
Cate Swannell doi: 10.5694/mja17.n0105

MJA InSight Poll

The Medicare rebate freeze must be lifted in the Federal Budget on 9 May 2017.

Total votes = 159

- Strongly agree
- Agree
- Neutral
- Disagree
- Strongly disagree



Take part in next week's poll on: www.mja.com.au/insight

MJA Podcasts

Dr Yash Gawarikar is a consultant neurologist and director of the Acute Stroke Service at Calvary Hospital in Canberra. He discusses clot retrieval and acute stroke care to accompany his co-authored perspective in this issue.

Professor Jeffrey Rosenfeld AM, OBE, is director of the Monash Institute of Medical Engineering, and is a senior neurosurgeon in the Department of Neurosurgery at the Alfred Hospital in Melbourne. His co-authored narrative review of neurobionics and the brain–computer interface appears in this issue.

Dr Thanuja Dharmadasa is a neurologist specialising in motor neurone disease, and is based at the Brain and Mind Centre at the University of Sydney. She recently returned from the University of Oxford in the UK and discusses her co-authored narrative review of motor neurone disease published in this issue.

Podcasts are available at www.mja.com.au/multimedia/podcasts and from iTunes. Also available as videos at www.mja.com.au/multimedia

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