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IN BRIEF

NEURODEGENERATIVE DISEASE

Towards transplant therapy

The long-term safety and efficacy of using induced pluripotent stem cell (iPSC)-derived neurons to replace midbrain dopaminergic neurons lost in Parkinson disease (PD) have not been tested in primates. Kikuchi *et al.* grafted human iPSC-derived midbrain dopaminergic progenitors into the putamen of macaques that had been treated with the toxin MPTP (which ablates nigral dopaminergic neurons). Three or four monkeys per group received vehicle or cells derived from healthy controls or individuals with PD. Cell transplants markedly improved neurological and movement scores at 12 months, and imaging and histological analyses confirmed that the grafts were tumour-free after as long as 24 months, supporting the utility and safety of this cell-based therapy.

ORIGINAL ARTICLE Kikuchi, T. *et al.* Human iPS cell-derived dopaminergic neurons function in a primate Parkinson's disease model. *Nature* **548**, 592–596 (2017)

VISUAL PROCESSING

Face off

How face patches — areas of cortex that process visual faces — develop is not known. Livingstone and colleagues reared three macaques for ~200 days without any visual exposure to faces and used functional MRI to measure neural responses to images of faces, objects and hands. Face-deprived monkeys did not develop normal face patches but showed stronger neural and behavioural selectivity for images of hands than did control monkeys, suggesting that visual experience of faces is necessary for the proper development of face patches.

ORIGINAL ARTICLE Arcaro, M. J. *et al.* Seeing faces is necessary for face-domain formation. *Nat. Neurosci.* http://dx.doi.org/10.1038/nn.4635 (2017)

TECHNIQUES

Having a field day

Recording magnetic fields associated with neuronal activity could have certain advantages over voltage recordings, but current probes to detect such fields are too large for *in vivo* use. Caruso *et al.* developed micron-scale 'magnetrodes' and used these in the cat visual cortex to show that the amplitudes of visually evoked event-related fields are on the order of several nanoteslas, thus providing proof of concept for intracortical magnetic field recordings.

ORIGINAL ARTICLE Caruso, L. *et al. In vivo* magnetic recording of neuronal activity. *Neuron* http://dx.doi.org/10.1016/j.neuron.2017.08.012 (2017)

NEURODEVELOPMENTAL DISORDERS

A transcription-targeting target

Fragile X mental retardation protein (FMRP) represses the transcription of many target genes, including genes that encode chromatin-associated proteins. Loss of FMRP leads to fragile X syndrome (FXS); however, whether misregulation of chromatin-associated proteins contributes to FXS is unclear. Korb *et al.* showed that neurons from mice that lack FMRP have increased levels of several chromatin-associated proteins, including bromodomain-containing protein 4 (BRD4). Inhibition of BRD4 using the small molecule JQ1 reversed many of the gene expression changes observed in the FMRP-null neurons, and rescued memory and social deficits in FXS mice.

ORIGINAL ARTICLE Korb, E. *et al.* Excess translation of epigenetic rgulators contributes to fragile X syndrome and is alleviated by Brd4 inhibition. *Cell* http://dx.doi.org/10.1016/j.cell.2017.07.033 (2017)