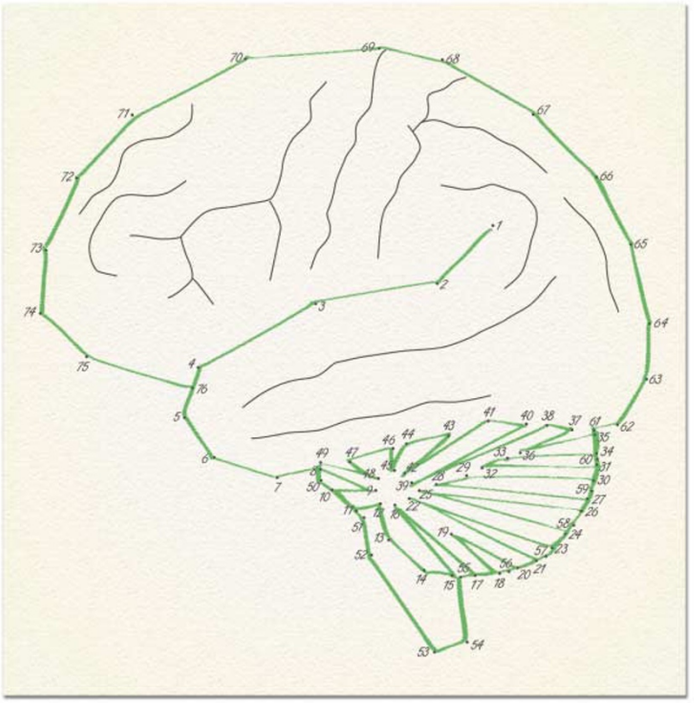

precise identification of labelled cells and quantification of their projections and geometry, should make it easier to address this question, as well as others relating to the functional anatomy of cortical connections.

Rachel Jones

References and links

ORIGINAL RESEARCH PAPER Stettler, D. D. *et al*. Lateral connectivity and contextual interactions in macaque primary visual cortex. *Neuron* **36**, 739–750 (2002)

WEB SITES

Gilbert's laboratory: http://www.rockefeller.edu/labheads/gilbert/gilbert.html

nature of this functional relationship.

The authors found that Shh upregulates *Nmyc* expression in cerebellar granule neuron precursors (CGNPs) *in vitro*. Using transgenic embryos, they showed that *Nmyc* is upregulated ectopically in the dorsal spinal cord when Shh expression is driven by a *Wnt1* regulatory element. Two further observations indicated that *Nmyc* acts downstream of Shh. Overexpression of *Nmyc* activated G_1 cyclins in CGNPs and stimulated ongoing proliferation, independent of Shh activity. Shh also promoted CGNP proliferation, but its potency was reduced by the addition of a Myc antagonist.

These findings indicate that *Nmyc* is a direct downstream target of Shh that mediates its effects on cell proliferation during cerebellar development and in medulloblastomas. The upregulation of *Nmyc* by Shh seems to be context-dependent, as *Nmyc* is expressed in the cerebellar granule cell precursor layer, but not in the floorplate, which is another key site of Shh expression. This finding represents a significant step in piecing together a signalling pathway that is important to both neuroscientists and cancer biologists.

Heather Wood

References and links

ORIGINAL RESEARCH PAPERS Kenney, A. M. *et al*. *Nmyc* upregulation by sonic hedgehog signaling promotes proliferation in developing cerebellar granule neuron precursors. *Development* **130**, 15–28 (2002)

FURTHER READING Knoepfler, P. S. *et al*. N-myc is essential during neurogenesis for the rapid expansion of progenitor cell populations and the inhibition of neuronal differentation. *Genes Dev*. **16**, 2699–2712 (2002) | Berman, D. M. *et al*. Medulloblastoma growth inhibition by hedgehog pathway blockade. *Science* **297**, 1559–1561 (2002)

IN BRIEF

FUNCTIONAL IMAGING

The neural correlates of feeling sympathy.

Decety, J. & Chaminade, T. *Neuropsychologia* **41**, 127–138 (2003)

The authors use functional imaging to show that watching actors telling sad stories leads to activation of neural structures that are involved in processing emotion. It also activates a 'shared motor representation' network that includes the right inferior parietal cortex, and which is normally activated by both observed and executed actions. If the actors used emotive gestures and facial expressions, there was also a specific increase in activity in the left inferior frontal gyrus. These areas are proposed to form a network that is important for feeling sympathy.

CIRCADIAN RHYTHMS

Rhythmic histone acetylation underlies transcription in the mammalian circadian clock.

Etchegaray, J.-P. *et al. Nature* 11 December 2002 (doi:10.1038/nature01304)

In mammals, the circadian clock is controlled by a transcriptional feedback loop. Clock and Bmal1 drive the expression of *Per1–3*, *Cry1* and *Cry2*, and the Cry proteins in turn block Clock/Bmal1-mediated transcription. Here, Etchegaray *et al*. show that in the mouse liver, the *Per1*, *Per2* and *Cry1* gene promoters exhibit oscillations in histone acetylation that are synchronized with circadian rhythms. The data imply a new role for chromatin rearrangements in circadian rhythmicity.

NEUROTROPHIC FACTORS

Brain-derived neurotrophic factor can act as a pronecrotic factor through transcriptional and translational activation of NADPH oxidase.

Kim, S. H. *et al*. *J. Cell Biol.* 2 December 2002 (doi:10.1083/jcb.200112131)

As brain-derived neurotrophic factor (BDNF) can cause cell death, Kim *et al*. set out to explore the underlying mechanisms. They found that BDNF-induced neuronal death depended on the production of free radicals and the synthesis of new proteins. Microarray analysis revealed that BDNF increased the synthesis of a subunit of NADPH oxidase. The authors obtained evidence for the involvement of this enzyme in the death process, as blocking its activity prevented BDNF toxicity, but not the antiapoptotic action of this neurotrophin.

STEM CELLS

A nucleolar mechanism controlling cell proliferation in stem cells and cancer cells.

Tsai, R. Y. L. & McKay, R. D. G. *Genes Dev*. **16**, 2991–3003 (2002)

The authors identify nucleostemin as a nucleolar protein that is expressed in neuronal and other stem cells, as well as in cancer cells. When stem cells differentiate, the expression of nucleostemin decreases before they exit the cell cycle. Increasing or decreasing the levels of nucleostemin reduces stem cell proliferation, leading to apoptosis. The data point to a new nucleolar mechanism that controls the progression of stem cells through the cell cycle.