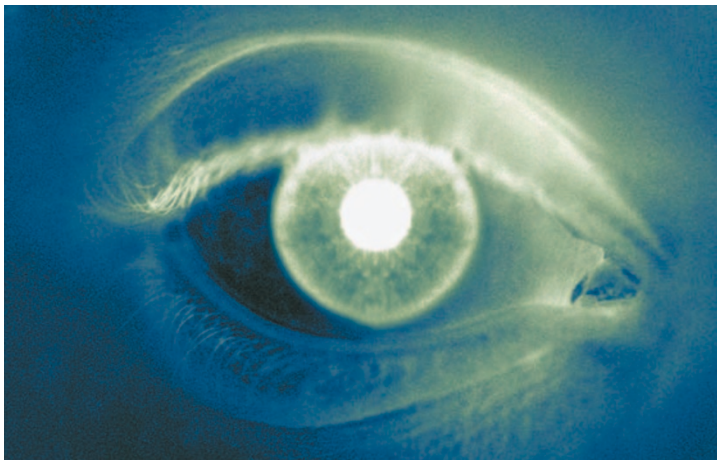

DEVELOPMENT

Determining eyedentity

The development of the CNS depends on regionally-specific progenitors acquiring and maintaining neural potential. But what are the signals that control this process? Reporting in *Neuron*, Vetter and colleagues present compelling evidence that Frizzled 5 (Fz5) regulates the neural potential of progenitors and their proliferation in the developing *Xenopus laevis* retina.

During retinal development, specific signalling mechanisms are thought to underlie important regional differences in progenitor proliferation and differentiation. The rate of progenitor proliferation increases to provide sufficient numbers of cells for neural differentiation. Progenitors then trigger the expression of factors that are required for retinal neurogenesis. *Sox2*, a Group B1 *Sox* gene, is thought to be involved in neurogenesis in the developing retina as it is specifically expressed in this region. However, until now, the signals that control *Sox2* expression have not been identified. Previous work has implicated various components of the Wnt/Frizzled signalling pathway — including the transmembrane receptor Fz5 — in retinal development, which indicates that this pathway might be a good starting point for determining the mechanisms involved in signalling neural potential.

Vetter and colleagues report that blocking Fz5 in *X. laevis* results in lower proliferation rates of progenitors, lack of expression of the proneural genes that are required for neurogenesis and a bias towards the non-neural fate of Müller glial cells in the developing retina. The action of Fz5, which was mediated through the canonical Wnt/β-catenin signalling pathway, did not directly influence the expression of progenital markers in the retina. Instead, the effects of blocking Fz5 resulted from a reduction in the expression of its downstream effector, *Sox2*, at the stage of optical vesicle formation, as inhibition of *Sox2* produced similar effects to inhibition of Fz5. These results therefore indicate that both Fz5 and *Sox2* are crucial for determining neural potential and the proliferation of progenitors.

As the authors predict, it is likely that further signalling mechanisms that influence the expression of *Sox2*, and additional factors necessary for progenitor proliferation, will be identified in future studies. Nevertheless, these results mark an important step in the identification of the molecular mechanisms that underlie the acquisition of neural potential in the developing CNS.

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References and links

ORIGINAL RESEARCH PAPER Van Raay, T. J. *et al.* Frizzled 5 signaling governs the neural potential of progenitors in the developing *Xenopus* retina. *Neuron* **46**, 23–36 (2005)

FURTHER READING Dyer, M. A. & Cepko, C. L. Regulating proliferation during retinal development. *Nature Rev. Neurosci.* **2**, 333–342 (2001)

IN BRIEF

ELECTROENCEPHALOGRAPHY

Alpha phase synchronization predicts P1 and N1 latency and amplitude size.

Gruber, W. R. *et al. Cereb. Cortex* **15**, 371–377 (2005)

There are two possible mechanisms for the generation of event-related evoked potentials (ERPs) — phase resetting of existing oscillations in the brain, or the imposition of stimulus-locked activity on random oscillations. Gruber and colleagues used several methods of analysis and found that the ERP components P1 and N1 are probably produced by phase resetting and synchronization between frequencies.

NEURODEVELOPMENT

A centrosomal mechanism involving CDK5RAP2 and CENPJ controls brain size.

Bond, J. *et al. Nature Genet.* **37**, 353–355 (2005)

Autosomal recessive primary microcephaly (MCPH) causes a reduction in the size of the cerebral cortex with reduced cognitive functions. The authors describe two mutations that can cause this disorder, in the genes *CDK5RAP2* and *CENPJ*. In mouse embryos, these genes are expressed in the developing neuroepithelium and are localized at the centrosome during mitosis, which indicates that MCPH might be caused by an abnormality in the number of neurons generated that results from a centrosomal mechanism.

NEURAL CODING

Identification of network-level coding units for real-time representation of episodic experiences in the hippocampus.

Lin, L. *et al. Proc. Natl Acad. Sci. USA* **102**, 6125–6130 (2005)

Individual neurons in the brain show considerable variability in their responses to stimuli, and this variability makes it difficult to investigate how experiences are encoded in the brain. Lin and colleagues used a 96-channel array to record from neurons in the mouse hippocampus during episodes of 'startling', and identified network-level functional coding units that can represent these experiences in real time. The neurons within these units show 'co-spiking' dynamics that allow the variability of individual responses to be overcome.

GLIA

Resting microglial cells are highly dynamic surveillants of brain parenchyma *in vivo*.

Nimmerjahn, A. *et al. Science* 14 April 2005 (10.1126/science.1110647)

The authors used *in vivo* two-photon microscopy to study microglia in the uninjured neocortex of mutant mice that showed specific expression of enhanced green fluorescent protein in brain microglia. They found that 'resting' microglia, far from being dormant, are highly active. The cells extend motile protrusions into the surrounding environment and respond immediately to disruption of the blood–brain barrier with local activation and a switch to a 'shielding' role.