IN BRIEF

SENSORY SYSTEMS

A key role of starburst amacrine cells in originating retinal directional selectivity and optokinetic eye movement.

Yoshida, K. *et al. Neuron* **30**, 771–780 (2001)

Direction-selective ganglion cells in the retina respond to stimuli that are moving in one, but not in the opposite, direction. Yoshida *et al.* eliminated another type of retinal neuron — the starburst amacrine cells — and found that direction selectivity of ganglion cells was abolished. The optokinetic eye reflex that is normally elicited by moving stimuli was also abolished, indicating that starburst cells are crucial for detecting direction of movement.

DEVELOPMENT

Synergy between *Hoxa1* and *Hoxb1*: the relationship between arch patterning and the generation of cranial neural crest.

Gavalas, A. *et al. Development* **128**, 3017–3027 (2001)

The *Hox* genes are well known for their roles in neural crest patterning. Gavalas *et al.* now propose an additional role in neural crest generation. In mouse embryos mutant for *Hoxa1* and *Hoxb1* in the ectoderm, rhombomere 4 (r4) was reduced in size and failed to generate neural crest. Although r4 crest cells normally migrate to the second pharyngeal arch, its early patterning was unaffected, indicating that this does not depend on neural crest.

GLIA

Spontaneous astrocytic Ca^{2+} oscillations *in situ* drive NMDA receptor-mediated neuronal excitation.

Parri, H. R. *et al. Nature Neurosci.* **4**, 803–812 (2001)

The idea that glial cells can directly affect synaptic activity has received significant support. But in most cases, glial cells act as reactive elements; synaptic activity triggers a response in astrocytes, which then release transmitters that act back on the synapse. Parry *et al.* show that spontaneous Ca^{2+} waves among astrocytes can trigger NMDA (*N*-methyl-D-aspartate)-receptor-mediated responses in neighbouring neurons, providing evidence that glial cells do not solely react to synaptic activity, but can also participate directly in its generation.

NEURODEGENERATIVE DISORDERS

Tauopathy in *Drosophila*: neurodegeneration without neurofibrillary tangles.

Wittmann, C. W. *et al. Science* 14 June 2001 (10.1126/science.1062382)

Tau is a microtubule-binding protein that has been implicated in Alzheimer's disease. The authors found that expressing human tau in *Drosophila* caused neurodegeneration, which shared some of the features seen in humans. However, neurofibrillary tangles, a key characteristic of Alzheimer's disease, were absent in the fly, indicating that tangle formation might not be directly related to cell death. This genetic model might allow the identification of suppressors and enhancers of neurodegeneration.

SENSORY SYSTEMS

The two sides of a painful story

The sensation of pain not only involves the perception of basic stimulus parameters, such as intensity, but it also has an affective dimension — how unpleasant a painful stimulus feels. How does the brain distinguish between these components? Where are they processed? Three recent papers shed new light on these questions.

Johansen *et al.* injected formalin into the paws of rats and placed them in a specific environment to test whether they could learn to avoid it (conditioned place avoidance). The authors reasoned that the acquisition of place avoidance would reflect the affective component of pain, whereas the acute behavioural responses (lifting and licking the injected paw) would correspond to the sensory dimension of the stimulus. Using this model, they found that destruction of the anterior cingulate cortex (ACC), a structure implicated in the affective representation of pain in humans, prevented the rats from acquiring place avoidance, but did not affect the acute behavioural responses.

If ACC is important for the affective side of pain, where is the sensory dimension represented? Hofbauer *et al.* addressed this question by exposing subjects under hypnosis to painful stimuli, and they used positron emission tomography to measure cerebral activity. During hypnosis, the authors prompted the subjects to change their perception of pain intensity, but not of its affective properties. In contrast to changes in pain affect, which are known to engage the ACC, changes in pain intensity correlated with activity in the primary somatosensory cortex.

Zubieta *et al.* found a similar double dissociation while performing imaging experiments to evaluate the function of μ-opioid receptors during sustained pain. They found that the degree of activation of this system in the nucleus accumbens and amygdala correlated with sensory ratings of the pain experience, whereas opioid activation in the ACC correlated with affective ratings. So, it seems that two complementary brain systems process the two faces of pain, although we experience pain as a unitary phenomenon. A colossal challenge in the field is to understand how the two systems interact to generate this unity.

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References and links

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FURTHER READING Hunt, S. P. & Mantyh, P. W. The molecular dynamics of pain control. *Nature Rev. Neurosci.* **2**, 83–91 (2001) | Paus, T. Primate anterior cingulate cortex: where motor control, drive and cognition interface. *Nature Rev. Neurosci.* **2**, 417–424 (2001)

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