came the finding that the N^{Mcd} alleles produce truncated forms of Notch that lack an intracellular binding site for the Dishevelled protein, which can act as a Deltex antagonist. The authors also argue that Deltex and Su(H) antagonize one another, so the lateral inhibition and Deltex-mediated pathways cannot be active at the same time.

What are the implications of these findings? Ramain *et al.* suggest that the Deltex-dependent Notch signalling pathway might be active in all neuroectodermal cells early in development, perhaps preventing them from differentiating too early. For a cell to make a fate choice through lateral inhibition, it is necessary first to repress Deltex, and Dishevelled might be one of the factors responsible for this inhibition. This mechanism could be compared to releasing the handbrake on a car; although this allows the car to move, it does not determine where it will go. Similarly, repressing the Deltex-mediated pathway gives cells the freedom to choose their fate, but does not tell them what to become.

Heather Wood

References and links

ORIGINAL RESEARCH PAPER Ramain, P. *et al.* Novel Notch alleles reveal a Deltex-dependent pathway repressing neural fate. *Curr. Biol.* **11**, 1729–1738 (2001)

FURTHER READING

Artavanis-Tsakonas, S. *et al*. Notch signaling: cell fate control and signal integration in development. *Science* **284**, 770–776 (1999)

colonies in which ants have the *b* allele are polygynous.

Krieger and Ross have now sequenced *Gp-9* and found that it encodes a pheromone-binding protein. These proteins are crucial for chemical communication in insects — they transport odorant molecules from pores in the cuticle to receptors on sensory neurons. Worker ants control the number of egg-laying queens in their colony by recognizing their chemical signatures — carried by pheromones — and either accepting or destroying each queen. So the pheromone-binding protein encoded by *Gp-9* could be involved in the recognition of the chemical signature of queens by workers. Moreover, the nine-nucleotide difference between the two alleles could alter the ability of workers to recognize queens.

This study is the first to identify a single gene with such a fundamental and far-reaching effect on a complex social behaviour. The existence of such a gene is cause for hope that by combining genetic, behavioural and neurobiological techniques, it should be possible to elucidate details of the complete pathway by which queen number in fire ant colonies is controlled, from the gene to the behaviour. Study of this gene will also, as Krieger and Ross point out, allow greater understanding of the evolution of social behaviour.

Rachel Jones

References and links

ORIGINAL RESEARCH PAPER Krieger, M. J. B. & Ross, K. G. Identification of a major gene regulating complex social behaviour. *Science* 15 November 2001 (10.1126/science.1065247)

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IN BRIEF

NEUROLOGICAL DISORDERS

Structure of human monoamine oxidase B, a drug target for the treatment of neurological disorders.

Binda, C. *et al. Nature Struct. Biol.* 26 November 2001 (10.1038/nsb732)

Monoamine oxidase B (MAO-B) hydrolyses catecholamines and is a common target of drugs for Parkinson's disease and depression. The authors solved the structure of MAO-B, determined the properties of its active site, and identified the residues involved in substrate recognition. Knowledge of this structure will lead to insights into the catalytic mechanism of MAO-B, explain the differences between MAO-B and MAO-A, and guide the development of MAO-B-specific drugs.

NEUROLOGICAL DISORDERS

Endogenous β-amyloid production in presenilin-deficient embryonic mouse fibroblasts.

Armogida, M. *et al. Nature Cell Biol.* **3**, 1030–1033 (2001)

Recent findings have questioned the idea that the presenilins are responsible for γ-secretase activity and the production of β-amyloid peptides in Alzheimer's disease. This paper shows that fibroblasts that lack presenilins produce Aβ40 and Aβ42. By contrast, processing of Notch, which also depends on γ-secretase, was abolished in these cells. These findings point to the existence of additional γ-secretases that are distinct from the presenilins.

DEVELOPMENT

The *Drosophila* neuregulin Vein maintains glial survival during axon guidance in the CNS.

Hidalgo, A. *et al*. *Dev. Cell* **1**, 679–690 (2001)

Glial cells contribute to axonal pathfinding by providing guidance cues and trophic support. This paper shows that the relationship is reciprocal: *Drosophila* neurons promote glial survival by expressing a protein called Vein. Vein shows homology to neuregulin, a molecule known to promote glial survival in vertebrates. The authors provide evidence that Vein expression prevents glial apoptosis, and that loss of Vein function induces glial cell death.

SYNAPTIC PLASTICITY

Remodeling of synaptic actin induced by photoconductive stimulation.

Colicos, M. A. *et al. Cell* **107**, 605–616 (2001)

This paper provides compelling evidence that synaptic activity is accompanied by structural changes. The authors used photoconductive neuronal stimulation, in which cells are cultured on a silicon chip and light is applied to single neurons. As the conductivity of silicon changes in response to light, it is possible to pair local illumination with subthreshold electrical stimulation of the whole culture, and detect changes solely in the illuminated neuron. This approach allowed the authors to show that pre- and postsynaptic actin reorganizes transiently in response to a train of stimuli, whereas repeated trains lead to stable actin redistribution and the formation of new synapses.