## IN THE NEWS

**Turning left?**
Ultrasound scanning is generally considered to be a safe procedure for monitoring the health of unborn babies. However, research published recently in the journal *Epidemiology* casts doubt on this assumption by hinting that ultrasound might affect brain development.

The study, from the Karolinska Institute, indicated that boys who are scanned in the womb are significantly more likely to be left handed than those who are not scanned. This might not sound like a big handicap in itself, and the *Independent*'s (UK, 10 December) assertion that left handedness is "recognized as a mild form of brain damage" is sure to offend many left-handed people. However, the switch in handedness could indicate more serious problems. As the *Sunday Telegraph* (UK, 9 December) points out, "these people face a higher risk of conditions ranging from learning difficulties to epilepsy", although it is worth mentioning that no such adverse effects have yet been reported.

So, should expectant mothers think twice before having a scan? Maternity service campaigner Beverly Beech thinks so: "I am not sure that all the benefits of ultrasound scans outweigh the downsides. We should be advising women to think very, very carefully before they have scans" (*Sunday Telegraph*). The researchers, on the other hand, were keen to play down the risks. One team member, Juni Palmgren, said "I would urge people not to refuse ultrasound scanning as the risk of brain damage is only a possibility — but this is an interesting finding and needs to be taken seriously" (*BBC News*, 9 December).

Not everyone was pessimistic however. The web site *Anything Left-Handed* commented "if having ultrasound tests encourages left-handedness, that seems to us to be a GOOD THING!"

*Heather Wood*

SIGNAL TRANSDUCTION

# Top-Notch result

Notch signalling is probably best known for its pivotal role in neural cell fate choice through lateral inhibition. In the *Drosophila* neuroectoderm, for example, cells that are destined for a neural fate upregulate the Notch ligand Delta, which activates Notch signalling in neighbouring cells, causing them to adopt an epidermal fate through the repression of the *achaete/scute* proneural genes. However, as Ramain *et al.* have now shown, lateral inhibition is not the only pathway that Notch can use to prevent neural differentiation.

The small sensory bristles (microchaetae) on the fly thorax are known to be specified through lateral inhibition, and the authors screened for mutations that resulted in their loss. They identified several *Notch* mutant alleles that produced such a phenotype, and they named these alleles $N^{Mcd}$ (Mcd stands for microchaetae defective). These were classed as gain-of-function mutations, because they seemed to enhance Notch's normal function in repressing neural cell fate. The authors reasoned that if the mutant forms of Notch acted through the lateral inhibition pathway, then inactivating downstream components of this pathway (such as Suppressor of Hairless (Su(H)) and Groucho) in the $N^{Mcd}$ mutants should restore the development of microchaetae. However, no such effect was seen, indicating that another pathway must be involved.

The adaptor protein Deltex has already been implicated in an alternative Notch signalling pathway that also represses *achaete/scute*, so Ramain *et al.* tried inactivating Deltex on an $N^{Mcd}$ background. This time, the $N^{Mcd}$ mutant phenotype was rescued, indicating that it was caused by abnormal activation of a Deltex-mediated pathway. Because the $N^{Mcd}$ phenotype depends on Deltex activation, this pathway must presumably have to be repressed in order for microchaetae to develop. How might this be achieved? A clue

BEHAVIOURAL GENETICS

# Ants and Bs


**When trying to understand how complex social behaviour — as displayed by social insects, such as ants and bees, for example — evolved or is controlled, one problem is knowing where to start. It has been very difficult even to find examples of complex social traits that are clearly heritable, let alone ones in which the genetic basis of variation in behaviour can be indentified. But a striking exception to this rule has been described by Krieger and Ross in *Science*. They have identified a gene that determines whether a colony of fire ants (*Solenopsis invicta*) will have one queen (a monogynous colony) or many (polygynous). The identity of the gene gives us interesting clues as to the possible neural mechanisms of the control of queen number in fire ant colonies.**

**Red fire ants are native to South America, but were imported into the United States in the 1930s, and have since spread across much of the southeast United States. Colonies of red fire ants can be monogynous or polygynous, and it was previously shown that a single gene (*Gp-9*) controls the choice between the two. Colonies consisting of ants that are homozygous for the *B* allele at *Gp-9* are always monogynous, whereas**