In the news

MAKING WAVES

That first ultrasound scan is an exciting time for parents-to-be, letting them see the newest member of their family for the first time and reassuring them that their baby is healthy. But could ultrasound harm the fetus? Pasko Rakic and colleagues at Yale Medical School have recently found that, in pregnant mice, prolonged exposure to ultrasound disrupts neuronal migration during fetal brain development.

It is too early to translate the team's results in mice to humans, as Rakic comments: "We do not have any evidence ourselves that ultrasound waves ... have any effect on the developing human brain" (*Reuters*, 7 August 2006). The conditions used in their studies were very different to those seen in the clinic. The ultrasound source was maintained in a fixed position and mice were exposed to the sound waves for 30 mins or more. When a scan is performed on expectant mothers, it is just that — a scan. The ultrasound source is continuously moved and so the fetal brain is typically exposed to ultrasound for only a few seconds.

Moreover, the changes induced by ultrasound exposure were small and it is not known whether they were significant enough to affect behaviour. The authors plan to investigate this further in monkeys.

Concerns about the effects of ultrasound on the fetal brain have been voiced before, with exposure to ultrasound being linked to decreased birth weight and delayed speech development, as well as an increased probability of being left-handed. However, the medical value of ultrasound scans is not disputed, as Rakic says: "...the benefits of the ultrasound for diagnosis are so big that I would not hesitate to use it at all" (*New Scientist*, 7 August 2006). Instead, in an age when ultrasound machines can be bought for use in the home, researchers suggest that expectant mothers minimize any risk by only having scans that are medically necessary.

Samantha Barton

NEUROLOGICAL DISORDERS

Mind over machine

The brain is thought by some to be the most powerful computer known. Harnessing these impressive processing powers to control machines, including computers and robots, has been considered to be in the realms of fantasy. However, two reports in *Nature* describing advances in electronic neuronal implants indicate that in the future the mind could be used to manipulate machines, with the potential to improve mobility and communication for those with severe spinal cord injuries.

Currently available neuromotor assistive technologies are limited by low performance. They are typically slow and inaccurate, and can require the user to adapt the neuronal activity in their brain, which requires months of intensive training. However, the work of Hochberg, Santhanam and their respective colleagues suggests that there are solutions to these problems.

Hochberg and co-workers developed a brain–computer interface (BCI) that used a 96-microelectrode array implant to measure neuronal firing patterns directly in the motor cortex. The clinical usefulness of the team's BCI hinged on whether movement signals persist in the motor

NEURODEGENERATIVE DISORDERS

Receptor traffic — parkin rules

Parkinson's disease (PD), a progressive neurodegenerative disorder that causes tremors, stiffness, and impaired balance and coordination, results from the loss of dopamine-producing neurons in the midbrain. Defects in the ubiquitin–proteasome system and protein aggregation in Lewy bodies have typically been associated with the development of PD. A study in *Nature Cell Biology* now indicates that a proteasome-independent ubiquitylation pathway also contributes to the aetiology of this disease.

Parkin encodes an E3 ubiquitin ligase that is inactivated in ~50% of early-onset PD cases. Edward Fon and colleagues showed that, in addition to targeting proteins for degradation, parkin can delay epidermal growth

factor (EGF) receptor (EGFR) trafficking by ubiquitylating the endocytic scaffolding protein EPS15, and can promote signalling through phosphoinositide 3-kinase (PI3K)–Akt, an important pathway for neuronal survival.

The ubiquitin-like domain of wild-type parkin, but not of a PD-associated form, binds to the ubiquitin-interacting motif (UIM) of EPS15. This interaction is regulated by EGF, the addition of which transiently increased the formation of a parkin–EPS15–EGFR complex and EPS15 ubiquitylation. The authors propose that intramolecular UIM–ubiquitin binding not only decreases the capability of EPS15 to induce EGFR internalization, but also prevents EPS15 from interacting with parkin, possibly explaining the transient nature of the complex.