the expression of the gene that encodes the VGF nerve growth factor. Microinfusion of a synthetic VGF peptide into the lateral ventricles of two rodent models of depression decreased immobility, while heterozygous *Vgf*$^{+/-}$ mice showed increased immobility compared with their wild-type litter mates. Although depression is difficult to model in rodents, decreases in immobility in these models have been correlated with antidepressant actions in humans.

The authors conclude that the VGF signaling cascade could prove a suitable target for future antidepressant drugs.

Original article Hunsberger JG *et al.* (2007) Antidepressant actions of the exercise-regulated gene VGF. *Nat Med* **13:** 1476–1482

Somatosensory cortex thickening in patients with migraine

The pathophysiology of migraine has been suggested to involve neurogenic inflammation, and studies using diffusion tensor imaging have demonstrated that patients with migraine show permanent interictal changes in the trigeminal somatosensory pathway. To investigate this phenomenon further, DaSilva *et al*. examined whether the somatosensory cortex (SSC), which receives noxious and innocuous sensory inputs from the trigeminal nerve, shows morphological changes in patients with migraine.

Measurements of SSC thickness were made *in vivo* in 24 patients with migraine (12 with aura and 12 without aura) and compared with SSC measurements from healthy controls matched for age and sex. Patients with migraine had an average SSC thickness that was significantly greater than that of controls. The greatest differences in thickness were observed in the caudal SSC, which somatotopically represents the head and face.

The findings suggest that migraine involves thickening of gray matter in the SSC, regardless of whether patients experience aura during attacks. Together with the diffusional changes in the trigeminal somatosensory pathway found in previous studies, this indicates that the processes that underlie migraine involve the somatosensory system.

Further studies are needed to determine whether thickening of the SSC predisposes to migraine, or whether repetitive migraine attacks lead to thickening of the SSC over time.

Original article DaSilva AFM *et al.* (2007) Thickening in the somatosensory cortex of patients with migraine. *Neurology* **69:** 1990–1995

Plasticity of propriospinal relays facilitates spontaneous recovery from spinal cord injury

Spontaneous recovery following spinal cord injury is generally attributed to the maintenance or regeneration of long axons descending from the brain past the incomplete lesion, but the mechanism is unclear. Courtine *et al.* have now shown in mice that reorganization of the CNS along new, indirect pathways—involving propriospinal relay connections that bypass the lesion—facilitate functional recovery even when long-tract supraspinal pathways are interrupted.

Following lateral hemisection at T12 in adult mice, hind limb stepping was initially impaired ipsilaterally, but limited function was gradually regained over time. Retrograde tract tracing from below the injury revealed a significant and near-total reduction in the number of neurons in the brainstem and spinal cord of injured mice compared with uninjured mice ($P<0.001$) immediately following spinal cord injury. The number of retrogradely labeled propriospinal neurons increased gradually following the injury, and after 10 weeks was 40% of the level observed in uninjured mice, whereas no increase was found in the brain. Mice that received two spatially separated hemisections at T12 and T7 recovered stepping only when the second, contralateral hemisection was performed after a 10-week time delay and not simultaneously. Infusion of the excitotoxin *N*-methyl-D-aspartate above the level of the lesion or lesions, which ablated propriospinal neurons but spared descending supraspinal fibers, eliminated spontaneous recovery, indicating that recovery depends on changes in propriospinal circuits that occur over time.

This mechanism, which enables the relay of information past lesion sites, might be harnessed to circumvent the need to regenerate long axons following incomplete spinal cord injury in humans.

Original article Courtine G *et al.* (2008) Recovery of supraspinal control of stepping via indirect propriospinal relay connections after spinal cord injury. *Nat Med* **14:** 69–74