treatment with interferon-β (IFN-β) than in those receiving other treatments. Results of a recent study investigating the lifetime prevalence of primary headaches in patients with MS provide initial evidence to associate IFN-β treatment with headache.

Researchers in Italy carried out an assessment of 150 patients recruited from their MS clinic who had been receiving treatment with either an IFN-β therapy ($n = 109$) or another immunotherapy ($n = 41$) for at least 3 months. Patients were interviewed about headache symptoms with the use of an ad hoc questionnaire that included questions relating to lifetime occurrence of headache, and questions on the location, frequency and duration of attacks. Patients were also asked if they considered that their headaches had started after commencing treatment for MS, and whether there had been any changes to the features of their headaches since the start of MS treatment.

A total of 79 patients treated with an IFN-β therapy reported headaches, and of these 38 reported first experiencing them after commencement of MS treatment. Seventeen patients treated with an IFN-β therapy reported changes to pre-existing headaches. None of the patients receiving other treatments for MS reported either a *de novo* headache or a change to a pre-existing headache after commencing therapy.

The authors suggest that *de novo* headaches of the migraine or tension-type variety, and the aggravation of pre-existing primary headaches, should be considered as potential adverse effects of IFN-β treatment.

Original article La Mantia L *et al.* (2006) Interferon treatment may trigger primary headaches in multiple sclerosis patients. *Mult Scler* **12:** 476–480

A mutant cell-cycle protein that promotes axon growth

Researchers in the US have elucidated part of the mechanism controlling the growth and differentiation of nerve cells, and their discovery might provide a means of promoting the regrowth of damaged neurons. The researchers investigated the role of Id2, a protein that promotes cell growth in the developing nervous system, but which is eliminated in mature neurons. Id2 is also abundantly expressed in many cancers. Id proteins inhibit transcription factors that promote cell differentiation, thereby locking cells into an unspecialized, dividing state.

The authors found that Id2 is destroyed by the anaphase-promoting complex (APC), an enzyme involved in cell-cycle regulation. The destruction of Id2 halts neuroblast cell division and leads to the development of mature neurons. The researchers produced a mutant Id2 protein that lacked the destruction sequence targeted by APC. Expression of this mutant protein led to prolonged axon growth in cerebellar granule neurons, cortical neurons and neuroblast cell lines; this effect could be reversed by the overexpression of E47, a transcription factor that drives differentiation of neurons and is normally inhibited by Id2. Mutant Id2 was able to stimulate axon growth even in the presence of myelin, which normally inhibits axon regrowth in damaged neurons.

Degradation-resistant Id2 offers the possibility of permitting damaged neurons to regenerate, with potential therapeutic applications for spinal cord damage and neurodegenerative conditions such as Alzheimer's disease.

Original article Lasorella A *et al.* (2006) Degradation of Id2 by the anaphase-promoting complex couples cell cycle exit and axonal growth. *Nature* **442:** 471–474

Influence of dopamine transporter genotype on response to ADHD medication

A study in children with attention-deficit hyperactivity disorder (ADHD) suggests that genetic variability in the dopamine transporter gene (*DAT1*) influences the response to ADHD treatment. Short-interval cortical inhibition (SICI), measured in the motor cortex by transcranial magnetic stimulation, is reduced in individuals with ADHD and correlates with severity of symptoms.

In a double-blind, crossover study, Gilbert *et al.* compared the effect on SICI of two ADHD medicines, the psychostimulant methylphenidate and the selective norepinephrine reuptake inhibitor atomoxetine, in 16 children (14 male) aged 8–17 years. Patients were given either a single dose of methylphenidate followed 1 week later by a single dose of atomoxetine, or the reverse. SICI was measured before and 90 min after each treatment. Analysis of the variable number of tandem repeats (VNTR) at the 3' end of *DAT1* was performed for each patient.