to the excitable phenotype that is seen in patients with the mutation. Early detection of susceptible individuals may permit therapeutic intervention to ameliorate the severity of epilepsy before the onset of overt symptoms.

Original article Chiu C *et al.* (2008) Developmental impact of a familial $GABA_A$ receptor epilepsy mutation. *Ann Neurol* **64:** 284–293

Alemtuzumab limits multiple sclerosis but is associated with autoimmune complications

Immunotherapy might only affect long-term disability in multiple sclerosis (MS) if administered early in the disease course. A phase II, blinded trial investigated the efficacy of alemtuzumab, a humanized monoclonal antibody that causes prolonged T-cell depletion, in patients with early relapsing–remitting MS.

Previously untreated patients who had developed relapsing–remitting MS no more than 36 months earlier were randomly assigned to receive alemtuzumab 12 mg or 24 mg daily, or to 44 μg interferon β1a (IFN-β1a) three times a week, for 36 months.

The risk of sustained disability was 71% lower and the rate of relapse was 74% lower in the 223 patients who were receiving alemtuzumab than in the 111 individuals on IFN-β1a. In addition, the mean disability on a 10-point scale improved by 0.39 of a point in the alemtuzumab group but worsened by 0.38 of a point in the IFN-β1a group ($P<0.001$). The incidence of infection and of thyroid associated adverse events was higher in patients who received alemtuzumab than in those on IFN-β1a. Immune thrombocytopenic purpura developed in six (2.8%) patients in the alemtuzumab group, one of whom died, and in one (0.9%) patient in the IFN-β1a group.

Alemtuzumab is effective at preventing relapse and at limiting the accumulation of disability in MS but is associated with autoimmune complications. The authors point out that most patients with early relapsing–remitting MS are young and have little disability; it remains to be confirmed whether it is reasonable to treat such individuals with alemtuzumab given the severe adverse effects.

Original article CAMMS223 Trial Investigators (2008) Alemtuzumab vs. interferon beta-1a in early multiple sclerosis. *N Engl J Med* **359:** 1786–1801

Variation in cortical thickness is associated with cocaine addiction

Cocaine use is thought to affect functional activation of brain structures involved in reward regulation and attention function, such as the cortex. Little is understood of what causes differences in judgments and decisions between cocaine-dependent individuals and healthy individuals. Makris and colleagues investigated whether cortical thickness abnormalities can identify individuals who are predisposed to addiction, or if cocaine use rather alters structures in the brain.

The team used T1-weighted MRI to measure cortical thickness in 20 cocaine-dependent patients and 20 matched controls and assessed possible behavioral implications. Cocaine addicts had a notably lower total cortical volume and a thinner cortex in the region involved in reward regulation than did controls. Also, the cortex, which is normally thicker in the right hemisphere, had reversed asymmetry in cocaine users. Impaired performance in preference-based tests and attention tasks were associated with a thinner cortex in cocaine-dependant individuals.

The authors' observation that cortical thinness in the region involved in reward regulation was associated with abnormal control of judgments and decision making is suggestive of a factor that predisposes individuals to drug addiction. By contrast, further investigation into the abnormalities in cortical regions associated with attention function indicated that the changes in the region were the result of cocaine abuse.

Original article Makris N *et al.* (2008) Cortical thickness abnormalities in cocaine addiction—a reflection of both drug use and a pre-existing disposition to drug abuse? *Neuron* **60:** 174–188

Some 'sporadic' brain tumors are hereditary

A hereditary component to the incidence of brain tumors (other than those associated with well-established hereditary syndromes) has long been suspected. A retrospective study of the Utah Population Database—which includes the genealogy of the Utah Pioneers