Suri *et al.* examined GBM samples from 30 children aged 9 months to 18 years. Pediatric GBM had similar morphological characteristics to adult GBM, and a similar proliferative potential as measured by MIB-1 antibody binding. Amplification of epidermal growth factor receptor (*EGFR*) is a common feature of adult GBM, but only one patient's sample in this pediatric study showed this feature—although EGFR protein overexpression was found in seven patients (23%). Phosphatase and tensin homolog (*PTEN*) deletion is another common feature of adult GBM; however, only one instance of *PTEN* deletion was observed in this pediatric population. By contrast, p53 expression was seen in 19 cases (63%), a far higher rate than that seen in adult primary GBM. Loss of the cyclin-dependent kinase inhibitors p16 and p27 was seen in 68% and 54% of pediatric cases, respectively; these figures are similar to those observed for adult GBM. One child had polysomy of chromosomes 7 and 10.

The study suggests that pediatric and adult GBM have distinct molecular expression profiles, a finding that has implications for the use in children of therapies designed for adults—for example, therapies that target the EGFR and PTEN pathways might have reduced efficacy in pediatric GBM. Further elucidation of the differences between adult and pediatric GBM may aid the identification of novel molecular targets for GBM therapy.

Original article Suri V *et al.* (2008) Pediatric glioblastomas: a histopathological and molecular genetic study. *Neuro-Oncology* [doi:10.1215/15228517-2008-092]

Substantial amyloid deposition without cognitive impairment

The relationship between amyloid β (Aβ) deposition and cognitive function is unclear: previous studies have included participants with normal cognition without knowledge of their Aβ deposition status. Researchers at the University of Pittsburgh measured amyloid deposition and cognitive function in clinically unimpaired elderly volunteers. They found that 21% of participants had substantial amyloid deposits, but that these deposits did not influence cognitive ability.

Aizenstein *et al.* used PET imaging with the marker Pittsburgh compound B (PiB), which binds amyloid deposits, to measure Aβ deposition in 43 volunteers with a mean age of 74.4 years. Of these participants, 9 were classified as amyloid-positive, 29 as amyloid-negative, and 5 as intermediate (within 2.5% of the cut-off point). *APOE ε4* allele frequency was significantly lower among amyloid-negative participants (1 of 29) than among the remaining participants (4 of 14). The Aβ deposits in positive volunteers had a similar distribution to those found in patients with Alzheimer disease, but were present in substantially smaller quantities, and were not associated with impairment of cognitive function.

The results suggest that Aβ deposition might precede the development of clinical symptoms of cognitive impairment, a finding that might have implications for the development of anti-amyloid therapies. If PiB retention is indeed a predictor of Alzheimer disease, PiB PET might provide an early indication of risk—years in advance of overt symptoms.

Original article Aizenstein HJ *et al.* (2008) Frequent amyloid deposition without significant cognitive impairment among the elderly. *Arch Neurol* **65:** 1509–1517

Somatosensory cortex reorganization linked to neuropathic pain

Phantom limb pain following amputation correlates with primary somatosensory cortex (S1) reorganization. Wrigley *et al.* hypothesized that a similar mechanism underlies persistent neuropathic pain after spinal cord injury. They found that patients with spinal cord injury undergo S1 reorganization, and that the degree of reorganization correlates with pain intensity.

The study population comprised 20 patients with complete spinal cord injury—10 with and 10 without neuropathic pain below the level of the injury—and 21 controls without spinal cord injury. All participants underwent functional MRI during light brushing of the lip, right thumb and right little finger. The scans showed that the S1 region activated by little-finger brushing was significantly displaced in participants with neuropathic pain, compared with both controls and patients without pain. The amount of displacement correlated with reported pain intensity as measured by a visual analog scale. In addition, the region that represented thumb activation was significantly displaced in patients with neuropathic pain