# IN BRIEF

## ALZHEIMER DISEASE

The specific involvement of microglia in Alzheimer disease is still widely debated within the scientific community. Grathwohl *et al.* have demonstrated in transgenic mice that almost-complete transient ablation of microglia does not affect amyloid plaque formation and maintenance, or amyloid-associated neuritic dystrophy. These results indicate that resident microglia have a limited capacity to influence *de novo* amyloidosis.

**Original article** Grathwohl, S. A. *et al.* Formation and maintenance of Alzheimer's disease β-amyloid plaques in the absence of microglia. *Nat. Neurosci.* doi:10.1038/nn.2432

## STROKE

In a community-based cohort study, Elkind *et al.* have demonstrated that C-reactive protein, a nonspecific biomarker of inflammation, is associated with risk of mortality and myocardial infarction. Contrary to some previous reports, however, this marker was not shown to be associated with increased stroke risk, thereby fueling the controversy regarding the link between C-reactive protein and stroke.

**Original article** Elkind, M. S. V. *et al*. High-sensitivity C-reactive protein predicts mortality but not stroke: the Northern Manhattan Study. *Neurology* **73**, 1300–1307 (2009).

## PAIN

Schnakers *et al.* have developed a new scale that assesses behavioral responses to noxious stimuli in patients who are minimally conscious or in a vegetative state. This new scale demonstrates good inter-rater agreement and good concurrent validity with four previously validated scales. Furthermore, scores obtained from patients who are minimally conscious are significantly different from scores obtained from patients in a vegetative state.

**Original article** Schnakers, C. *et al*. The nociception coma scale: a new tool to assess nociception in disorders of consciousness. *Pain* doi:10.1016/j.pain.2009.09.028

## EPILEPSY

Six recurrent microdeletions have previously been shown to be associated with a large number of neuropsychiatric disorders, and one of these microdeletions is known to be a risk factor for idiopathic generalized epilepsy. De Kovel *et al.* have now demonstrated that two more of these microdeletions are also associated with this disease. Together, these three microdeletions might underlie a substantial proportion of cases of idiopathic generalized epilepsy.

**Original article** de Kovel, C. G. F. *et al*. Recurrent microdeletions at 15q11.2 and 16p13.11 predispose to idiopathic generalized epilepsies. *Brain* doi:10.1093/brain/awp262