IN BRIEF

TRAUMATIC BRAIN INJURY

Amyloid-β deposits accumulate in the brain after traumatic brain injury, thereby increasing the risk of developing Alzheimer disease. In a study conducted in a mouse model, Loane *et al.* found that amyloid precursor protein secretases could represent new targets for the treatment of traumatic brain injury. Inhibition of β-secretase or γ-secretase led to a reduction in post-traumatic tissue loss and improvements in motor and cognitive recovery.

Original article Loane, D. J. *et al.* Amyloid precursor protein secretases as therapeutic targets for traumatic brain injury. *Nat. Med.* **15**, 377–379 (2009).

PARKINSON DISEASE

Patients with Parkinson disease who are treated with dopamine agonists are at an increased risk of developing compulsive gambling and hypersexuality. A retrospective study by Bostwick and colleagues showed these character traits occurred in 18.4% of patients with Parkinson disease taking dopamine agonists. Patients taking subtherapeutic agonist doses, those taking either carbidopa or levodopa, and those receiving no treatment did not develop such traits.

Original article Bostwick, J. M. *et al.* Frequency of new-onset pathologic compulsive gambling or hypersexuality after drug treatment of idiopathic Parkinson disease. *Mayo Clin. Proc.* **84**, 310–316 (2009).

STROKE

Exceeding guideline physical activity levels considerably reduces the risk of incident stroke. Williams *et al.* observed a 12% and 11% decrease in risk of stroke per kilometer run per day in men and women, respectively. Risk was notably lower in individuals who ran ≥2 km per day than in those who ran less; those who ran ≥8 km per day were 60% less likely to develop stroke than those who ran <2 km per day.

Original article Williams, P.T. Reduction in incident stroke risk with vigorous physical activity: evidence from 7.7-year follow-up of the national runners' health study. *Stroke* **40**, 1921–1923 (2009).

EPILEPSY

The risk of febrile seizures can be attributed to a common polymorphism in the sodium channel gene *SCN1A*. Schlachter and colleagues found that the polymorphism was responsible for a threefold increased risk of febrile seizures in patients homozygous for the gene variant. The researchers suggest that much of the genetic etiology of febrile seizures could be explained by this polymorphism.

Original article Schlachter, K., Alterman, R. L. & Tagliati, M. A splice site variant in the sodium channel gene SCN1A confers risk of febrile seizures. *Neurology* **17**, 974–978 (2009).