$\textit{Nature Reviews Neurology} \ \textbf{10}, 674 \ (2014); \ published \ online \ 18 \ November \ 2014;$ 

doi:10.1038/nrneurol.2014.218; doi:10.1038/nrneurol.2014.219:

doi:10.1038/nrneurol.2014.219;

doi:10.1038/nrneurol.2014.221

### IN BRIEF

#### **NEURO-ONCOLOGY**

## Nuclear export protein inhibitors could limit glioblastoma growth

Selective inhibitors of nuclear export (SINEs) might offer hope for patients with glioblastoma. Green *et al.* found that SINEs, such as KPT-276, slowed down the growth of patient-derived glioblastoma neurosphere cultures, and prolonged survival in glioblastoma mouse model grafted with patient-derived glioblastoma cells. Because KPT-276 has excellent brain penetration and has been previously investigated for treatment of other forms of cancer, the researchers have initiated a clinical trial in patients with relapsed glioblastoma.

**Original article** Green, A. L. *et al.* Preclinical antitumor efficacy of selective exportin 1 inhibitors in glioblastoma. *Neuro-Oncology* doi:10.1093/neuonc/nou303

#### **NEURODEVELOPMENTAL DISORDERS**

# Whole-exome sequencing elucidates genetic architecture of autism spectrum disorder

The results of the largest whole-exome sequencing study so far in autism spectrum disorder (ASD) suggest that interplay between common and rare genetic variants contributes to neurodevelopmental disorders. Comparison of 3,871 individuals with ASD and 9,937 parental or ancestry-matched controls identified 129 autosomal genes that were associated with ASD. Many of the genetic variants were *de novo* mutations in genes implicated in synaptic formation, transcriptional regulation and chromatin-remodelling pathways, thus providing insight into neurobiology of ASD.

 $\begin{tabular}{ll} \textbf{Original article} De Rubeis, S. et al. Synaptic, transcriptional and chromatin genes disrupted in autism. {\it Nature} doi:10.1038/nature13772 \end{tabular}$ 

#### SLEEF

### Sleep deprivation linked to blood-brain barrier disruption

Sleep restriction of just 6 days impairs several aspects of blood–brain barrier function in mice, according to new research published in *The Journal of Neuroscience*. These findings might have widespread implications for public health given the common occurrence of sleep restriction and disturbances in the general population. Chronic sleep impairment has previously been connected to neurodegenerative disorders, such as Alzheimer disease, but little was known about the mechanisms through which sleep restriction increases susceptibility to neurodegeneration.

Original article He, J. et al. Sleep restriction impairs blood-brain barrier function. J. Neurosci. doi:10.1523/JNEUROSCI.2111-14.2014

### PARKINSON DISEASE

# SNCA genetic variability and head injury could jointly increase the risk for PD

Head injury and genetic variants of the  $\alpha$ -synuclein-encoding SNCA might co-influence susceptibility to Parkinson disease (PD), a new study shows. Regression analysis of history of head injury and SNCA alleles in 561 individuals with idiopathic PD and 721 controls indicated that certain SNCA variants were associated with 3–4.5-fold risk for PD after head injury. Interplay between head injury and  $\alpha$ -synuclein variants could explain why previous reports of the link between PD and head injury have been inconclusive.

**Original article** Lee, P. C. *et al.* Head injury, α-synuclein genetic variability and Parkinson's disease. *Eur. J. Neurol.* doi:10.1111/ene.12585