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IN BRIEF

MULTIPLE SCLEROSIS

On the TRAIL of IFN- β response

A single nucleotide polymorphism (SNP) in the *TRAILR-1* gene is associated with the clinical response to IFN- β therapy in patients with multiple sclerosis (MS), according to a study conducted in a Spanish cohort. *TRAILR-1* encodes a receptor for TRAIL, a proapoptotic cytokine that has been implicated in MS pathogenesis. The CC genotype of the rs20576 SNP, which results in a Glu228Ala substitution in TRAILR-1, was found to correlate with an enhanced response to IFN- β , and might be a useful biomarker to predict the outcome of this treatment.

Original article López-Gómez, C. *et al.* Candidate gene study of *TRAIL* and *TRAIL Receptors*: association with response to interferon beta therapy in multiple sclerosis patients. *PLoS ONE* 8, e62540 (2013)

SLEEP

Obstructive sleep apnoea raises the risk of cerebral white matter change

Research from Korea indicates that obstructive sleep apnoea (OSA) is a risk factor for cerebral white matter change (WMC) in middle-aged and older populations. Among 503 individuals with an average age of 59.63 years, moderate to severe OSA increased the odds of cerebral WMC approximately twofold. The authors suggest that early detection and treatment of OSA could aid the prevention of vascular dementia and stroke.

Original article Kim, H. *et al.* Obstructive sleep apnea as a risk factor for cerebral white matter change in a middle-aged and older general population. *Sleep* 36, 709–715 (2013)

MOVEMENT DISORDERS

SCA37—a new subtype of spinocerebellar ataxia

A distinct spinocerebellar ataxia (SCA) phenotype that includes impaired vertical eye movements has recently been identified in a family from Spain. This new SCA subtype, designated SCA37 by the Human Genome Nomenclature Committee, maps to an 11-Mb region of chromosome 1p32. The altered eye movements seem to manifest early in the disease course, and may even precede the development of overt ataxia.

Original article Serrano-Munuera, C. *et al.* New subtype of spinocerebellar ataxia with altered vertical eye movements mapping to chromosome 1p32. *JAMA Neurol.* doi:10.1001/jamaneurol.2013.2311

STROKE

Could PTEN nuclear translocation be a target for therapy after stroke?

Nuclear translocation of the phosphatase PTEN increases the susceptibility of neurons to excitotoxic and ischaemic injury, new research reveals. Zhang *et al.* found that excitotoxic stimulation of cultured neurons caused PTEN to accumulate in the nucleus. In a rat model of stroke, a short interfering peptide that inhibited this translocation event protected against ischaemic brain damage. These findings suggest that nuclear translocation of PTEN could be targeted to limit ischaemic brain damage after stroke.

Original article Zhang, S. *et al.* Critical role of increased PTEN nuclear translocation in excitotoxic and ischemic neuronal injuries. *J. Neurosci.* 33, 7997–8008 (2013)