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# IN BRIEF

#### **HEADACHE**

### Self-reported trigger factors might not provoke migraine

A migraine attack can be provoked by external factors such as bright light or strenuous exercise. Hougaard and colleagues exposed 27 patients with migraine with aura to their self-reported trigger factor, to examine causal links between trigger and migraine. Surprisingly, only three (11%) patients experienced migraine after provocation, suggesting the need for prospective validation of patient-identified trigger factors in future studies.

**Original article** Hougaard, A. et al. Provocation of migraine with aura using natural trigger factors. *Neurology* **80**, 428–431 (2013)

#### PARKINSON DISEASE

#### Transcription factor Nurr1 maintains dopaminergic neurons

The transcription factor Nurr1 has been implicated in Parkinson disease (PD)—a movement disorder caused by selective destruction of dopaminergic neurons. Researchers have now shown that targeted ablation of Nurr1 in dopaminergic neurons in adult mice recapitulates early features of PD, including motor impairment. Nextgeneration RNA sequencing of these neurons revealed that mitochondrial genes are a key regulatory target of Nurr1, in line with the known involvement of mitochondrial dysfunction in PD. Together, the findings suggest Nurr1 as a potential therapeutic target in PD.

Original article Kadkhodaei, B. et al. Transcription factor Nurr1 maintains fiber integrity and nuclear-encoded mitochondrial gene expression in dopamine neurons. Proc. Natl Acad. Sci. USA 110, 2360–2365 (2013)

# NEUROIMMUNOLOGY

# BAFF as a target of IVIg therapy for autoimmune disease

Intravenous immunoglobulin (IVIg) is used to treat the autoimmune disease chronic inflammatory demyelinating polyneuropathy (CIDP), but the precise mechanism of action is not known. Bick et al. measured serum levels of B-cell activating factor (BAFF), which regulates B-cell homeostasis and antibody production, in patients with CIDP BAFF levels were higher in patients than in controls, and were significantly reduced by IVIg treatment. Inhibition of BAFF production could, therefore, contribute to the therapeutic effect of IVIg.

**Original article** Bick, S. et al. Intravenous immunoglobulin inhibits BAFF production in chronic inflammatory demyelinating polyneuropathy—a new mechanism of action? *J. Neuroimmunol.* doi:10.1016/j.jneuroim.2013.01.001

## **GENETICS**

# Phenotypic spectrum associated with newly identified mutations in episodic ataxia type 1

Episodic ataxia type 1 (EA1) is caused by mutations in KCNA1—the gene encoding potassium channel  $K_{\rm V}1.1$ . A recent study of 15 affected individuals from four families led to identification of three new KCNA1 mutations, which all resulted in loss of  $K_{\rm V}1.1$  function. Four of the study participants reported impairment of hearing, which expands the clinical phenotype of EA1. The study also included clinical documentation dating back to 1928 in one family, representing the earliest clinical record of EA1.

**Original article** Tomlinson, S. E. *et al.* Clinical, genetic, neurophysiological and functional study of new mutations in episodic ataxia type 1. *J. Neurol. Neurosurg. Psychiatry* doi:10.1136/jnnp-2012-304131