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IN BRIEF

MULTIPLE SCLEROSIS

Risk of comorbid inflammatory diseases in MS might not be genetically determined

Patients with multiple sclerosis (MS) often present with other immune-mediated disorders, such as ulcerative colitis and type 1 diabetes. Researchers in Sweden analysed data from a large register of patients with MS and their parents to determine whether the risk of such comorbidity has a genetic component. They found that, in contrast to index cases, parents of patients with MS did not show an increased likelihood of having an immune-mediated disorder compared with control parents, arguing against a genetic influence.

Original article Roshanisefat, H. *et al.* Shared genetic factors may not explain the raised risk of comorbid inflammatory diseases in multiple sclerosis. *Mult. Scler.* doi:10.1177/1352458512438240

EPILEPSY

Immunotherapy could benefit treatment-resistant epilepsy

Accumulating evidence has suggested an autoimmune component to drug-resistant epilepsy. Now, immunotherapy in 27 patients with uncontrolled seizures with suspected autoimmune aetiology has shown striking results. At a median follow-up of 10 months, 22 patients (81%) exhibited substantial improvements in seizure status, and 18 patients (67%) were seizure-free. These results suggest that the treatment of autoimmunity could help to address the large unmet medical need of drug resistance in epilepsy.

Original article Quek, A. M. *et al.* Autoimmune epilepsy: clinical characteristics and response to immunotherapy. *Arch. Neurol.* doi:10.1001/archneurol.2011.2985

MOVEMENT DISORDERS

Interferon-γ shows promise in a mouse model of Friedreich ataxia

Friedreich ataxia is a hereditary disease caused by mutation of the frataxin (*FXN*) gene and resultant low levels of the FXN protein. In a mouse model of the disease, Tomassini *et al.* found that subcutaneous injections of interferon-γ for 14 weeks restored FXN levels in dorsal root ganglion neurons, which are particularly vulnerable in this disease, and improved motor coordination. The findings open therapeutic avenues for Friedreich ataxia, a disease for which no specific treatments are currently available.

Original article Tomassini, B. *et al.* Interferon gamma upregulates frataxin and corrects the functional deficits in a Friedreich ataxia model. *Hum. Mol. Genet.* doi:10.1093/hmg/dds110

ALZHEIMER DISEASE

Targeting apolipoprotein E improves AD phenotype in mice

Apolipoprotein E (APOE) is indirectly involved in the proteolytic degradation of amyloid-β, and allelic variation in the *APOE* gene is the leading genetic risk factor for Alzheimer disease (AD). As *APOE* expression is transcriptionally regulated by retinoid X receptors (RXRs), Cramer *et al.* investigated the oral RXR agonist bexarotene in AD mice. The resultant increase in APOE levels was accompanied by increased clearance of amyloid-β, rapid reduction in plaque volume and improvements in functional deficits associated with AD. Bexarotene is already approved as an anticancer drug.

Original article Cramer, P. E. *et al.* ApoE-directed therapeutics rapidly clear β-amyloid and reverse deficits in AD mouse models. *Science* **335**, 1503–1506 (2012)