Nature Reviews Neurology **11**, 369 (2015); published online 23 June 2015;
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IN BRIEF

NEUROPSYCHIATRIC DISORDERS

A common genetic basis for creativity and psychosis?

Though the link between creative genius and mental illness is often accepted as conventional wisdom, a new study presents evidence that the association has a genetic basis. Polygenic risk scores for schizophrenia and bipolar disorder were found to be higher in members of national artistic societies than in nonmembers. A similar effect arose in people whose creativity was assessed via a questionnaire about artistic pursuits. The correlation between creativity and the genetic risk of psychosis disorders could not be explained by differences in cognitive function, education or family history of schizophrenia.

Original article Power, R. A. *et al.* Polygenic risk scores for schizophrenia and bipolar disorder predict creativity. *Nat. Neurosci.* doi:10.1038/nn.4040

EPILEPSY

Thalamic stimulation for epilepsy can disrupt sleep

Polysomnography in seven patients with epilepsy has revealed that deep brain stimulation can interfere with deep sleep. During active 5 V stimulation to the anterior thalamus, patients demonstrated brief periods of arousal. Lowering stimulation voltage to 0–4 V during the night reduced the frequency of arousal events, and patients reported improvements in sleep and neuropsychiatric symptoms such as mood and anxiety. Seizure frequency was not affected by the new stimulation parameters.

Original article Voges, B. R. *et al.* Deep brain stimulation of anterior nucleus thalami disrupts sleep in epilepsy patients. *Epilepsia* doi:10.1111/epi.13045

MOTOR NEURON DISEASE

Data from knockout mice might rule out *C9orf72* loss of function as a cause for amyotrophic lateral sclerosis

C9orf72 repeat expansions cause amyotrophic lateral sclerosis (ALS) in humans, but whether this effect arises from a gain or loss of function is unclear. Koppers and co-workers generated conditional *C9orf72* knockout mice, which showed no changes in survival, motor function or motor neuron morphology. These results suggest that loss of *C9orf72* is not sufficient to cause ALS.

Original article Koppers, M. *et al.* *C9orf72* ablation in mice does not cause motor neuron degeneration or motor deficits. *Ann. Neurol.* doi:10.1002/ana.24453

MOTOR NEURON DISEASE

New mouse model of amyotrophic lateral sclerosis with a *C9orf72* repeat expansion

Viral delivery of a *C9orf72* allele containing 66 GGGGCC repeats produces symptoms and pathology consistent with amyotrophic lateral sclerosis (ALS) in mice, according to a new study. Jeannie Chew and colleagues administered an adeno-associated viral vector into the cerebral ventricles of neonatal mice. When assessed 6 months later, the mice demonstrated behavioural and motoric deficits reminiscent of ALS, and showed neuronal loss and TDP-43 inclusions. This new mouse model could be used to elucidate how the *C9orf72* repeat expansion leads to ALS, thereby revealing new avenues for treatment.

Original article Chew, J. *et al.* *C9ORF72* repeat expansions in mice cause TDP-43 pathology, neuronal loss, and behavioral deficits. *Science* **348**, 1151–1154 (2015)