Nature Reviews Neurology **8**, 660 (2012); published online 13 November 2012;
doi:10.1038/nrneurol.2012.238;
doi:10.1038/nrneurol.2012.237;
doi:10.1038/nrneurol.2012.239

IN BRIEF

ALZHEIMER DISEASE

Hormone therapy and the risk of AD—timing may be key

Conflicting data have led to uncertainty over the effect of hormone therapy on risk of Alzheimer disease (AD): some observational studies report a reduced risk, whereas clinical trials suggest an increased risk of AD following hormone therapy. In a recent population-based study, Shao *et al.* assessed whether the association between hormone therapy and AD varies with the type or timing of therapy. 1,768 women were included in the study, with follow-up over an 11-year period. During this time, 176 individuals developed incident AD. Use of hormone therapy within 5 years of menopause was associated with a 30% decrease in AD risk. However, initiation of hormone therapy beyond this 5-year window carried an increased risk of disease.

Original article Shao, H. *et al.* Hormone therapy and Alzheimer disease dementia. *Neurology* doi:10.1212/WNL.0b013e318271f823

NEURO-ONCOLOGY

Invasiveness of glioblastoma cells is driven by deregulation of microRNA-376 editing

Deregulation of microRNAs (miRNAs)—short, noncoding RNAs involved in gene silencing—in glioblastoma cells could switch their role from inhibiting to promoting cell invasiveness, according to a recent study. Small epigenetic changes in miRNAs, such as replacement of adenosine with inosoine (A-to-I editing), generates variant or 'edited' miRNAs, and evidence suggests that the frequency of A-to-I editing is reduced in glioblastoma. The miRNA-376 cluster expressed in human brain has nine adenosine residues susceptible to such editing. Choudhury and colleagues found that unedited miRNA-376a* accumulated in high-grade gliomas and promoted migration and invasivesness compared with the edited form, which suppressed these features. miRNA-376a* is proposed as a therapeutic target in glioblastoma cells.

Original article Choudhury, Y. *et al.* Attenuated adenosine-to-inosine editing of microRNA-376a* promotes invasiveness of glioblastoma cells. *J. Clin. Invest.* doi:10.1172/JCI62925

EPILEPSY

Low levels of trace elements found in paediatric epilepsy

Paediatric epilepsy is associated with a deficiency of trace elements, including selenium (Se), Zinc (Zn), chromium (Cr) and Iron (Fe), according to two recent studies. Such elements participate in the formation of enzymes that prevent accumulation of free radicals—an event that may lead to seizures and increase the risk of seizure recurrence. Seven *et al.* examined serum levels of trace elements in 70 patients with idiopathic intractable epilepsy and 60 healthy children. Patients had significantly lower levels of serum Se and Zn than did healthy controls. Wojciak *et al.* observed a reduction of serum Zn and Cr concentrations in all 23 paediatric patients examined, together with low levels of Fe in female patients. Such findings provide insight into the aetiology of paediatric epilepsy, and indicate a need for trace-element monitoring in this disorder.

Original articles Seven, M. *et al.* Deficiency of selenium and zinc as a causative factor for idiopathic intractable epilepsy. *Epilepsy Res.* doi:10.1016/j.eplepsyres.2012.09.013 | Wojciak, R. W. *et al.* The serum, zinc, copper, iron and chromium concentrations in epileptic children. *Epilepsy Res.* doi:10.1016/j.eplepsyres.2012.09.009