Functionally different antiepileptic drugs have differing effects on male sexual function

Men with epilepsy often suffer from low sexual drive and potency. A recently published study suggests that enzyme-inducing antiepileptic drugs (AEDs) could exacerbate this problem by causing serum estradiol levels to rise, leading to reduced testosterone synthesis and impaired sexual function. Non-enzyme-inducing AEDs, on the other hand, could help prevent these effects.

Herzog *et al.* measured various indicators of sexual function and reproductive hormones in four groups of men with localization-related epilepsy (25 taking the non-enzyme-inducing AED lamotrigine, 25 taking carbamazepine and 25 phenytoin—both enzyme-inducing AEDs—and 10 'untreated' individuals who had taken no AED for at least 6 months before analysis) and 25 nonepileptic controls. All men were aged between 18 and 50 years. A high proportion of men with epilepsy scored below the range of results seen in the control group on various indicators of sexual function. Lamotrigine treatment, however, was associated with levels of sexual function, bioactive testosterone and gonadal efficiency comparable to those of controls and untreated epileptics, and significantly better than those in patients on carbamazepine or phenytoin ($P \leq 0.05$). Lamotrigine also produced a much smaller decline in bioactive testosterone with age than did the non-enzyme-inducing AEDs.

The authors note that the lower-than-normal bioactive testosterone levels in untreated epileptics could be due to their previous enzyme-inducing AED treatment rather than to epilepsy itself. In addition, some AEDs might have direct toxic effects on testicular function. The relationship between low sexual function scores, low bioactive testosterone levels and AED use requires further study.

Pippa Murdie

Original article Herzog AG *et al.* (2005) Differential effects of antiepileptic drugs on sexual function and hormones in men with epilepsy. *Neurology* **65:** 1016–1020

The EphA2 receptor in glioblastoma multiforme

Researchers in the USA recently investigated expression of the EphA2 receptor and its ligand ephrinA1 in glioblastoma multiforme (GBM) cells. EphA2 and ephrinA1 have previously been associated with the endothelial cells of tumor neovasculature, and the results of this study indicate that EphA2 could be a reliable marker of GBM.

Wykosky *et al.* quantified the expression of EphA2 and ephrinA1 in human GBM cells and normal brain using western blotting and immunohistochemical techniques. They found that in the vast majority of cases the level of EphA2 was much higher in GBM tumors than in normal brain tissue. By contrast, ephrinA1 was expressed at a very low level in both GBM and normal brain specimens.

By growing GBM cell lines demonstrating high levels of EphA2 expression either in the presence or absence of ephrinA1, the authors found that ephrinA1 had a dose-dependent inhibitory effect on the growth of GBM cells. The effect of exogenous ephrinA1 exposure on the invasive quality of high EphA2-expressing GBM cells was also investigated, and it was found that higher ephrinA1 concentrations were associated with a reduction in the invasiveness of GBM cells.

The authors suggest that the overexpression of EphA2 in GBM cells—possibly resulting from decreased interaction between the receptor and the inhibitory ephrinA1 in malignant cells—has importance in the oncogenic properties of GBM. They propose that the EphA2/ephrinA1 system could be a novel target for the development of molecular therapeutic interventions against GBM.

Christine Kyme

Original article Wykosky J *et al.* (2005) EphA2 as a novel molecular marker and target in glioblastoma multiforme. *Mol Cancer Res* **3:** 541–551

Early cardiac screening and genetic analysis in patients with muscular dystrophy

Dilated cardiomyopathy—the most frequent cardiac abnormality in Duchenne muscular dystrophy (DMD) and Becker muscular dystrophy (BMD)—can lead to congestive heart failure or premature death.

Jefferies *et al.* have investigated whether early diagnosis and treatment of dilated cardiomyopathy can lead to ventricular remodeling in boys with DMD and BMD. They also assessed