

T cells were found in mice treated with 3,4-DAA after onset of EAE than in control mice, and 3,4-DAA-treated mice also had fewer and less severe relapses of disease. Adoptive transfer of activated lymph node cells from mice with EAE that had been treated with tryptophan catabolites delayed EAE onset and reduced EAE symptoms in recipient mice that had not been treated with these catabolites. Lymph-node cells from mice treated with tryptophan catabolites produced less interferon- $\gamma$  and more interleukin-10 than vehicle-treated mice.

The data indicate that tryptophan metabolites can slow the progression of EAE, possibly through altering the profile of secreted cytokines and reducing the activation of local antigen-presenting cells. Derivatives of tryptophan catabolites could form a new class of drugs for the treatment of autoimmune disease mediated by type 1 T-helper lymphocytes.

Kate Matthews

**Original article** Platten M *et al.* (2005) Treatment of autoimmune neuroinflammation with a synthetic tryptophan metabolite. *Science* **310**: 850–855

## Vitamin B<sub>2</sub> shows promise as a treatment for traumatic brain injury

Vitamin B<sub>2</sub> (riboflavin) is a potential treatment for traumatic brain injury (TBI), according to the Restorative Neuroscience Laboratory from Southern Illinois University, Carbondale, USA.

Hoane and colleagues compared the effects of B<sub>2</sub> and saline administration in 41 rats that had undergone cortical contusion injury (CCI)—an experimental model of TBI—or sham surgery. Animals were tested on aspects of behavioral ability such as sensorimotor performance (measured with the bilateral tactile removal test) and acquisition of reference and working memory (measured with the Morris water maze). The researchers also investigated whether B<sub>2</sub> administration affected lesion volume, edema formation and levels of glial fibrillary acidic protein (GFAP) expression after CCI.

Compared with saline, B<sub>2</sub> administration significantly reduced the deleterious effects of CCI on sensorimotor performance ( $P < 0.001$ ), and also produced marked improvements in the acquisition of reference and working memory. Immunohistochemical analysis of GFAP expression around the CCI lesion sites revealed significantly lower numbers of GFAP<sup>+</sup>

astrocytes after B<sub>2</sub> administration ( $P < 0.001$ ). Edema formation and lesion size were also significantly reduced after B<sub>2</sub> administration ( $P < 0.001$  and  $P < 0.03$ , respectively).

Previous studies from this laboratory indicated that vitamin B<sub>3</sub> holds promise as a therapy for TBI. On the basis of these new findings, the authors propose that vitamin B<sub>2</sub> also merits further examination as a potential treatment for patients with TBI.

Christine Kyme

**Original article** Hoane MR *et al.* (2005) Administration of riboflavin improves behavioral outcome and reduces edema formation and glial fibrillary acidic protein expression after traumatic brain injury. *J Neurotrauma* **22**: 1112–1122

## Distinguishing VV1 sporadic CJD from variant CJD

Six sporadic Creutzfeldt–Jakob disease (CJD) subtypes have been identified by genetic and molecular analysis; they differ in disease course, age of onset and diagnostic features. The rarest of these subtypes—VV1—exhibits a prolonged disease course, young age of onset, psychiatric symptoms and relatively slow progressive dementia. Meissner and colleagues attempted to further characterize the VV1 subtype to enable it to be distinguished from variant CJD, which shows similar clinical features.

From 1993 to 2003, nine VV1 cases (eight male) were diagnosed at the CJD Surveillance Unit in Göttingen, Germany. Median age at onset was 44 years (range 19–55 years), and median duration of illness was 21 months (range 17–41 months).

In addition to dementia, all nine patients exhibited personality changes during the study, including aggressive behavior, childish behavior and fear. Focal neurological deficits were seen in five patients, including hemiparesis and hemineglect. The 14-3-3 protein was detected in the cerebrospinal fluid in all eight cases tested, but there were no characteristic electroencephalographic findings. Cortical signal increase on MRI was detected in all patients, with the temporal cortex, hippocampus and insula affected most often.

The authors conclude that the VV1 subtype might be distinguished by the presence of the 14-3-3 protein in the cerebrospinal fluid and by the MRI signal increase in the hippocampus, as these findings are rather atypical in variant CJD.