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IN BRIEF

TRAUMATIC BRAIN INJURY

Minocycline reduces microglial activation but increases neurodegeneration after TBI

Chronic microglial activation (CMA) has been observed in conjunction with neurodegeneration after traumatic brain injury (TBI), prompting researchers to explore CMA inhibition as a possible therapeutic strategy. In a randomized, open-label trial that was recently reported in *Brain*, David Sharp and colleagues examined the effects of the antibiotic minocycline on CMA and neurodegeneration in 15 patients who had sustained a TBI at least 6 months earlier. The investigators found that minocycline treatment reduced CMA but, somewhat surprisingly, was also associated with increases in brain atrophy and plasma levels of neurofilament light protein, both of which are indicative of neurodegeneration. These findings raise the prospect that microglial activation has a beneficial role after TBI, which might be harnessed to improve patient outcomes.

ORIGINAL ARTICLE Scott, G. *et al.* Minocycline reduces chronic microglial activation after brain trauma but increases neurodegeneration. *Brain* http://dx.doi.org/10.1093/brain/awx339 (2017)

STROKE

Kv1.3 inhibition shows therapeutic potential in animal models of ischaemic stroke

Inhibition of the voltage-gated K^+ channel Kv1.3 can reduce secondary brain injury after ischaemic stroke, according to a new study published in *Annals of Clinical and Translational Neurology*. Kv1.3 is upregulated on activated 'M1-like' microglia, which are thought to contribute to inflammatory damage following brain ischaemia and reperfusion. Researchers at the University of California, Davis, USA, tested the small-molecule Kv1.3 inhibitor PAP-1 in mouse and rat models of ischaemic stroke. They found that the drug reduced the size of the infarct and ameliorated neurological deficits in both models. The authors conclude that Kv1.3 inhibition is a promising approach, but it needs to undergo additional testing in models that more closely resemble human stroke before it can enter clinical trials.

ORIGINAL ARTICLE Chen, Y.-J. *et al.* Inhibition of the potassium channel Kv1.3 reduces infarction and inflammation in ischemic stroke. *Ann. Clin. Transl Neurol.* http://dx.doi.org/10.1002/acn3.513 (2017)

DEMENTIA

Peripheral inflammation could be a prodromal indicator of dementia

Evidence is accumulating that the pathogenesis of Alzheimer disease (AD) and other neurodegenerative conditions involves mechanisms that operate outside the brain, and new research indicates that peripheral inflammation is an early event in the disease course of both AD and dementia with Lewy bodies (DLB). Eleanor King and co-workers at Newcastle University, UK, measured levels of inflammatory cytokines in plasma samples from 37 patients with DLB, 20 patients with AD, 48 patients with mild cognitive impairment (MCI) — 38 with a DLB profile and 20 with an AD profile — and 20 healthy controls. Levels of the inflammatory markers IL-1β, IL-2, IL-4 and IL-10 were elevated in both of the MCI groups but not in the patients with more-advanced disease. These findings suggest that the prodromal phases of AD and DLB provide a window of opportunity for intervention with anti-inflammatory therapies.

ORIGINAL ARTICLE King, E. *et al.* Peripheral inflammation in prodromal Alzheimer's and Lewy body dementias. *J. Neurol. Neurosurg. Psychiatry* http://dx.doi.org/10.1136/jnnp-2017-317134 (2017)