061 NEUROINFLAMMATION AND AMYLOID PATHOLOGY AFTER TBI

Gregory Scott, ¹ David J Sharp, ¹ Anil Ramlackhansingh, ¹ Peter Hellyer, ¹ Rob Leech, ¹ Richard Greenwood, ² Federico Turkheimer, ¹ Rolf Heckemann, ³ Paul Matthews, ¹ David Brooks ¹. ¹Division of Brain Sciences, Imperial College London; ²Institute of Neurology, University College London; ³Neurodis Foundation, Centre for Medical Research with Positron Emission Tomography, Lyon, France

10.1136/jnnp-2014-309236.61

Traumatic brain injury (TBI) can lead to late neurodegeneration, including Alzheimer's Disease and Chronic Traumatic Encephalopathy. These problems are separated by many years, and the links between them are unclear. Diffuse axonal injury and neuroinflammation occur acutely but can persist, by which stage amyloid pathology may be present. Here we investigate whether these factors are linked in TBI patients with persistent neurological impairments. Using [11C]-PK11195 PET to measure microglial activation we have demonstrated chronic thalamic inflammation after TBI. We now show that this is:

- 1. positively correlated with the amount of damage in thalamocortical white matter tracts measured with diffusion tensor imaging; and
- 2. Positively correlated with amyloid pathology in the thalamus measured by [11C]-Pittsburgh compound-B (PIB). Increased PIB binding was also observed in other cortical areas, overlapping posterior parts of the default mode network. This suggests a link between damage to the connectome, persistent neuroinflammation and late neurodegeneration.