

P026 Hepcidin, an iron regulatory hormone, crosses the blood-brain barrier and provides a mechanistic link between the iron mishandling and inflammation that coexist in neurodegenerative diseases
Jing Wei Zhao¹, Animesh Alexander Raha², Jamie Marland¹, Adrian Bomford², Ruma Raha-Chowdhury¹
¹Department of Clinical Neuroscience, Cambridge Centre for Brain Repair, University of Cambridge, ²Institute of Liver Studies, King's College London School of Medicine, Cambridge, UK

Introduction: Hepcidin, a small circulatory peptide hormone, regulate systemic iron homeostasis. During inflammation it is markedly upregulated, therefore we investigated Hepcidin expression after inflammation and cortical stab injury.

Methods: Groups of rats were injected with lipopolysaccharide (100 µg/kg) to induce systemic inflammation, or mechanically injured in the cerebral cortex. Protein, mRNA expression and cell phenotypes were analysed.

Results: Hepcidin mRNA expression was upregulated in liver after LPS treatment but no expression in control brains, and dramatically high expression was found in the brain after stab injury or after LPS stimulation.

Conclusion: The iron regulatory hormone hepcidin could provide a mechanistic link between the iron mishandling and inflammation that coexist in neurodegenerative diseases. Hepcidin protein was increased in brain during acute phase systemic inflammation. It did not originate *in situ*, since little hepcidin mRNA was detectable in brain, rather it was synthesised in the liver and crossed the blood brain barrier. This provides a mechanism by which expression of Hepcidin in response to systemic inflammation can promote neuronal cell survival.