Dénes: *Intrinsic Activity*, 2016; **4**(Suppl.3):A4.21
http://www.intrinsicactivity.org/2016/4/S3/A4.21
published online: 5 September 2016

INTRINSIC ACTIVITY

22nd Scientific Symposium of the Austrian Pharmacological Society: Joint Meeting with the Hungarian Society for Experimental and Clinical Pharmacology

Vienna, 8–10 September 2016

MEETING ABSTRACT

A4.21

Neuroinflammation and brain injury: recent lessons and novel mechanisms

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Background: Inflammation is an important contributor to brain injury, but the mechanisms involved are improperly defined. Microglia, the main inflammatory cells in the brain become activated in various brain diseases, but their functional role in neuronal injury remains controversial.

Methods: Using selective microglia manipulation approaches, imaging, transgenic models and advanced microscopy, inflammatory actions through which microglia shape neuronal activity and injury can be investigated. Early inflammatory changes including microglia–neuron interactions, neuronal calcium responses, blood–brain barrier (BBB) injury, oxidative stress and perfusion changes are investigated in real time with two-photon and SPECT imaging or with super-resolution microscopy after cerebral ischemia and in other models of neuroinflammation.

Results: Microglia react rapidly to early changes in neuronal calcium responses, BBB injury and oxidative stress. Dysregulation of neuronal calcium responses is observed within 30 min after the onset of ischemia in the absence of functional microglia, leading to larger brain injury. We show that BBB injury after acute cerebrovascular events can be detected much earlier (within 2 h) than by using histology. Changes at the capillary/small vessel level as assessed by two-photon imaging show a good correlation with BBB injury seen in full brain hemispheres based on SPECT imaging studies. However, successful reperfusion after cerebral ischemia is followed by spontaneously occurring perfusion deficits later, which is further impaired by preceding systemic inflammation. Systemic inflammation also leads to larger BBB injury, which is apparent as early as 2 h after the onset of ischemia and is associated with impaired functional outcome.

Discussion: Understanding central and systemic inflammatory mechanisms is essential for the development of novel diagnostic and therapeutic tools in brain diseases.

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