Introductory Remarks to Symposium 7

Calcium homeostasis in neuroinflammation and -degeneration: new targets for therapy of multiple sclerosis?

Ricarda Diem and Sarah Williams, Heidelberg

Recent paradigm shifts in our understanding of multiple sclerosis (MS) have led to opposing hypotheses about the sequence of pathophysiological events and the identity of cell types involved in disease initiation and propagation. Irrespective of whether MS is classified as being primarily either a neuroinflammatory, a neurodegenerative or a glial disorder, calcium signals are essential for the function of all cellular systems involved including the immune system, the neurovascular unit, glial cells and neurons/axons. Additionally, calcium is not only an important messenger within specific cells, but also serves as a crucial link between different "compartments" involved in MS pathophysiology. Due to its ubiquitous role through-out all tissues and its importance for intra- as well as intercellular and network functions, understanding disturbances in calcium homeostasis would allow both the simultaneous targeting of multiple pathophysiological mechanisms in addition to the development of cell type and context-specific therapies depending upon the pathways targeted.

To this end, a team of researchers from diverse institutions and scientific fields has been assembled (comprising anatomy, biophysics, neurobiology, pharmacology, physiology as well as experimental and clinical neurology and neuroimmunology) to elucidate principle calcium-related disease mechanisms of MS, to develop cutting-edge methodologies including novel imaging techniques, and to identify new therapeutic targets. The anticipated synergistic outcome of the Research Unit 2289 will have a profound impact on the understanding of acquired channelopathies, disturbances of calcium signaling and energy imbalance under neuroinflammatory and neurodegenerative conditions. Since this consortium is focused on as yet underestimated aspects of MS pathophysiology and applies a highly interdisciplinary approach, it is expected to break new ground in clinical neurology.

FOR 2289
CALCIUM & NEUROINFLAMMATION

Symposium 7

Thursday, March 23, 2017
11:30 – 13:30, Lecture Hall 104

Chairs: Ricarda Diem and Sarah Williams, Heidelberg

11:30 **Opening Remarks**

11:40 Barbara Niemeyer, Homburg
REGULATION OF STORE-OPERATED CALCIUM ENTRY (SOCE) IN HEALTH AND DISEASE (S7-1)

12:00 Richard Fairless, Heidelberg
SOURCE AND INFLUENCE OF CALCIUM ENTRY IN RETINAL GANGLION CELLS DURING THE PRECLINICAL PHASE OF AUTOIMMUNE OPTIC NEURITIS (S7-2)

12:20 Frank Winkler, Heidelberg
ADVANCED INTRAVITAL MICROSCOPY OF CALCIUM HOMEOSTASIS AND CELLULAR INTERACTIONS IN THE CNS: FROM TUMORS TO INFLAMMATION (S7-3)

12:40 Frank Schmitz, Homburg
SYNAPTIC COMMUNICATION AT PHOTORECEPTOR RIBBON SYNAPSES OF THE RETINA: RELEVANCE FOR SIGNALLING IN THE RETINA UNDER NORMAL AND PATHOLOGICAL CONDITIONS (S7-4)

13:00 Anemari Horvat,
DISTINCT TEMPORAL CHARACTERISTICS OF INTRACELLULAR CA2+ AND CAMP/PKA RESPONSES UPON ADRENERGIC STIMULATION IN SINGLE RAT ASTROCYTES (S7-5)

13:10 Franziska Oschmann,
COMPUTATIONAL MODELING OF CA^{2+} SIGNALS IN ASTROCYTES (S7-6)

13:20 **Concluding Remarks**