Invitation

On behalf of the Hamburg Center of Neural and Cognitive Systems (HCNS), we would like to invite you

to the

24rd HCNS Lecture

February 25th 2025 | 5.15 pm

UKE, lecture hall N27, seminar room 14

We are looking forward to meeting you there!

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DEUTSCHES STIFTUNGSZENTRUM

Prof. Hans Lassmann

Medical University of Vienna

“Multiple Sclerosis and Related Disorders: What does Neuropathology tell us about Disease Mechanisms and Therapy ”

Major advances have been made in recent decades in identifying molecular neurobiological and immunological mechanisms of brain inflammation and neurodegeneration. However, their relevance for multiple sclerosis (MS) remains incompletely understood, and translation into effective therapies has been only partially successful. Neuropathology helps to bridge this translational gap by placing molecular findings into the context of lesion development in the human central nervous system, leading to a revised view of MS pathology. The traditional definition of MS as a disease of focal white matter lesions with spatial and temporal dissemination is insufficient, as similar lesions occur in other immune-mediated CNS disorders. MS-specific features include widespread subpial cortical demyelination associated with meningeal inflammation and the slow expansion of pre-existing white and grey matter lesions. The inflammatory pattern suggests a chronic, compartmentalized immune response involving tissue-resident T and B lymphocytes targeting antigens in meningeal and perivascular compartments. Chronic microglial activation contributes to oxidative injury, particularly in early active lesions. Tissue damage occurs through distal cytopathy, leading to degeneration of cellular processes and programmed cell death. Oligodendrocytes are especially vulnerable, causing demyelination, while neurons, astrocytes, and oligodendrocyte progenitor cells show degeneration, barrier disruption, and remyelination failure, respectively. These processes are likely driven by oxidative injury and mitochondrial dysfunction, resulting in “virtual hypoxia”. MS evolves through three overlapping phases: an early inflammatory phase with new lesions, a compartmentalized inflammatory phase with cortical demyelination and lesion expansion, and a later neurodegenerative phase influenced by prior inflammation, aging, and comorbidities.

The HCNS Managing Board

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