RESEARCH PROJECT

“Balancing Act: Axon Health in MS”

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Calcium dysregulation in Multiple Sclerosis

Jonas Huber is studying in the Elite Master Program “Human Biology” at the LMU Munich. As part of the program’s curriculum, students undertake two laboratory internships that last 8 weeks. For his first internship, Huber worked in the laboratory of Prof. Dr. Martin Kerschensteiner, where he contributed to the examination of calcium-mediated axon degeneration processes in multiple sclerosis (MS).

Can calcium be a promising target for MS therapies?

Calcium plays many critical roles in the nervous system. It is for instance involved in synaptic transmission, neuronal plasticity, and cellular signaling – important neurological mechanisms. Unfortunately, in the autoimmune disease MS, calcium levels are elevated inside axons, which can lead to breakage of important neural connections. The focus of Huber’s internship was to contribute to the ongoing research question on whether buffering calcium levels can prevent that axonal degeneration.

The Kerschensteiner lab works with an established mouse model, in which MS can be simulated. These mice are called EAE mice. EAE stands for experimental autoimmune encephalomyelitis. In his internship, Huber established protocols for immunohistochemistry stainings of central nervous system tissue of those mice. With such a staining, protein expression can be visualized. He aimed to determine, whether a previously induced overexpression of calcium-binding proteins was successful, and if that overexpression could have neuroprotective effects by removing excess intra-axonal calcium.

Looking at MS under the microscope

Next to protein expression studies, the laboratory uses state of the art microscopy techniques to visualize the progression of axonal breakage in MS. With transgenically fluorescently labeled axons, spinal cords of deeply anesthetized EAE mice can be imaged for several hours. Within that time, the axons can break or stay healthy. As mentioned above, calcium seems to play a critical role in that transition. Huber started with the analysis of those data to help better understand what could contribute to the maintenance of healthy axons in MS. The question on whether buffering calcium levels within the axon has a neuroprotective effect, has not finally been answered. It will be continued by his colleagues.

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