Advancing Research on Degenerative Neurological Illnesses

Manuela Neumann, whose work focuses on amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD), has accepted a new professorship at DZNE and the University of Tübingen.

Bonn/Tübingen, June, 18th, 2012. Professor Manuela Neumann has been appointed to a joint professorship at the DZNE and the University of Tübingen. In June, she has been starting a the research group “neuropathology” at the DZNE site Tübingen and will also serve as the medical director of the neuropathology department at the Institute for Pathology and Neuropathology of the medical faculty and the university clinic. In her past research work, Neumann achieved important insights for understanding the cellular mechanisms that lead to ALS and FTD. Now she is seeking to illuminate these mechanisms in greater detail in order to develop new approaches for treatment and diagnosis.

To assure the optimal translation of research into clinical practice, she intends to collaborate with other research institutions in Germany to establish a brain tissue bank. ALS and FTD are two degenerative diseases affecting the nervous system: ALS is an uncommon illness that leads to rapidly progressive muscular weakness. As the second-most common form of dementia after Alzheimer’s disease, FTD results in alterations in personality and social behavior. Based on a similar underlying mechanism. Several years ago, Manuela Neumann and her colleagues showed that changes in the DNA/RNA binding protein TDP-43 play a critical role in these degenerative diseases. In healthy individuals, most of this protein is found in the nuclei of nerve cells (neurons), where it is involved in processing genetic information (mRNA). In the neurons of patients with ALS or FTD, TDP-43 accumulates outside the nucleus, forming pathological deposits in the cytoplasm. This major discovery was published in 2006 in the renowned journal “Science”, and opened up a new area for research.

Since that time, Neumann has been investigating the precise function of TDP-43 and an additional DNA/RNA binding protein known as FUS. Her aim is to identify abnormal changes in these two proteins and to elucidate their role in cellular death as it occurs in these illnesses. In her research, she utilizes model organisms as well as methods in cellular and molecular biology in order to study the brain tissue of deceased ALS and FTD patients. “Many diseases of the nervous system, including various types of dementia and ALS, only occur in humans,” Neumann explains. “For this reason, we ultimately have to test all of our findings in human tissues.” Yet drawing inferences is also essential, Neumann states. “If we detect abnormalities in human tissues, then we investigate them using molecular biology or in an animal model.” This is an essential means for assuring the rapid transfer of laboratory findings to clinical research. In addition, by performing histological studies on tissues to establish a definitive diagnosis, she hopes to contribute to improving existing clinical methods for diagnosing several forms of dementia, including FTD.

Fig.: Pathological TDP-43 deposits (brown) in the hippocampus in a patient with ALS. Source: M. Neumann
Neumann has many years of experience in the field of neuropathology. She played a significant role in the development of the German Brain Tissue Bank Network (Brain-Net), and gained additional experience working at the Brain Bank at the University of Pennsylvania, Philadelphia (USA). The 43-year old physician and researcher completed her medical studies in Munich and Göttingen. After research stays at the University of Pennsylvania and at LMU Munich, she worked until the end of May 2012 as an Assistant Professor of Experimental Neuropathology and Senior Physician at the Institute of Neuropathology at Zurich University Hospital. Among the distinctions she has earned is the Hans und Ilse Breuer Foundation research award. Her work will be funded by the Helmholtz Association's Initiative and Networking Fund for excellent women in science.

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