Introductory Remarks to Symposium 19

From clinical symptoms to motoneuron pathobiology: most recent insights into amyotrophic lateral sclerosis (ALS)

Jochen Weishaupt and Albert C. Ludolph, Ulm

New aspects of the clinical picture of ALS together with neuropathological findings and human genetic discoveries have fostered insights into the pathogenesis of this devastating neurodegenerative disease in the past few years. A deeper understanding of the ALS phenotype could be connected to neuropathological discoveries, which strongly suggest a focal initiation of the disease followed by a sequential cortico-fugal spreading of pathology. Moreover, the discovery of key molecules involved in ALS pathogenesis by human genetics helped to identify principally novel cell biological principles. Examples are the role of aggregation-prone RNA binding proteins with prion-like properties that play a role in the formation and (dys)regulation of membrane less organelles but contribute also to the intercellular propagation of protein pathology. Possibly linked to the aspect of "RNA dysmetabolism" is the disturbance of proteostasis in ALS, which is underscored by the discovery of several ALS genes that are involved in autophagy. As a consequence, different layers of the disease, ranging from clinical symptoms to molecular pathology, could recently be integrated into a more comprehensive and complete picture and have advanced our understanding of motoneuron biology in general. Importantly, after two decades of basic research, the first gene-specific, individualized therapies are now in clinical trials for the treatment of ALS patients. In this symposium, we will highlight some of the fascinating topics of this rapidly developing field of clinically oriented neuroscience and neurology.

Symposium 19

Friday, March 22, 2019
11:30 - 13:30, Lecture Hall 104

Chairs: Jochen Weishaupt and Albert C. Ludolph, Ulm

11:30 Albert C. Ludolph, Ulm
CLINICAL TRANSLATION OF THE NEURO-ANATOMIE OF ALS (S19-1)

11:55 Jochen Weishaupt, Ulm
FROM ALS GENES TO PATHOGENIC PRINCIPLES AND TARGETS FOR INDIVIDUALIZED THERAPIES (S19-2)

12:20 Dorothee Dormann, Munich
MOLECULAR MECHANISMS OF ALS - FROM NUCLEAR TRANSPORT DEFECTS TO PROTEIN AGGREGATION (S19-3)

12:45 Karin Danzer, Ulm
TDP-43 AGGREGATION - IMPLICATIONS FOR ALS (S19-4)

13:10 Diana I. Babaevskaia, Moscow, Russia
NEUROINFLAMMATION IN A MOUSE MODEL OF AMYOTROPHIC LATERAL SCLEROSIS WITH FUS GENE MUTATION AND EFFECTS OF STANDARD AND NEW THERAPIES (S19-5)

13:20 Diane Penndorf, Jena
REPLICATIVE REPROGRAMMING IN THE CONTEXT OF PHYSIOLOGICAL CNS AGING AND AGE-RELATED NEURODEGENERATION (S19-6)