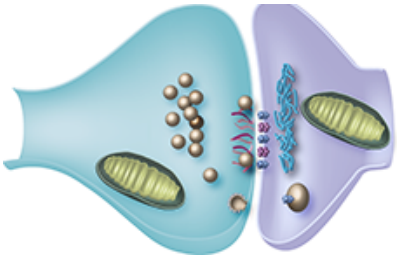

UNIVERSITÄTSMEDIZIN GÖTTINGEN UMG

SFB 1286 Quantitative Synaptology

Seminar

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Institute of Biomedical Sciences, University of Chile

ER proteostasis unbalance: from aging to neurodegenerative diseases

Tuesday, 07. May 2019 at 12:00, Seminar room,
Waldweg 33, 37073 Göttingen

Abstract: The clinical manifestation of neurodegenerative diseases is initiated by the selective alteration in the functionality of distinct neuronal populations. At the molecular level, most neurodegenerative diseases share common molecular signatures including the accumulation of misfolded proteins in the brain. Alteration in the buffering capacity of the proteostasis network is proposed as one of the triggering steps leading to abnormal protein aggregation, which is also a central hallmarks of brain aging. The endoplasmic reticulum (ER) is a major node of the proteostasis network altered in brain diseases and during aging. ER stress triggers a signaling reaction known as the unfolded protein response (UPR), which aims restoring proteostasis through the induction of adaptive programs or the activation of cell death programs when damage is chronic and cannot be repaired. Here I discuss our efforts to assess the significance of the UPR to brain aging and its contribution to disease, in addition to develop gene therapy strategies to alleviate ER stress. A new concept is emerging where depending on specific UPR component targeted and the disease model tested distinct and even opposite effects can be observed on the pathology.

Hosted by Prof. Tiago Outeiro