Introductory Remarks to Symposium 16

Mitochondrial dysfunction in neurodegeration

Ira Milosevic and Nuno Raimundo, Göttingen

Neurodegenerative diseases will become of great concern as life expectancy increases and the world population continues to age. While the precise molecular underpinnings remain elusive, among the commonalities of several neurodegenerative diseases (e.g. Parkinson, Alzheimer, Lewi-body dementia, etc.) is mitochondrial dysfunction within neuronal and glial cells. Mitochondria are key organelles in cellular metabolism, long established as the cellular ATP production engines. The role of mitochondria as signaling platforms is also prominent: these organelles coordinate several cellular processes, such as autophagy and cell death, by modulating signaling pathways and/or via physical contact sites with other organelles (e.g. lysosomes, ER, peroxisomes). Thus, a detailed understanding of mitochondrial function and pathology in the brain in the context of neurodegenerative disorders and ageing is pivotal. This symposium will highlight recent advances in the understanding of mechanisms that control mitochondrial function and dynamics, and the use of novel animal models and innovative technologies to access mitochondrial dysfunction.
Specifically, the integrity of the mitochondrial network is maintained by mitophagy, which is pivotal for proper functioning of the synapse. Nektarios Tavernarakis (Greece) will shed light on the mitochondrial turnover and homeostasis in ageing and neurodegeneration. Another level of mitochondrial quality control takes place at the level of protein homeostasis within the organelle, by a number of dedicated proteases, whose dysfunctions have profound consequences to mitochondrial morphology and function. Thomas Langer (Germany) will report on the metabolic and regulatory roles of mitochondrial proteostasis, and on consequences of its defects for neurodegenerative phenotypes. Elena Rugarli (Germany) will take us to the role of mitochondria in axon degeneration, which is a key feature of many neurodegenerative diseases. Patrik Verstreken (Belgium) uses fruit flies to address mitochondrial glitches that lead to synaptic malfunction and neurodegeneration, which is particularly relevant for the pathology of Parkinson's disease. In sum, this symposium will cover diverse aspects concerning the mechanisms of mitochondrial dysfunction in neurodegenerative diseases, building bridges between different systems of mitochondria quality control and different model systems, thereby providing the state-of-the art in the field and the path of its development in the short- and longer-term future.

Symposium 16

Thursday, March 21, 2019
14:30 - 16:30, Lecture Hall 105

Chairs: *Ira Milosevic and Nuno Raimundo, Göttingen*

14:30 Thomas Langer, Cologne
PROTEOLYTIC CONTROL OF MITOCHONDRIAL DYNAMICS AND NEURODEGENERATION (S16-1)

14:55 Elena Rugarli, Cologne
CLUH IS A POST-TRANSCRIPTIONAL REGULATOR OF MITOCHONDRIAL FUNCTION (S16-2)

15:20 Nektarios Tavernarakis, Heraklion, Greece
MITOCHONDRIAL TURNOVER AND HOMEOSTASIS IN AGEING AND NEURODEGENERATION (S16-3)

15:45 Jorge De Sousa Valadas, Leuven, Belgium
THE ORIGIN OF SLEEP DEFECTS IN PARKINSON DISEASE (S16-4)

16:10 Sindhuja Gowrisankaran, Göttingen
ROLE OF RABCONNECTIN-3a IN VESICLE ACIDIFICATION, TRAFFICKING AND NEURODEGENERATION (S16-5)

16:20 King Faisal Yambire, Göttingen
LYSOSOMAL AND MITOCHONDRIAL CROSSTALK: A CASE FOR NEURODEGENERATION IN LYSOSOMAL STORAGE DISEASES? (S16-6)