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The NO-Age and NO-AD Seminar Series # 71

The development of *C. elegans* models for the study of Alzheimer's disease

by

Dr. Janine Kirstein
Department of Cell Biology, Universität Bremen, Germany

'**Protein homeostasis and healthy ageing**'

by

Dr. Johnathan Labbadia,
Institute of Healthy Ageing, University College London, UK

10:00-12:00 (CET), Monday, 12th June 2023
Room: Ahus B2: Grupperom B203.007
On-line: Registration ahead
https://uio.zoom.us/webinar/register/WN_V5ETQbZuQ5mCUcM5EGj9lQ

Organizers:
Evandro F. Fang (UiO), Jon Storm-Mathisen (UiO), Lene Juel Rasmussen (KU), W.Y. Chan (CUHK)
Queries: e.f.fang@medisin.uio.no
Previous recorded talks are available here: https://noad100.com/videos-previous-events/

Name: Prof. Janine Kirstein

Institute: Department of Cell Biology, Universität Bremen, Germany

Email: kirsteinuni@bremen.de

Lab website: https://www.uni-bremen.de/en/zellbiologie/team

Speaker: Prof. Janine Kirstein

Title: The development of *C. elegans* models for the study of Alzheimer's disease

Abstract:
To be updated

Biography:
Prof. Janine Kirstein's lab studies Huntington's and Alzheimer's disease using the nematode as well as cell culture and in vitro techniques. The long-term health of all metazoan cells is inextricably linked to protein quality control. An imbalance in protein homeostasis (proteostasis) can result in severe molecular damage to the cell, directly leading to tissue pathology and to enhanced susceptibility to diseases not only including metabolic disease and neurodegeneration, but also cancer and immunodeficiency. Our group aims to uncover the complex mechanisms of the proteostasis network, strategies to cope with protein aggregates and its changes with aging.

Name: Dr. Johnathan Labbadia

Institute: Institute of Healthy Ageing; Genetics, Evolution and Environment; University College London, UK

Email: j.labbadia@ucl.ac.uk
Web: https://www.ucl.ac.uk/biosciences/people/dr-john-labbadia

Speaker: Dr. Johnathan Labbadia
Title: Choosing the right path in life: manipulating mitochondria to enhance proteostasis capacity and promote healthy ageing

Abstract:
Cells routinely maintain proteome integrity through the action of the Proteostasis Network (PN), a collection of protein quality control mechanisms and stress response pathways that spans all compartments of the cell. As cells age, the ability of the PN to react and respond to environmental and physiological challenges declines, leaving tissues vulnerable to the appearance and persistence of misfolded, mislocalised and aggregated proteins. This “loss of protein homeostasis (proteostasis)” is at the heart of numerous age-related diseases. Therefore, finding ways to preserve the capacity of the PN and maintain proteome integrity with age may be a powerful way to promote long-term tissue health. Using the nematode worm *Caenorhabditis elegans* as a model system, we have discovered that mitochondrial activity is intimately coupled to the composition and capacity of the PN. Intriguingly, we find that different forms of mitochondrial stress remodel the PN through distinct mechanisms involving the PP2A complex, programmed cell death factors, mitochondrial sirtuins and chromatin remodelling factors. In this seminar, I will present my groups evidence for the existence of two new mitochondria-to-HSF1 stress response pathways and discuss the relevance of these mechanisms for our understanding of basic cell biology and our ability to protect the ageing proteome.

Biography:
Dr. John Labbadia is a BBSRC David Phillips Fellow within the Department of Genetics, Evolution and Environment, at University College London. John has dedicated his research career to understanding how cells maintain protein homeostasis, first as a PHD student working on the molecular basis of Huntington’s Disease with Prof. Gillian Bates’ at Kings College London, and then as an ALS Association Postdoctoral Fellow in Prof. Rick Morimoto’s laboratory at Northwestern University, USA, where he began using the roundworm *Caenorhabditis elegans* to investigate the origins of age-related protein homeostasis collapse. Following this, John was awarded a prestigious BBSRC David Phillips Fellowship to establish his own lab within the Institute of Healthy Ageing at UCL. John’s work has revealed that stochastic and programmed changes in the activity of protein quality control pathways, can leave cells vulnerable to the build-up of toxic proteins with age, thereby resulting in disorders such as Huntington’s, Parkinson’s and Alzheimer’s disease. Thanks to funding from the BBSRC, the Academy of Medical Sciences and the Wellcome Trust, his group are continuing to focus on understanding how cells prevent toxic proteins from accumulating, and how these mechanisms can be exploited to suppress the occurrence of age-associated diseases throughout the population.