



U. of  
Oslo

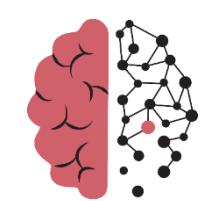
U. of  
Copenhagen

Chinese U. of  
Hong Kong

Norwegian U. of  
Science and Technology

K.G. Jebsen Centre for  
Alzheimer's Disease

Kavli Institute for  
Systems Neuroscience



# The NO-Age and NO-AD Seminar Series 017

## ‘Cellular stress responses – signals, sensors and outcomes’

by

Prof. Simon Bekker-Jensen

Center for Healthy Aging, University of Copenhagen, Denmark

at

14:00-15:00 (CET), Monday, 3<sup>rd</sup> May 2021

Register in advance for this webinar:

[https://uio.zoom.us/webinar/register/WN\\_09NEWQIvTw2Hwxj6ggEEGg](https://uio.zoom.us/webinar/register/WN_09NEWQIvTw2Hwxj6ggEEGg)

Organizers:

Evandro F. Fang (UiO), Jon Storm-Mathisen (UiO), Menno P. Witter (NTNU),  
Lene Juel Rasmussen (KU), W.Y. Chan (CUHK)

Queries: [e.f.fang@medisin.uio.no](mailto:e.f.fang@medisin.uio.no)

Previous recorded talks are available here: <https://noad100.com/videos-previous-events/>



**Speaker: Simon Holst Bekker-Jensen**

**Title: Cellular stress responses – signals, sensors and outcomes**

**Abstract:**

In response to harmful environmental conditions and other stresses, cells can mount stress responses that rewire cellular functions to cope with these challenges. Besides serving as acute defence mechanisms, such responses are also required to maintain the fitness of cells over time by constantly responding to low levels of stress originating from e.g. oxidative byproducts of metabolism and gradual age-dependent decay of key cellular constituents. Consequently, the ability of cells to cope with such insults is tightly linked to the ageing process both on the cellular and organismal level. The MAP kinases p38 and JNK are central transducers of a subset of these responses, activating a plethora of signalling pathways that collectively act to modulate and rewire cellular function to cope with stress. How these kinases are activated by endogenous stress signals is only poorly elucidated and this represents a considerable gap in our knowledge about MAP kinase signalling pathways.

Here, I will present our data on how a poorly described MAP3 kinase, ZAK, acts as a key sensor for several physiologically relevant stress signals. The long isoform ZAK-alpha is activated upon interference with ribosome function, a property that protects *C. elegans* when challenged with a host of stress conditions. The short isoform ZAK-beta, does not contain ribosome-binding domains, but instead react to mechanical perturbations of cells via a distinct sensor domain. This function is specific to vertebrate organisms and protects against muscle pathology in mice and humans.

In sum, our efforts will continue to establish novel connections between molecular stress signals, the underlying sensing mechanisms and their physiological relevance.

**Biography:**

Simon Bekker-Jensen has a PhD in Cancer Biology from the Technical University of Denmark and the Danish Cancer Research Centre. Prior to establishing his independent group within cellular stress signaling in 2016, he has 10 years of experience in research within the DNA damage response. Amongst others, he holds a Consolidator Grant from The European Research Council and is an EMBO Young Investigator.