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 | NO-Age | NO-AD | MIT-AD

The NO-Age and NO-AD Seminar Series 048

'Mechanistic exploration of Alzheimer's disease using iPSC techniques ' (tentative)

by

Prof. Christopher D Link

Department of Integrative Physiology, University of Colorado Boulder, USA

at

14:00-15:15 (CET), Monday, 18th April. 2022

Register in advance:

https://uio.zoom.us/webinar/register/WN_3-jxuiYcRZWY2ffJ8mUhdg

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. Christopher D Link
Institute: Department of Integrative Physiology
University of Colorado Boulder
Email: linkc@colorado.edu
Lab website:
https://www.colorado.edu/neuroscience/christopher-d-link

Speaker: Prof. Christopher D Link

Title: 'Mechanistic exploration of Alzheimer's disease using iPSC techniques ' (tentative)

Abstract: To be updated

Biography:

Dr. Link's background is originally in molecular genetics and developmental biology, and his lab initially began using the invertebrate worm Caenorhabditis elegans for classic developmental genetic studies. In 1992, Dr. Link's group initiated a new series of experiments by transgenically engineering C. elegans to express the human beta amyloid peptide, which is centrally involved in the pathogenesis of Alzheimer's disease. In this model, the beta amyloid peptide accumulates intracellularly, forms amyloid, and results in cellular pathology. The goal of his studies is to understand the molecular and cellular basis of this toxicity, and to investigate how these processes might be involved in Alzheimer's disease. Dr. Link takes advantage of experimental tools available in C. elegans, such as forward genetic screens, microarray-based gene expression studies, and dsRNA-based gene inhibition, to help identify specific genes involved in beta amyloid toxicity.