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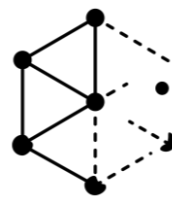
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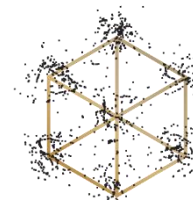
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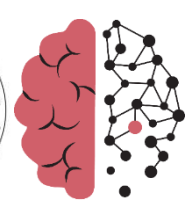
K.G. Jebsen Centre for
Alzheimer's Disease



Kavli Institute for
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NO-Age



NO-AD



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The NO-Age and NO-AD Seminar Series 38

'Convergent mechanisms of longevity'

by

Prof. Dr. Adam Antebi

Max Planck Institute for Biology of Ageing, Cologne, Germany

at

14:00-15:15 (CET), Monday, 14th June 2021

Registration ahead

https://uio.zoom.us/webinar/register/WN_6TQ-KJd9Q1mWixE3kWWAPA

Organizers:

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

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



Speaker: Adam Antebi, Max Planck Institute for Biology of Ageing, Cologne, Germany

Title: Convergent mechanisms of longevity

Research in model organisms over the last several decades has revealed that animal lifespan is plastic and regulated by conserved metabolic signaling pathways, which work through specific transcription factors to extend life. Whether these pathways affect common downstream mechanisms remains largely elusive. Our studies demonstrate that NCL-1/TRIM2/Brat tumor suppressor extends lifespan and limits nucleolar size in the major *C. elegans* longevity pathways, as part of a convergent mechanism focused on the nucleolus. Animals representing distinct longevity pathways exhibit small nucleoli, and decreased expression of rRNA, ribosomal proteins, and the nucleolar protein fibrillarin, dependent on NCL-1. Fibrillarin is not only a marker but itself is a causal factor whose knockdown reduces nucleolar size and extends lifespan. Long-lived dietary restricted fruit flies and insulin-like-peptide mutants also exhibit reduced nucleoli and fibrillarin expression. Similarly, tissues derived from long-lived dietary restricted and reduced insulin/IGF signaling IRS1 knockout mice, and humans who undergo modest dietary restriction coupled with exercise display reduced nucleoli. We suggest that small nucleoli are a cellular hallmark of longevity and metabolic health conserved across taxa.

Biography:

Dr. Antebi received his PhD in Biology from Massachusetts Institute of Technology in 1992, and performed his post-doctoral studies at the Johns Hopkins University Baltimore, MD. From 1997 he worked as a Max Planck Independent Group Leader at the Max Planck Institute for Molecular Genetics, Berlin, and then moved to Baylor College of Medicine, Houston, Texas becoming an Associate Professor in 2007. Dr. Antebi is currently one of the founding directors of the Max Planck Institute for Biology of Ageing in Cologne, Germany. He is also an Honorary Professor at the University of Cologne, Center of Excellence Cluster on Cellular Stress Responses and Ageing-associated Disease. Dr. Antebi's work has focused on regulation of longevity in the small roundworm, *Caenorhabditis elegans*, an important model system for aging research. Among his findings, he has discovered that components of hormone driven developmental clocks regulate animal life span and that small nucleoli are a cellular hallmark of longevity across taxa. He has served as an editor-in-chief of the journal *Aging Cell*, and on the scientific advisory boards of the Friedrich Miescher Institute, Basel, Fritz Lipmann Institute, Jena, Neuromyogene University of Lyon, Nathan Shock Center, University of Michigan and served on commissions for the appointment of Wellcome Trust Investigators and Max Planck Directorships. He helped established the Cologne Graduate School of Ageing Research, and the Systems Biology of Ageing Network, Cologne. Dr. Antebi is a member of EMBO, a recipient of the prestigious ERC Advanced Grant, Ellison Medical Foundation Senior Scholar in Aging Award, the Runnstrom Lecture, the Paul Glenn/American Federation for Aging Research Breakthrough in Gerontology Award, the ADPS Longevity Research Award, and the Bennett J. Cohen Award in Aging Research.

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<https://www.mpg.de/365410/biology-of-ageing-antebi>

Photo: from the speaker