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Minisymposium on Mitochondrial Genome Dynamics and Aging

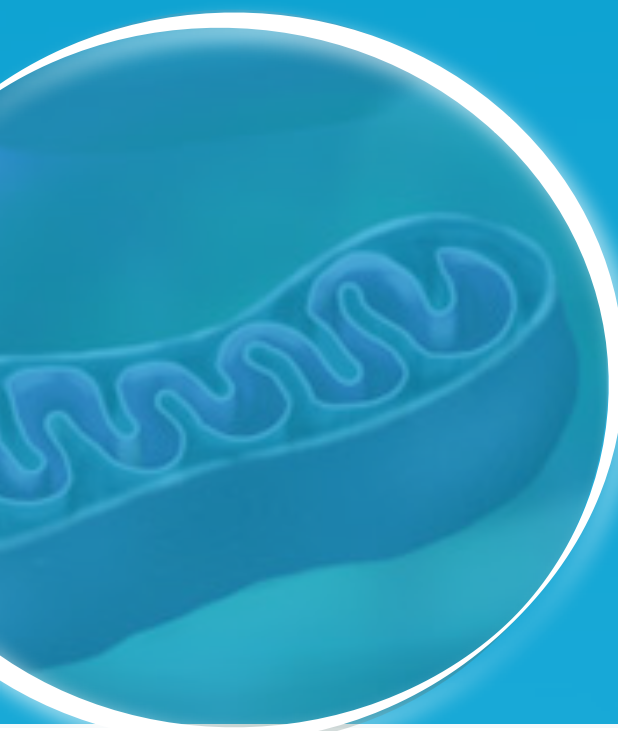
January 21st and 22nd, 2010
University of Aarhus, Lakeside Auditorium #2

Organizers

Tinna Stevnsner
Lene Juel Rasmussen
Vilhelm A. Bohr

Mitochondria are organelles that serve as centres for cellular energy production, ion homeostasis, and apoptosis. During the aging process the mitochondrial function gradually declines, and damage accumulates in the mitochondrial DNA.

Mitochondrial DNA damage is potentially critical and may result in inefficient ATP production and increased release of reactive oxygen species. An elevated level of cellular oxidative stress is observed in tissues and cultured cells from elderly individuals. Recently, mitochondrial dysfunction has also been implicated in a variety of human age-associated diseases, including diabetes, cancer, and neurodegeneration. Thus, mitochondrial genome dynamics is an important player in the aging process, but the precise mechanisms involved still remain unclear. This minisymposium is focused on recent advances in understanding the mechanisms involved in mitochondrial genome dynamics and alterations associated with the aging process.



Programme:

Thursday January 21

Session I

Chair: Tinna Stevnsner

10.00-10.05 Tinna Stevnsner and Lene J. Rasmussen:

10.05-10.20 Vilhelm A. Bohr:

10.20-10.50 Alexander Bürkle, Germany:

10.50-11.10 Henrik Rossing, Denmark:

11.10-11.45 Coffee

Welcome

Advances in mitochondrial DNA repair

Association of mitochondrial antioxidant enzymes with mitochondrial DNA as integral nucleoid constituents

The daily life with Cockayne Syndrome

Session II

Chair: Lene J. Rasmussen

11.45-12.15 Nils-Göran Larsson, Germany:

12.15-12.45 William Copeland, USA:

12.45-13.05 Marit Otterlei, Norway:

13.05-14.30 Lunch

Mutations in mtDNA in disease and ageing

Mitochondrial Diseases from mutations in DNA polymerase gamma

Base Excision Repair in mitochondria

Session III

Chair: Vilhelm A. Bohr

14.30-15.00 Bennett Van Houten, USA:

15.00-15.30 Thomas von Zglinicki, UK:

15.30-16.00 Coffee

16.00-16.20 Lars Eide, Norway:

16.20-16.40 Bjørn Quistorff, Denmark:

16.40-17.00 Per Bo Jensen, Denmark:

ROS, mitochondrial DNA damage and bioenergetics neurodegenerative diseases

Nuclear DNA damage response and mitochondrial dysfunction – chicken and egg in cell senescence

Impaired mitochondrial DNA integrity alters differentiation outcome of neural stem/progenitor cells in vitro

Foetal programming of mitochondrial function - for life ?

Measurement of mitochondrial function/dys-function

Programme:

Friday January 22

Session IV

Chair: Lene J. Rasmussen

9.00-9.30 Albert Gjedde, Denmark:

9.30-9.50 Ricardo Gredilla, Spain:

9.50-10.10 Deborah Croteau, USA:

10.10-10.40 Bernd Epe, Germany:

10.40-11.10 Coffee

Aging and Uncoupling of Human Brain Energy Metabolism in Vivo
Brain aging and DNA repair: mitochondrial base excision
repair at the synapses
RECQ Helicase In Mitochondria?
Endogenous oxidative DNA base damage in mitochondrial and
nuclear DNA of mouse liver

Session V

Chair: Tinna Stevnsner

11.10-11.40 Claudio Franceschi, Italy:

11.40-12.00 Peter Bross, Denmark:

12.00-12.20 Claus Desler, Denmark:

12.20-12.40 Peter Møller, Denmark:

12.40-14.00 Lunch

Mitochondrial genome inherited and somatic variability in ageing,
longevity and age-related diseases
Investigations of molecular mechanisms underlying early and late
onset neurodegeneration caused by mutations in the
mitochondrial chaperone Hsp60
Mitochondrial dysfunction and dNTP regulation
Aging and oxidatively damaged nuclear DNA in animal organs

Session VI

Chair: Vilhelm A. Bohr

14.00-14.20 Niels Gregersen, Denmark:

14.20-14.40 John Vissing, Denmark:

14.40-15.10 Keshav Singh, USA:

15.10-15.30 *Panel discussion and future directions*

Misfolded proteins and mitochondrial oxidative stress
Mitochondrial DNA dynamics and oxidative capacity in patients
with mitochondrial diseases
Inter-Genomic Cross Talk Between Mitochondria and the Nucleus
in Aging and Cancer

Please note the duration of the talk. Speakers are requested to leave at least 5 minutes of their time for discussion.