Dear All,

We are pleased to invite you to participate in '**Epigenetics & Metabolism**' international live web seminar series. Everyone is welcome.

Speaker:

Prof. Michael O. Hottiger

Professor

Department of Molecular Mechanisms of Disease (UZH)

University of Zurich, Switzerland

Topic: "Mitochondrial NAD^+ levels influence nuclear PARP1-induced ADP-ribosylation and subsequently the DNA damage response"

When: Thursday, 01. December 2022, 16:00-17:00 CET.

Where: On Zoom (Meeting ID: 867 4943 1922; Passcode: 64199593). **Free** registration
Link: https://us02web.zoom.us/webinar/register/WN_ijmLJCzYTPK0ayCVxwfzQA

Summary: In addition to its role as an electron transporter, mitochondrial nicotinamide adenine dinucleotide (NAD^+) is an important co-factor for protein ADP-ribosylation. We provide evidence that knockdown of the mitochondrial NAD^+ transporter SLC25A51 decreased the NAD^+ concentration in mitochondria but increased the NAD^+ concentration in the cytoplasm and nucleus. This NAD^+ redistribution restrained mitochondrial function and energy metabolism but increased PARP1-mediated nuclear ADP-ribosylation and allowed a faster repair of DNA lesions. Similarly, H_2O_2-induced oxidative stress induced strong nuclear ADP-ribosylation, but reciprocally reduced mitochondrial NAD(H) levels. In contrast, elevation of mitochondrial NAD(H) by mitochondrial electron transport chain dampened H_2O_2-triggered nuclear ADP-ribosylation and increased MMS-induced PARP1 chromatin retention. Together, our results suggest that subcellular NAD^+ availability regulates different cellular processes in a dynamic manner and provides evidence for a NAD^+-mediated mitochondrial-nuclear crosstalk.

We are looking forward to see you at our web seminar.
Best wishes,
Indra & Carlos

Hosts: Dr. Indrabahadur Singh (German Cancer Research Center) &
Dr. Carlos Sebastian (University of Barcelona)

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