LUXEMBOURG INSTITUTE OF HEALTH
RESEARCH DEDICATED TO LIFE

UNIVERSITÉ DU LUXEMBOURG

LECTURE SERIES & WORKSHOPS 2019

CANCER RESEARCH

25
JUNE 2019
Tuesday

LECTURE
CHL Luxembourg
Amphitheatre
11.00 - 12.00 pm

MEET, GREET & EAT *
light lunch provided
CHL Luxembourg
Foyer
12.00 - 1.00 pm

9 10 11 12 1 2 3 4

* Registration is mandatory by sending an email to florence.henry@lih.lu

SPEAKER
Prof Magnar BJØRÅS
Professor, Clinic of Laboratory Medicien, University of Oslo and Oslo University Hospital and Univ of Oslo
Professor, Dept of Clinical and Molecular Medicine, Norwegian University of Science and Technology (NTNU), Trondheim
Head of Unit of Laboratory Medicine, NTNU, Trondheim

HOST:
LIH

RESPONSIBLE LIH SCIENTIST:
Simone Niclou
(simone.niclou@lih.lu)

IMPACT OF OXIDATION RESISTANCE GENE 1 (OXR1) ON NEURONAL DEVELOPMENT AND METABOLISM

ABSTRACT

Emerging evidence strongly suggest that the TLDc family of proteins, including OXR1, are very important enzymes/proteins involved in neuronal development. Several neurodegenerative diseases are linked to SNPs in OXR1 and other proteins of the TLDc family, including Parkinson disease, Alzheimer disease and Amyotrophic lateral sclerosis. The main hypothesis is that TLDc containing proteins such as OXR1 plays an important role in balancing ROS to avoid oxidative stress and development of neurological disorders and metabolic syndromes. We are using OXR1 deficient patient cells and Oxr1A deficient mice as models to investigate the impact of TLDc containing proteins on human disease, in particular neurological disorders and metabolic syndroms. We have data demonstrating that patients with mutations in the TLDc domain of OXR1 develop severe neurodegenerative disorders. Mice with brain specific isoform A knockout (Oxr1A-/-) develop fatty liver associated with reduced growth hormone (GH) levels and indications of decreased GH receptor signaling in the liver. Our results further suggest that OXR1A acts as a coactivator of protein arginine methyltransferase 5 (PRMT5) increasing symmetrical dimethylation of histone H3 arginine 2 at *Gh* promoter in the pituitary gland and, consequently, regulate *Gh* transcription. We propose a novel role of OXR1A in oxidative stress signaling as a regulator of protein arginine methylation.

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