LECTURE SERIES & WORKSHOPS
INFECTION & IMMUNITY

LECTURE
Lycée Technique
d’Esch/Alzette
Salle de Projection *

11.00 am - 12.00 pm

MEET & EAT *
light lunch provided
House of BioHealth,
Room Françoise
Barré-Sinoussi
12.30 - 2.00 pm

*Please register sending a mail to florence.henry@lih.lu

MARCH 2018
Thursday

SPEAKER
Prof Rudi BEYAERT
Full professor at Ghent University, Faculty of Sciences, Dept Biomedical Molecular Biology, Associate Director of the Center for Inflammation Research at VIB, Principal Investigator of the Unit of Molecular Signal Transduction in Inflammation at Ghent University - VIB, Ghent, Belgium

HOST:
Department of Infection and Immunity

RESPONSIBLE LIH SCIENTIST:
Prof Dirk Brenner (dirk.brenner@lih.lu)

MALT1-MEDIATED SIGNALING IN IMMUNE HOMEOSTASIS AND DISEASE

ABSTRACT

MALT1 is a signaling protein that plays a key role in innate and adaptive immunity as well as in certain malignancies. MALT1 is essential for nuclear factor-κB (NF-κB) activation downstream of T and B cell receptors, and supports T/B-cell activation and proliferation. MALT1 hyperactivation is associated with autoimmunity and specific subtypes of B-cell lymphoma. For a long time, MALT1 was believed to function solely as a scaffold protein, providing a platform for the assembly of other NF-κB signaling proteins. However, this view changed dramatically when we and others found that MALT1 also has proteolytic activity that further fine-tunes signaling. MALT1 proteolytic activity is essential for T-cell activation and lymphomagenesis, suggesting that MALT1 is a promising therapeutic target for the treatment of autoimmunity and cancer. However, MALT1 is also involved in the development of natural Treg cells that keep the immune response in check, illustrating a potential risk of therapeutic targeting of MALT1 in the context of autoimmunity. In addition, MALT1 plays a role in the activation of other immune and non-immune cells. I will present our recent work on MALT1-mediated signaling in different cell types, its role in immunity and inflammation, and possible implications for therapeutic targeting.