LUXEMBOURG INSTITUTE OF HEALTH
RESEARCH DEDICATED TO LIFE

LECTURE SERIES & WORKSHOPS

INFECTION & IMMUNITY

28
NOV 2019
Thursday

LECTURE
Lycée Guillaume Kroll
d'Esch/Alzette
Salle de Projection *
11.30 - 12.30 pm

MEET & EAT *
light lunch provided
House of BioHealth,
Room Françoise
Barré-Sinoussi
1.00 - 3.00 pm

9 10 11 12 1 2 3 4

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

SPEAKER

Prof Tim SPARWASSER

W3-Professor, Director, Institute of Medical Microbiology and Hygiene, University of Mainz Medical Center of the Johannes Gutenberg-University, Mainz, Germany

HOST:
Department of Infection and Immunity (LIH)

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

METABOLIC PROGRAMS CONTROLLING IMMUNE CELL FUNCTION

ABSTRACT

Recent advances in the field of immunometabolism support the notion that essential processes in T cell biology, such as TCR-mediated activation and T helper lineage differentiation, are closely linked to changes in the cellular metabolic programs. Although the main task of the intermediate metabolism is to provide the cell with a constant supply of energy and molecular precursors for the production of biomolecules, the dynamic regulation of metabolic pathways also plays an active role in shaping T cell responses. Key metabolic processes such as glycolysis, fatty acid and mitochondrial metabolism are now recognized as crucial players in T cell activation and differentiation, and their modulation can differentially affect the development of T helper cell lineages. We only begin to understand the diverse metabolic processes that T cells engage during their life cycle from naïve towards effector and memory T cells. Many milestone discoveries in this active area of research are based on the use of chemical inhibitors that have been shown to possess off-target effects, emphasizing the importance of genetic models to study immunometabolism. Following activation, T cells switch to fatty acid synthesis, demonstrating that de novo lipid synthesis actively supports T cell proliferation and differentiation. We could show previously that pharmacological or genetic ACC1 inhibition impairs T helper cell induction, with the strongest impact on Th17 development. Here we discuss the molecular mechanisms that link metabolic changes with the control of gene expression.

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Supported by:
Fonds National de la Recherche Luxembourg

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