LUXEMBOURG INSTITUTE OF HEALTH
RESEARCH DEDICATED TO LIFE

LECTURE SERIES & WORKSHOPS

INFECTION & IMMUNITY

14 MARCH 2019
Thursday

LECTURE
Lycée Guillaume Kroll
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

9 10 11 12 1 2 3 4

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

SPEAKER

Prof. Burkhard BECHER

Professor in chair, University of Zurich,
Institute of Experimental Immunology,
Zurich, Switzerland

THE T CELL - PHAGOCYTE INTERFACE IN TISSUE

ABSTRACT

Whereas T cells are generally thought of as mediators of tissue damage in chronic tissue inflammation, the cellular infiltrate is always dominated by myeloid cells. The granulocyte-macrophage colony-stimulating factor (GM-CSF) was initially classified as a hematopoietic growth factor. However, unlike its close relatives macrophage CSF (M-CSF) and granulocyte CSF (G-CSF), the majority of myeloid cells do not require GMCSF for steady-state myelopoiesis. Instead, in inflammation, GM-CSF serves as a communication conduit between tissue-invading lymphocytes and myeloid cells. Even though lymphocytes are in all likelihood the instigators of chronic inflammatory disease, GM-CSF-activated phagocytes are well equipped to cause tissue damage. The pivotal role of GM-CSF at the T cell: myeloid cell interface might shift our attention toward studying the function of the myeloid compartment in immunopathology and targeting specifically the crosstalk between T cells and myeloid cells through GM-CSF holds promise for the development of therapeutics to combat chronic tissue inflammation. I will discuss how GM-CSF licenses phagocytes to initiate tissue damage in chronic inflammatory diseases.

HOSTS:
Department of Infection and Immunity (LIH)

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