LUXEMBOURG INSTITUTE OF **HEALTH**
RESEARCH DEDICATED TO LIFE

LECTURE SERIES & WORKSHOPS

INFECTION & IMMUNITY

18
MAY 2017
Thursday

LECTURE
Lycée Technique
d'Esch/Alzette
Salle de Projection *
11.45am-12.45pm

MEET & EAT *
light lunch provided
House of BioHealth,
Room Françoise
Barré-Sinoussi
1.00-2.30pm

9 10 11 12 1 2 3 4

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

SPEAKER

Prof. Max LÖHNING

Immunology & Osteoarthritis Research Head, Pitzer Laboratory of Osteoarthritis Research, Charité – Universitätsmedizin Berlin Department of Rheumatology and Clinical Immunology, German Rheumatism Research Center (DRFZ), Berlin, Germany

HOST:
Department of Infection and Immunity

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

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QUANTITATIVE CONTROL OF T CELL DIFFERENTIATION AND MEMORY IN INFECTIONS

ABSTRACT

CD4+ T helper (Th) cells secrete defined amounts of specific cytokines to determine not only the type of a particular immune response, but also its intensity. By single-cell analysis and mathematical modeling, we could show that during their initial activation, individual Th cells not only 'learn' which cytokines they should produce, but also in which quantity. This Quantitative Cytokine Memory is stably maintained in memory Th cells and recalled upon secondary antigen encounter (Helmstetter et al., Immunity 2015). While analyzing the signals inducing quantitative programming of T cells, we found that the alarmin interleukin (IL)-33 determines the intensity of activation of both CD8+ cytotoxic T cells (CTL) and Th type 1 (Th1) cells upon viral infection. IL-33 is required to enhance the clonal expansion of CTLs and Th1 cells and promote their differentiation to potent, polyfunctional effector cells (Bonilla et al., Science 2012; Baumann et al., PNAS 2015; Peine et al., Trends Immunol. 2016). Moreover, we demonstrated Th2 cell plasticity and a quantitative regulation of effector functions in individual Th cells via antagonizing key transcription factors (Hegazy et al., Immunity 2010). During parasite infections, hybrid Th1/Th2 cells are generated that stably co-express key Th1 and Th2 transcription factors. As these factors inhibit each other, a hybrid cell produces rather small amounts of Th1 and Th2 effector molecules. Yet, hybrid cells support both Th1 and Th2 immune responses while inducing less immunopathologic damage than classic Th1 or Th2 cells (Peine et al., PLoS Biol. 2013).

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