LUXEMBOURG INSTITUTE OF HEALTH
RESEARCH DEDICATED TO LIFE

UNIVERSITÉ DU LUXEMBOURG

LECTURE SERIES & WORKSHOPS

CANCER RESEARCH

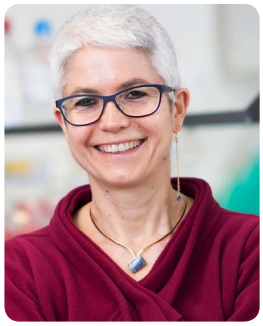
29
MARCH 2018
Thursday

LECTURE
CHL *Strassen Amphitheatre*
11.00 - 12.00 pm

MEET & EAT *
light lunch provided
BAM
Room Mc Clintock
12.30 - 14.00 pm

9 10 11 12 1 2 3 4

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



SPEAKER
Prof. Monika HEGI
Associate professor University of Lausanne and University Hospital Lausanne (CHUV)
Director of the Laboratory of Brain Tumor Biology and Genetics
Deputy director of the Neuroscience Research Centre (CRN),
Lausanne, Switzerland

HOST:
Department of Oncology
RESPONSIBLE LIH SCIENTIST:
Simone Niclou
(simone.niclou@lih.lu)

TARGETING EPIGENETIC VULNERABILITIES IN GLIOMA, BIOMARKERS AND NEW OPPORTUNITIES

ABSTRACT

Management of patients suffering from glioma remains a challenge. Over the last decade, the treatments have not changed, and consist of maximal save resection, followed by various schemes of chemo-radiotherapy. Therefore, new avenues have to be explored. The important role of epigenetic alterations in the development and treatment response of glioma has been recognized. Systematic analyses of the DNA methylome of gliomas has contributed to improved tumor classification, but has also identified vulnerabilities that can be exploited for therapy. Epigenetic silencing of the direct DNA repair gene MGMT predicts good response to alkylating agent therapy in glioblastoma, the most malignant form of glioma, and also in a subset of low-grade glioma. This can be used for stratified therapy and selection of patients into clinical trials. Additional, epigenetically altered pathways may be amenable to therapeutic targeting. In a second approach, the epigenetic landscape may be disturbed by interfering with the chromatin structure using epigenetic drugs, such as inhibitors of bromodomain and extra-terminal tail (BET) proteins. The BET proteins are chromatin readers that link the chromatin code to gene expression. Inhibition leads to changes in gene expression revealing potential pathway vulnerabilities that maybe actionable with a second drug.

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