LUXEMBOURG INSTITUTE OF HEALTH
RESEARCH DEDICATED TO LIFE

LECTURE SERIES & WORKSHOPS

INFECTION & IMMUNITY

08
SEP. 2016
Thursday

LECTURE
Lycée Technique
d'Esch/Alzette
Salle de Projection *
1.00 - 2.30 pm

WORKSHOP *
House of BioHealth
Room Françoise
Barré-Sinoussi
3.00 - 4.30 pm

10 11 12 1 2 3 4 5 6 7 8

* Registration is mandatory
by sending an email to florence.henry@lih.lu

SPEAKER
Prof Antonio IAVARONE
Professor with Tenure, Department of Neurology and Department of Pathology, Institute for Cancer Genetics, Member of Herbert Irving Comprehensive Cancer Center, Columbia University, New York

HOST:
Department of Infection and Immunity

RESPONSIBLE LIH SCIENTIST:
Prof Markus Ollert
(markus.ollert@lih.lu)

THE DRIVERS OF ONCOGENESIS AND THERAPEUTIC OPPORTUNITIES IN GLIOBLASTOMA

ABSTRACT

We are experiencing the exciting time of the genome era. For our work this means being able to contextualize the alterations of each genetic network in the natural environment of a specific tumor and identify the key driving modules on which specific tumor subgroups rely for growth, survival and progression. With this information in hand, we can target the critical alterations with specific drugs, often already available for other types of diseases. By focusing on one of the most lethal forms of human tumors, the glioblastoma multiforme (GBM), we have been able to make incredible progress along this line in the last two years.

Our work in this area first discovered two transcription factors – Stat3 and C/EBPβ –that are responsible for activation and maintenance of the most aggressive gene expression signature of high-grade glioma, the mesenchymal signature. We were able to efficiently target the two transcription factors in preclinical mouse models with consequent collapse of the mesenchymal signature and extended survival.

More recently, we have identified the first examples of highly oncogenic and recurrent gene fusions in GBM, target their dependency in a particular tumor subtype, and observe dramatic anti-tumor effects. Recurrent gene fusions in GBM result in the constitutive activation of receptor tyrosine kinase genes that render tumors addicted to the driver events. Among them, the FGFR-TACC gene fusion is the addicting oncogenic event with the highest therapeutic value.

The integrated computational-experimental pipeline that we developed plus our ability to functionalize any genetic brain tumor module was recently applied to the entire landscape of copy number variations, somatic mutations and gene fusions of human GBM. This information is quickly advancing our ability to translate each new genetic finding into the personalized context of the clinical setting.

* Opposite Luxembourg Institute of Health, House of BioHealth, 29, rue Henri Koch, L-4354 Esch/Alzette

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