SAPIENZA
UNIVERSITÀ DI ROMA

Dipartimento di Chimica
e Tecnologie del Farmaco

AVVISO DI CONFERENZA

Si comunica che il giorno 28 Maggio 2019, alle ore 14, nell'Aula Magna – F. Cacace della Facoltà di Farmacia e Medicina (Edificio CU019) dell'Università Sapienza il

Prof. Francesco Bertoni

(Università della Svizzera italiana, Institute of Oncology Research (IOR), Bellinzona, Switzerland)

terrà una conferenza sul tema

" Targeting PI3K in lymphoma"

La S.V. è invitata ad intervenire.

Il Direttore
Prof. Bruno Botta

The phosphatidylinositol 3-kinase (PI3K) pathway is among the most commonly affected pathways in human cancer. Lymphomas are no different as the PI3K pathway plays a crucial role down-stream not only to the B-cell receptor (BCR) but also to other receptors such as cytokine receptors. PI3Ks are composed of a catalytic subunit complexed with a regulatory subunit, that regulates the activity, localization and binding of the dimer. There are four different isoforms (p110α, p110β, p110δ, p110γ) of the catalytic subunit, which represent potential therapeutic targets to pharmacologically block PI3K signaling. The PI3Kδ inhibitor idelalisib (CAL-101, GS-1101) was the first PI3K inhibitor approved by the U.S. Food and Drug Administration (FDA) for patients with lymphoma. Copanlisib (BAY 80-6946) is a pan-class I phosphoinositide 3-kinase (PI3K) inhibitor with dominant activity towards PI3Kα and PI3Kδ. The drug has also shown clinical activity and is now FDA approved for relapsed follicular lymphoma patients after at least two systemic therapies. Similarly to other small molecules given as single agents, the number of complete remissions is limited. This observation, together with the extensive literature showing that targeting a single pathway is often not sufficient to eradicate tumor cells due the activation of additional pathways highlights the need of combinations to increase the cure rate. The seminar will present i) new data from a combination screening with copanlisib that has led to a new phase I study and ii) novel mechanisms of resistance to idelalisib.

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