ALMA MATER STUDIORUM
UNIVERSITÀ DI BOLOGNA

DIPARTIMENTO
DI FARMACIA
E BIOTECNOLOGIE

AVVISO DI SEMINARIO

Il giorno **28 ottobre 2025**
alle ore **15:00**

Prof. Glenn Marshall

Sydney Children's Hospital (SCH) and the Children's Cancer Institute Australia (CCIA), the University of New South Wales - Sydney
(ospite del Prof. G. Perini)

terrà un seminario in lingua inglese dal titolo:

MYCN as an embryonal oncogene: mechanisms of disease and therapeutic targeting

Area tematica: Cancer Biology

in presenza:
Aula ex-esercizi, via san Giacomo 12, Bologna

e in streaming:
https://teams.microsoft.com/l/meetup-join/19%3aN09c0NIyEssBnF7ObCyDOQwkgDWm1qdd9f7F2nJV9fw1%40thread.tacv2/1631519544944?context=%7b%22Tid%22%3a%22e99647dc-1b08-454a-bf8c-699181b389ab%22%2c%22Oid%22%3a%225a941351-ef41-4aa4-8771-fa50a6d62ca1%22%7d

L' evento è organizzato nell'ambito dei Corsi di Dottorato in Biologia Cellulare e Molecolare e Drug Discovery and Development

ABSTRACT

Human cancer results from a pathologic interaction between driver genes and environment. While adult malignancies arise after the slow accumulation of somatic mutations in mature postnatal cells over years, almost half of childhood cancer is initiated in embryonal cells. Pathologic postnatal persistence of some embryonal cells reflects defective apoptosis, cell fate decisions or arrested progenitor differentiation during embryogenesis. Neuroblastoma arises in sympathoadrenal neural crest progenitors, which are produced in utero in excess to requirements for normal organogenesis. The MYCN oncogene is essential for embryonal neural crest development and yet it is also a frequent driver in neuroblastoma and other embryonal child cancers. It is not known what determines the MYCN oncogenic or developmental roles, and whether MYCN collaborates with environmental factors in utero to initiate tumorigenesis. Here we show that MYCN and a high-fat environment can collaborate to reprogram mitochondrial metabolism at neuroblastoma initiation and progression.

The design of MYC or MYCN inhibitors has been difficult due to its intrinsically disordered protein structure. We recently identified a MYCN inhibitor, SE486-11, which, when combined with histone deacetylase (HDAC) inhibitors, reduced MYCN protein stability and tumour growth in MYCN-driven neuroblastoma. Here, we report the anticancer effects of SE486-11 analogues (UNSW-SCs), which demonstrated potent anticancer activity with preferential selectivity for MYC- and MYCN-driven childhood cancers: medulloblastoma and neuroblastoma.

BIOGRAPHICAL SKETCH

Glenn Marshall is a paediatric oncologist and clinician scientist in the Sydney Children's Hospital (SCH) and the Children's Cancer Institute Australia (CCIA) at the University of New South Wales Sydney. He is a past Director of the Kids Cancer Centre at SCH and Head of Translational Research at CCIA. He is currently Group Leader of the Embryonal Cancer Program. He has a longstanding focus on mechanisms of embryonal cancer in children, particularly the role of the MYCN and MYC oncogenes in neuroblastoma and medulloblastoma. His work led to the application of curaxins and polyamine synthesis inhibitors in MYCN-driven neuroblastoma, now in early phase trials, and more recently potent pyrimidobenzimidazoles as MYC degraders. His translational research has led to the discovery and application of molecular diagnostics for child cancer at high risk of relapse, such as childhood leukaemia. He co-created the Australian ZERO precision oncology program which uses molecular features and ex vivo drug responses to better match targeted drugs to the driver gene in high-risk child cancer.