ALMA MATER STUDIORUM
UNIVERSITÀ DI BOLOGNA

DIPARTIMENTO
DI FARMACIA
E BIOTECNOLOGIE

AVVISO DI SEMINARIO

Il giorno **13 febbraio 2026**
alle ore **14.30**

Prof. Simona Polo

Associate Professor of Pathology at the University of Milan
(ospite Prof.ssa Anna Maria Porcelli)

terrà un seminario in lingua inglese dal titolo:

β-catenin-RBM47 axis drives splicing reprogramming in colorectal cancer

Area tematica: Cancer Biology

in presenza:
Aula 1 SCIENZE FARMACEUTICHE, via Belmeloro 6, 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 del Corso di Dottorato in Biologia Cellulare e Molecolare/Colleghi e studenti sono cordialmente invitati

ABSTRACT

Alternative splicing (AS) is widely altered in cancer, yet the upstream signaling pathways that instruct these changes remain poorly understood. Aberrant activation of β-catenin signaling is a defining feature of colorectal cancer (CRC), where its nuclear accumulation is known to drive oncogenic transcriptional programs. Here, we uncover an unexpected function of β-catenin as a key regulator of AS in intestinal epithelial cells and CRC. Using an alternatively spliced exon cassette in myosin VI as a model, we show that β-catenin cellular localization controls a coordinated AS program. In polarized epithelial cells, E-cadherin–mediated sequestration of β-catenin at adherens junctions maintains an epithelial splicing pattern. By contrast, CRC-associated nuclear β-catenin accumulation drives tumor-associated splicing reprogramming. Mechanistically, β-catenin regulates AS through control of the RNA-binding protein RBM47. Perturbation experiments allow the identification of a β-catenin–RBM47 AS signature, evolutionary conserved. Together, our findings identify alternative splicing as a critical and previously unrecognized output of β-catenin signaling and reveal a β-catenin–RBM47–dependent AS program required for cancer metastasis.

BIOGRAPHICAL SKETCH

Simona Polo is Associate Professor of Pathology at the University of Milan and leads the Molecular Machines in Signaling Pathways laboratory at IFOM (The AIRC Institute of Molecular Oncology), which she established in 2005. Prof. Polo is an internationally recognized leader in the field of ubiquitin biology and signal transduction and EMBO member since 2016. Her laboratory was among the first to establish ubiquitin as an endocytic and signaling determinant, and to define the molecular logic by which E3 ligases confer substrate and chain-type specificity, thereby shaping the cellular ubiquitin landscape. Building on these foundations, Polo's group has expanded into studying how post-translational and co-transcriptional regulatory mechanisms intersect, focusing on alternative splicing (AS) as a means to diversify the proteome and fine-tune signaling outputs. Her recent work dissects how AS-driven protein isoforms influence cancer cell behavior, plasticity, and therapeutic resistance, with the long-term goal of identifying splicing-dependent vulnerabilities as novel therapeutic targets and biomarkers. Prof. Polo's research combines mechanistic precision with translational vision, positioning her laboratory at the interface of ubiquitin signaling, RNA processing, and tumor cell plasticity. https://pubmed.ncbi.nlm.nih.gov/?term=polo+simona&sort=date