European School of Molecular Medicine

2026 spring call PhD selections

AVAILABLE POSITIONS

Principal Investigator	**Matteo Cereda**
Institute of Affiliation	IFOM ETS – The AIRC Institute of Molecular Oncology, Milan; and Department of Oncology and Hemato-Oncology, University of Milan

PROJECT INFO	
Title of the proposed project:	Decoding the splicing code of KRAS G12C inhibitor resistance in lung adenocarcinoma
Short description of the project	KRAS G12C inhibitors and next-generation pan-KRAS drugs produce striking initial responses in non-small cell lung cancer, but durable benefit is cut short by adaptive, largely non-genetic resistance – a problem that becomes even more acute under the sequential and combination regimens now entering the clinic. Our previous work (Nokin et al., Nat Commun 2024) showed that KRAS G12C inhibition drives a coordinated transcriptional adaptation in lung adenocarcinoma cells, shifting them into a reversible drug-tolerant state rather than selecting for new mutations. In such drug-tolerant states, context-specific alternative splicing programmes reshape the proteome without altering the genome. This project will decode that code by combining AI-based splicing models with third-generation long-read sequencing, single-cell and Visium HD spatial transcriptomics, and matched proteomics across sequential drug exposures. Integrating these layers will pinpoint the RNA regulatory networks that sustain resistant cells in space and over time, providing new ground for RNA therapeutics. The project provides the PhD student access to a unique ecosystem of AI-driven splicing biology, multi-omics, and RNA therapeutics.
Main research area for the project	Computational RNA biology and AI-driven cancer transcriptomics
Second research area for the project	Molecular oncology of KRAS-driven lung cancer and RNA therapeutics
3 key words for the project	KRAS G12C resistance; alternative splicing; RNA therapy

LAB INFO	
Main topic/s of the lab	RNA biology and alternative splicing in cancer; AI-based decoding of the splicing code; non-genetic adaptive resistance to targeted therapy; RNA-based therapy; translational oncology
Short description of the lab activity	Our lab sits at the interface of computational RNA biology, AI-driven transcriptomics and translational oncology. We study how cancer cells survive targeted therapy without acquiring new mutations, and have shown that therapy-tolerant states are driven by rewired RNA regulatory networks.
Recent bibliography	Nokin M-J, et al. Therapy-induced shifts in KRAS G12C-mutant lung adenocarcinoma. Nat Commun 2024.PMID:39215000. Del Giudice M, et al. FOXA1 regulates alternative splicing in prostate cancer. Cell Reports 2022. PMID: 3617083.
Group composition	

	Current members include SEMM PhD students and pre-doctoral fellows working on computational splicing and experimental RNA biology, plus dedicated personnel on the KRAS G12C axis.
Institutional page link	https://www.ifom.eu/en/cancer-research/research-labs/research-lab-cereda.php
Lab website link	https://www.ceredalab.com
Social media links	X/Twitter: @ceredalab LinkedIn: linkedin.com/in/matteo-cereda