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HemaGRoove
JOINT HEMATOLOGY MEETINGS

Wednesday November 4th, 2026 – 14:00-15:00

Roger Monier Room – Gustave Roussy



Giulia BIANCON

Start-Up PI at Policlinico Hospital of Milan, Italy, and Assistant Professor Adjunct at Yale University, USA.
Invitée par Thomas Mercher

« Dissecting pathogenic RNA mechanisms in myeloid malignancies. »

Anyone wishing to meet with Dr. Giulia BIANCON during the day is invited to contact thomas.mercher@gustaveroussy.fr

Dr. Giulia Biancon obtained her PhD in Experimental Medicine and Medical Biotechnologies from the University of Milan, then trained as a postdoctoral fellow at Yale University, where she investigated RNA-related pathological mechanisms in myelodysplastic syndromes and acute myeloid leukemia (*Mol Cell*, 2022), developed a computational tool for annotating single-cell populations (*Nat Comm*, 2025) and characterized epitranscriptomic alterations in pediatric acute megakaryoblastic leukemia driven by RBM15-MKL1 (*Blood*, 2025). In 2024, she received an AIRC Start-Up Grant and established her laboratory in Milan. She is the co-Chair of the Milan RNA Salon and has received several awards, including the EvansMDS Young Investigator Award, the ASH Scholar Award and the Eclipse Award for Innovation in High Throughput Biology.

U2AF1 mutations are common in patients with myelodysplastic syndromes (MDS) and acute myeloid leukemia (AML) and are associated with poor outcome. The integration of high-resolution transcriptomics and imaging technologies has shown that U2AF1 mutations affect the biology of ribonucleoprotein (RNP) granules.
Specifically, U2AF1 mutations increase the availability of stress granule (SG) components, leading to enhanced stress adaptation and improved cell viability. Enhanced SGs and other RNP granule alterations may therefore represent a novel therapeutic vulnerability in MDS and AML.
We have now established a protocol to isolate and characterize SGs to identify the mechanisms underlying enhanced stress adaptation, and we are assessing the therapeutic potential of targeting SGs. Furthermore, we are investigating SG dependence in myeloid malignancies beyond the presence of U2AF1 mutations.