CRCT Centre de Recherches en Cancérologie de Toulouse ONCOPOLE

Inserm Institut national de la santé et de la recherche médicale

WORKSHOP

INSTITUT UNIVERSITAIRE DU CANCER DE TOULOUSE Oncopole

Innovations in immuno-oncology: from data to therapeutic insights

Thursday, October 16th, 2025

IUCT-O AMPHITHEATRE, TOULOUSE

INVITED SPEAKERS

JASMIN FISHER
UCL Cancer Institute, University College London, UK

SEBASTIEN BENZEKRY
COMPO, Inria and Cancer Research Center of Marseille
(Inserm, Aix-Marseille University, Paoli-Calmettes Institute)

NOEMIE LEBLAY
Arnie Charbonneau Cancer Institute, University of Calgary, Canada
Arthur J.E. Child Comprehensive Cancer Center

GAOUSSOU SANOU
IGH – Institut de Génétique Humaine
CNRS, Université de Montpellier

CALL FOR ABSTRACT SUBMISSION

ORAL PRESENTATIONS & POSTERS
(PhD students, post-docs)
DEADLINE: Friday, September 26th, 2025

Free registration required !

ARe Cancer, Ageing & Rejuvenation Graduate school

FONDATION TOULOUSE CANCER SANTE ONCOPOLE

B2S

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PROGRAM

IUCT-O AMPHITHEATRE, TOULOUSE

REGISTRATION 8:30 - 9:00

WORKSHOP OPENING 9:00 - 9:15

SESSION 1
MODELING AND DIGITAL TWINS IN CANCER

KEYNOTE 1: JASMIN FISHER **9:15 - 10:05**

UCL Cancer Institute, University College London, UK

Cancer Digital Twins

This presentation is possible thanks to the patronage of the Fondation Toulouse Cancer Santé

PHD AND POSTDOCTORAL PRESENTATIONS **10:05 - 10:45**

COFFEE BREAK AND POSTER SESSION **10:45 - 11:25**

KEYNOTE 2: SEBASTIEN BENZEKRY **11:25 - 12:15**

COMPO, Inria and Cancer Research Center of Marseille (Inserm, Aix-Marseille University, Paoli-Calmettes Institute)

Mechanistic learning to predict response and survival in immuno-oncology

PHD STUDENTS FLASH TALKS 12:15 - 12:35

LUNCH BREAK AND POSTER SESSION **12:35 - 14:15**

SESSION 2
ADAPTIVE IMMUNITY: MECHANISMS, RESISTANCE AND APPLICATIONS

KEYNOTE 3: NOEMIE LEBLAY **14:15 - 15:05**

Arnie Charbonneau Cancer Institute, University of Calgary, Canada
Arthur J.E. Child Comprehensive Cancer Center

Tolerogenic dendritic cells and immunosuppressive milieu cause resistance to daratumumab and IMiDs in multiple myeloma

PHD AND POSTDOCTORAL PRESENTATIONS **15:05 - 15:45**

COFFEE BREAK AND POSTER SESSION **15:45 - 16:25**

KEYNOTE 4: GAOUSSOU SANOU **16:25 - 17:05**

IGH – Institut de Génétique Humaine (CNRS, Université de Montpellier)

IMGT/mAb-KG : the IMGT-KG dedicated to monoclonal antibodies

CLOSING CEREMONY **17:05 - 17:10**

ABSTRACT - Keynote 1

Cancer Digital Twins

Dr. Jasmin Fisher

UCL Cancer Institute, University College London, UK

Cancer is a complex systemic disease driven by genetic and epigenetic aberrations that impact a multitude of signalling pathways operating in different cell types. The dynamic, evolving nature of the disease leads to tumour heterogeneity and an inevitable resistance to treatment, which poses considerable challenges for the design of therapeutic strategies to combat cancer. Digital twins for cancer tumours are emerging as a transformative tool in oncology to enable a more personalised and dynamic approach to cancer treatment. In this talk, I will showcase a growing library of mechanistic, data-driven computational models, focused on the signalling pathways within tumour cells and their microenvironment in various types of cancer (namely triple-negative breast cancer, non-small cell lung cancer, melanoma and glioblastoma). These computational models are mechanistically interpretable, enabling us to understand and anticipate emergent resistance mechanisms and to design patient-specific treatment strategies that counteract the forces of clonal selection driving treatment relapse, to improve outcomes for patients with hard-to-treat cancers.

ABSTRACT - Keynote 2

Mechanistic learning to predict response and survival in immuno-oncology

Dr. Sebastien Benzekry

COMPO, Inria and Cancer Research Center of Marseille (Inserm, Aix-Marseille University, Paoli-Calmettes Institute)

This presentation will explore methodological tools integrating machine learning, mechanistic modeling and nonlinear mixed-effects modeling (NLME for pharmacometrics) to analyze data pre- and on-treatment data of advanced cancer patients treated with immune-checkpoint inhibitors (ICI). The objective is to predict primary resistance to immunotherapy. The first dataset comes from the PIONeeR RHU project. It comprises multi-modal deep biomarkers derived both from tumor tissue (multiplex immunohistochemistry) and blood samples (immune-monitoring, vasculo-monitoring, hematology and biochemistry). Together, this dataset contains 439 patients and p = 433 pre-treatment biomarkers. I will focus on the of stable variable selection in high dimension with complex. We will see that classical approaches such as LASSO are not stable and will present novel methods to tackle this issue. Second, recent results from the SChISM (Size cfDNA Immunotherapies Signature Monitoring) clinical study will be presented. It investigates the utility of circulating cell-free DNA (cfDNA) fragment size profiles — collectively known as the fragmentome — as a non-invasive biomarker to predict early progression (EP) and progression-free survival (PFS) in patients with advanced carcinomas treated with ICIs. cfDNA profiles were obtained pre- and on-treatment across 128 pan-cancer patients, using a patented BIABooster-based technology that avoids DNA extraction. Pre-treatment fragmentome-derived features—such as the concentration of long fragments (≥1650 base pairs) — were statistically associated with treatment outcomes.

ABSTRACT - Keynote 2 (continued)

Mechanistic learning to predict response and survival in immuno-oncology

Dr. Sebastien Benzekry

COMPO, Inria and Cancer Research Center of Marseille (Inserm, Aix-Marseille University, Paoli-Calmettes Institute)

To gain mechanistic insight and improve predictive robustness, a biologically grounded mathematical model was developed to jointly describe the longitudinal kinetics of cfDNA concentration and tumor size under ICI. The model incorporates key biological assumptions: (1) cfDNA is released through apoptosis or necrosis from both immune and tumor cells, (2) cancer cells exhibit heterogeneous sensitivity to ICI, (3) DNA is cleared by liver and kidneys with different possible clearance dynamics, and (4) treatment induces changes in tumor burden and cfDNA shedding rates. The model is expressed as a system of ordinary differential equations and embedded in a nonlinear mixed-effects framework to account for inter-patient variability. This mechanistic approach successfully reproduced a wide range of cfDNA kinetics, including non-intuitive features like early concentration bumps, and revealed individual parameters that were significantly associated with clinical outcomes. Early model-derived parameters—estimated from truncated (first 1.5 months) data—demonstrated predictive value for long-term PFS.

ABSTRACT - Keynote 3

Tolerogenic dendritic cells and immunosuppressive milieu cause resistance to daratumumab and IMiDs in multiple myeloma

Dr. Noémie Leblay

Arnie Charbonneau Cancer Institute, University of Calgary, Canada
Arthur J.E. Child Comprehensive Cancer Center

The combination of the anti-CD38 monoclonal antibody daratumumab with immunomodulatory agents (IMiDs) has demonstrated high efficacy in multiple myeloma (MM). However, MM invariably relapses and the mechanisms mediating primary or acquired resistance to daratumumab-IMiDs therapies remain to be fully elucidated. We herein serially profiled (at baseline, cycle 3 and progression) the static and dynamic single-cells' transcriptomes of the bone marrow (BM) immune residents (CD138-) and plasma cells (CD138+) in relapsed MM patients (n=29) treated with daratumumab-IMiDs. At baseline, an activated immune microenvironment enriched with highly cytotoxic T and NK cells was noted in responders. In contrast, non-responders exhibited a highly inflammatory microenvironment dominated by indoleamine 2,3-dioxygenase IDO1 and tolerogenic classical dendritic cells (cDC1 and cDC2). At relapse, a comparable transcriptomic profile to baseline non-responders was also observed, with increased number of exhausted CD8+ T cells, loss of NK cytotoxic markers and enriched tolerogenic classical dendritic cells. Tumor-intrinsic factors such as CD38 downregulation and increased expression of CD47 were commonly noted at progression. Plasma and BM cell-cell interactions also identified SIRPG-CD47 and MIF-CD74:CXCR4 as mediators of the immunosuppressive response seen at progression. Therefore, we herein defined the static and dynamic BM immune profiles of daratumumab-IMiDs treated MM patients and identified novel druggable drivers of resistance.

ABSTRACT - Keynote 4

IMGT/mAb-KG : the IMGT-KG dedicated to monoclonal antibodies

Dr. Gaoussou Sanou

IGH – Institut de Génétique Humaine (CNRS, Université de Montpellier)

IMGT® is the international benchmark in immunoinformatics, integrating immunogenetic knowledge from gene to protein. It provides access to seven relational databases, 17 analysis tools and a wealth of web page resources. Given the complexity and connected nature of immunogenetic entities, IMGT created IMGT-KG, the first knowledge graph in the immunogenetics domain aiming to answer complex biological questions involving different databases, but also to discover new or implicit knowledge. Knowledge graphs (KG) are ontological models describing the entities of interest in a domain and the relationships between them. They enable the integration and federation of knowledge from various data sources. Lastly, to handle efficiently therapeutic monoclonal antibodies (mAbs), IMGT implemented IMGT/mAb-KG, the IMGT-KG dedicated to mAbs. MGT/mAb-KG integrates data from the IMGT/mAb-DB, a unique expertised resource on mAbs, with related data in IMGT-KG including genomics and proteomics information. IMGT/mAb-KG provides access to over 1,500 mAbs, approximately 500 targets, and over 500 clinical indications. It offers detailed insights into the mechanisms of action of mAbs, their construction, and their various products and associated studies. Linked to other resources such as Thera-SAbDab, PubMed, and HGNC, IMGT/mAb-KG is an essential resource for mAb development with a user-friendly web interface. IMGT/mAb-KG can be used to repurpose mAbs in new clinical application by embedding the mAbs, the clinical indications and the related knowledge with machine learning.

Organizing committee

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