CONGRESS BOOKLET

2nd Young Scientist Cancer Congress (YS2C)

New translational approaches in cancer therapy

10-11th October, 2024 - Montpellier, France

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Welcome!

The Young Scientist Cancer Congress (YS2C) was born from the meeting of 2 young French researchers and a young German clinician-scientist. They noticed the difficulties in starting an academic career after the doctorate in our two countries due to the lack of an academic network of young researchers. Consequently, they created the postdoc association at the Centre de Recherches en Cancérologie de Toulouse (CRCT) to build bridges between them.

To move things forward, they decided to organize the 1st YS2C of the GSO at a network of cancer research centers in South-Western France on October 5, 2023, at the Oncopole, Toulouse.

The primary aim was to encourage interdisciplinary collaboration among biologists, computational biologists, mathematicians, physicists, epidemiologists, clinicians, and others working in cancer research, ultimately striving to enhance patient care.

Postdoctoral researchers and clinicians from the GSO region were invited to join the organizing committee for this scientific and human adventure, to which they enthusiastically responded: “Yes!”

The first edition of YS2C was a remarkable success, drawing 240 participants from France and even Germany. Following discussions with the Germans, we decided to co-organize the 2nd edition, forging new connections with postdocs and clinicians from Charité University Medicine Berlin and the Max Delbrück Center for Molecular Medicine.

For this 2nd edition, we invited an outstanding panel of keynote speakers from across Europe and the USA. To stimulate meaningful exchanges, we planned various activities, including a roundtable discussion on “Building a Career in Cancer Research,” a "Physician Corner," working groups in cancer research, a meeting with an editor, and the Get-together/Icebreaker.

The organizers extend their gratitude to the University of Montpellier for hosting us and their financial support, Institut de Recherche en Cancérologie de Montpellier (IRCM) for their organizational support, and Prof. Il-Kang Na and Dr. Thomas Kammerthöns (Charité University Medicine Berlin) for their invaluable contributions from Germany.

We hope you enjoy these two enriching days!

The YS2C committee

2nd Young Scientist Cancer Congress

Welcome from the SIRIC directors.

Welcome to Montpellier, a territory of research and innovation, historically anchored in biology & health and home of the oldest still active medical school in Europe!

As Scientific Director and Director of the SItes de Recherche Intégrée sur le Cancer (SIRIC) Montpellier Cancer, a consortium that brings together biologists, clinicians and researchers in physics, mathematics, humanities and social sciences dedicated to cancer, whose ambition is to have significant impact on clinical practices for the benefit of patients, we would like to extend a warm welcome to all the scientists that will participate at the 2nd Young Scientist Cancer Congress.

Organized by postdocs & young clinician scientists, but open to the whole scientific community, the outstanding program of this 2nd edition will offer unique opportunities to meet the experts, to discuss new translational approaches in cancer therapy, to establish new collaborations and networks to accelerate innovation and transfer to the patient in order to fight against cancer.

Together, let's push the limits!

Pr David AZRIA, SIRIC director

Dr Nathalie BONNEFOY, SIRIC scientific director

SIRIC
MONTPELLIER CANCER
SIte de Recherche Intégrée sur le Cancer

Welcome from the Montpellier's Master Biologie Santé

I'm very grateful to the organizers of the 2nd Young Scientist Cancer Congress for choosing to hold this scientific meeting at the “Faculté des Sciences” of the University of Montpellier.

It is remarkable that young researchers have shown such dynamism in organizing a congress for the second time, with a program of such high quality. I hope they will be a source of inspiration for the students of Montpellier's Master Biologie Santé, and in particular of the Cancer Biology program, which they were keen to invite and involve. I hope that our Master's students will appreciate the dynamism of their younger elders and take their deep motivation for their research work as a model, so that they in turn will one day contribute to a better understanding of the mechanisms involved in tumor development, innovate in the development of therapies and share their knowledge with future generations of students and young researchers.

Stéphane Bodin, Associate Professor, University of Montpellier

Co-responsible for the Master in Biology-Health, Cancer Biology program.

The YS2C organizing committee warmly thanks the academic and industrial sponsors who agreed to take part in this second edition.

The committee would like to extend its deep gratitude to these institutions for their invaluable support in logistics and management.

ORGANIZING COMMITTEE

Benoît Aliaga	Centre de Recherches en Cancérologie de Toulouse (FR)
Steffen Fuchs	Charité-University Medicine, Berlin (DE)
Anna Salvioni	Centre de Recherches en Cancérologie de Toulouse (FR)
Marcin Domagala	Centre de Recherches en Cancérologie de Toulouse (FR)
Serena Stadler	Charité-University Medicine, Berlin (DE)
Chloé Bessière	Centre de Recherches en Cancérologie de Toulouse (FR)
Sara Ovejero	Institut de Génétique Humaine (FR)
Pierre-Francois Roux	Institut de Recherche en Cancérologie de Montpellier (FR)
Roxana Khazen	Centre de Recherches en Cancérologie de Toulouse (FR)
Charline Ogier	Centre de Recherches en Cancérologie de Toulouse (FR)
Katyana Seba	Centre de Recherche en Biologie cellulaire de Montpellier (FR)
Oriana Villafraz	Institut de Recherche en Cancérologie de Montpellier (FR)
Thomas LeFeivre	Bordeaux Institute of Oncology
Laura Grunewald	Charité-University Medicine, Berlin (DE)
Javier Florido	Institut de Recherche en Cancérologie de Montpellier (FR)

PRACTICAL INFORMATION

Prizes for best oral and poster presentations

Several competitions are organized to reward young researchers/clinicians (PhD students, post-docs, young clinicians):

- Best oral presentation sponsored by European Association of Cancer Research (EACR)
- Best oral and poster presentations in pediatric oncology, sponsored by the association Enfants Cancers Santé
- Best poster presentation in cancer bioinformatics, sponsored by the Société Française de Bioinformatique (SFBI)
- Best oral and poster presentation by YS2C committee

Social media

Please help us to increase the visibility of our conference! When posting your photos on social media (Twitter/X, LinkedIn), please use our hashtag **#YS2C.**

Follow our Twitter/X accounts: @umontpellier, @Crct_Postdoc, @crctoncopole, @IUCTOncopole, @IRCM-MTP, @IGH_MTP, @SiricMontpel and @CanceropoleGSO

Sponsors' stands

During the coffee breaks and lunches, you can interact with the sponsors' stands. Below, you will find a list of the sponsors stands.

Sponsors	Type
BD Biosciences	Biotech company
Enfants Cancers Santé	Pediatric patient association and research support
Parse Biosciences	Biotech company
ThermoFisher Scientific	Biotech company
Immudex	Biotech company
10x Genomics	Biotech company

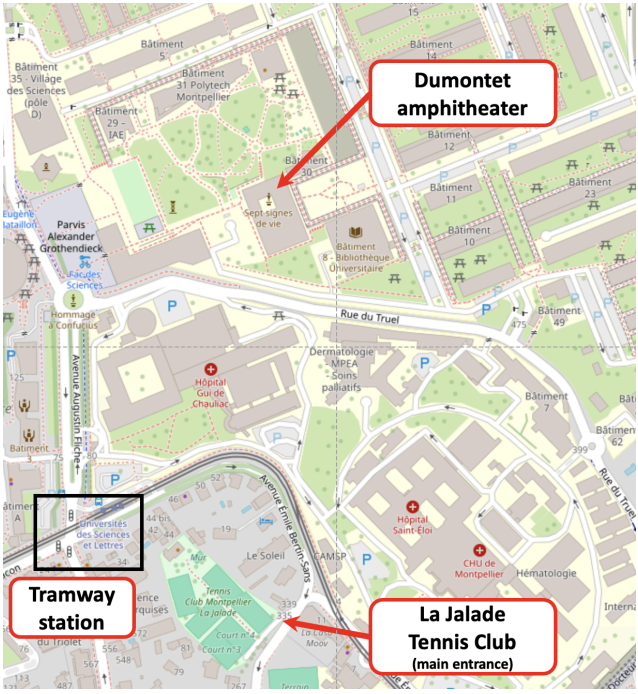
WiFi

Please use the *eduroam* network (free with an international university account).

Location of coffee breaks, lunch, and Icebreaker

The coffee breaks and lunches will be provided close to the Dumontet amphitheater (follow the indications).

Starting at 19.30 pm, we will have the *Icebreaker* at *La Jalade Tennis Club* located at 2 Rue de la Jalade, 34090 Montpellier.



Badges

Please return your badge at the end of the congress to the provided boxes.

Questions?
Don't hesitate to contact the congress office via ys2conference@gmail.com.

PROGRAM

October 10th

8:30 - 9:00	Registration
9:00 - 9:15	Welcome address
Session 1: Tumor immunology	
09:15-10:00	**Daniela Thommen**, Precision cancer immunotherapy Group, Netherlands Cancer Institute, Amsterdam, Netherland *Modelling and modulating T cell immunity in human cancers*
10:00-10:20	**Giulia Leonardi**, University Cancer Institute of Toulouse Oncopole, Toulouse, France *Differences in HPV-specific CD8 and CD4 T-cell responses in HPV-16 associated cervical cancer and head and neck cancer*
10:20-10:40	Coffee break + sponsors stands
10:40-11:00	**Lydia Dyck**, Max Delbrück Center for Molecular Medicine, Berlin, Germany *The secret life of antigen-specific T cells in cancer*
11:00-11:20	**Agata Mlynska**, National Cancer Institute, Vilnius, Lithuania *Exercise-induced extracellular vesicles delay tumor development by igniting inflammation in an immunologically cold triple negative breast cancer mouse model*
11:20-11:40	**Imene Gasmi**, Institut de Génomique Fonctionnelle, Montpellier, France *Tuft cell-dependent microenvironment remodeling promotes intestinal tumorigenesis*
11:40-12:00	Tech talk by **Michela Venturi**, Sales development manager, Immudex. *Characterization of antigen-specific immunity by flow cytometry and single-cell analysis with Dextramer® technology*
12:00-14:00	**Lunch buffet** + sponsors stands

	Meet the editor (starting at 13:00, an appointment is required) **Round table** *Building a Career in Cancer Research* (starting at 13: 00, Dumontet Amphitheater, no appointment)
Session 2: Tumor microenvironment	
14:00-14:45	**Mara Sherman**, Memorial Sloan Kettering Cancer Institute, New York, United States of America *Mechanisms and consequences of pancreatic cancer stromal evolution*
14:45-15:05	**Ilan Theurillat**, Berlin Institute for Medical Systems Biology, Berlin, Germany *A single-cell resolved spatiotemporal atlas of murine triple-negative breast cancer reveals myofibroblastic CAFs as drivers of aggressive tumor phenotypes*
15:05-15:25	**Ana Pestana**, Charité – Universitätsmedizin Berlin, Berlin, Germany *Preclinical models for rare tumors to advance precision oncology development*
15:25-15:45	Coffee break + sponsors stands
15:45-16:05	**Hala Shalhoub**, Cancer Research Center of Toulouse, Toulouse, France *Role of Vps34 in pancreatic cancer initiation*
16:05-16:25	Tech Talk by **Hafid Kora,** Technical sales manager, Parse Biosciences. *Expanding single Cell Capabilities with Evercode Technology*
16:25-17:10	**Inmaculada Martinez-Reyes,** Max Delbruck Center for Molecular Medicine, Berlin, Germany *Interventions in tumor and T cells metabolism to improve cancer immunotherapy*
17:10-18:40	**Poster session 1**
19:30	**Get-together/Icebreaker at La Jalade Tennis Club**

October 11th

Session 3: Cancer treatment and clinical trials	
09:00-9:45	**EMBO Young Investigator Lecture** **Nicholas McGranahan**, University College London, Cancer Institute, London, United Kingdom *Somatic evolution and the impact of treatment*
09:45-10:30	**Charles Herbaux**, CHU de Montpellier, Montpellier, France *BH3 profiling to characterize anti-apoptotic dependencies and identify new therapeutic approaches in T Prolymphocytic Leukemia*
10:30-10:50	Coffee break + sponsors stands
10:50-11:10	**Kuldeep LAHRY**, Max Delbrück Center for Molecular Medicine, Berlin, Germany *The secret life of antigen-specific T cells in cancer*
11:10-11:30	**Grégoire MANAUD**, BoRdeaux Institute of Oncology, Bordeaux, France *Deciphering the oncogenic properties of Fascin-1 in Hepatoblastoma*
11:30-11:50	**Bartolomeo BOSCO,** Charité-Universitätsmedizin Berlin and Max Delbrück Center for Molecular Medicine, Berlin, Germany *Unveiling interactions between senescent tumor cells and the host immune system - Implications for senolytic immunotherapy*
11:50-13:45	**Lunch buffet** + sponsors stands + company desk **Meet the editor** (starting at 13:00, an appointment is required) **The physicians' corner** (starting at 13: 00, Dumontet Amphitheater, an appointment is required) **Poster session 2**
13:45-14:05	Tech Talk by **Céline Jaimet,** Single-Cell MultiOmics Solutions Architect, BD Biosciences *Discover the Single Cell Multiomics with the Rhapsody*
Session 4: Cancer cells, tumor heterogeneity, and microenvironment	

14:05-14:25	**Thibault HOULES**, Institut de Génétique Moléculaire Montpellier, Montpellier, France *SUMOylation controls the AML surface proteome: role in immune response and signaling*
14:25-14:45	**Giulia Montuori**, Experimental and Clinical Research Center (ECRC) of the Max Delbrück Center (MDC) and Charité, Berlin, Germany *MYCN-driven oncogene dosage heterogeneity determines therapy response in neuroblastoma*
14:45-15:05	**Julien Boetto**, Institut de Génomique Fonctionnelle, Montpellier, France *Unmasking the early events of meningioma tumorigenesis*
15:05-15:25	Coffee break + sponsors stands
15:25-15:45	**Loredana Vecchione,** Charité - Universitätsmedizin Berlin, Berlin, Germany *Identification of molecular determinates for tailored treatments in BRAFV600E CRC by using preclinical models*
15:45-16:30	**Frances Balkwill,** Barts Cancer Institute, Queen Mary University of London, United Kingdom *Understanding and targeting the tumour microenvironment of ovarian cancer*
Session 5: The editor's perspective	
16:30-17:00	**Joanne Clancy**, Associate Editor, *Nature Communications* *Understanding the editorial process*
17:00	Closing remarks and Ceremony of poster & oral communication awards

THE PHYSICIAN CORNER

October 11th at 13.00
Chair: Steffen Fuchs

One of our aims is to bring young clinicians and researchers together. This will foster exchanges and potentially start future collaborations, all for the sake of translational research and ultimately for the patient's benefit. This is why we will have a dedicated space during the lunch break and poster session, "the physician's corner", where informal discussions between clinicians and researchers can happen in a relaxed atmosphere. Our panel of clinicians will be interdisciplinary having different specialties, such as medical oncology, neurosurgery, pediatric oncology, and early clinical trials. This will provide you with plenty of possibilities to discuss. Use this opportunity and just come around!

ROUND TABLE DISCUSSION

Building a Career in Cancer Research

October 10th at 13.00
Chairs: Benoît Aliaga and Laura Grunewald

The round table "*Building a Career in Cancer Research*" will start on the key advancements in cancer research, including spatial/single-cell technologies, immunotherapy, and the growing role of bioinformatics. Experts will discuss future directions in the field and practical career advice for engineers, lab technicians, PhD students, postdocs, and clinicians. A major theme is navigating the transition from postdoc to principal investigator (PI) and how clinician-scientists can balance clinical work, teaching, and research. The event will offer valuable insights for researchers at all stages of their careers.

THE WORK GROUPS MEETING

October 10th at 18.40
Chair: Benoît Aliaga

The organization of YS2C 2024 is a collaboration between researchers and clinicians working in France and Germany. YS2C aims to close the gap between PhDs and PIs and create an event for young scientists and clinicians that did not

exist in our countries so far. One of our aims is to bring young clinicians and researchers together, initiate potential new collaborations, and foster translational research that leads to fast-applicable benefits for patients. On **October 10th at 18:40**, we organize a meeting to present our project of work groups to develop our skills and build stronger interactions between our countries.

MEET THE EDITOR

October 10th and 11th at 13.00
Chair: Anna Salvioni

Meeting a scientific editor at YS2C is essential for several reasons. First, a scientific editor plays a key role in research publication. They can provide valuable advice on article submission, quality requirements, and editorial trends in cancer research. This helps young researchers refine their manuscripts to increase their chances of acceptance in high-impact journals. Additionally, editors are often aware of new directions and priorities in scientific research. They can help you understand which topics are currently prioritized and guide you toward innovative themes or methodological approaches. This type of exchange can influence future research directions and open doors to strategic collaborations. Finally, meeting an editor at a conference helps build a relationship of trust, which is crucial for the publication process. Young researchers can directly discuss specific aspects of their work and receive constructive feedback, not only on the structure of their articles but also on their scientific relevance and potential impact. In summary, establishing a dialogue with a scientific editor at a cancer conference can improve the visibility of research, guide authors toward a better publication strategy, and enhance opportunities for disseminating their work to a wider audience.

KEYNOTES SPEAKERS

The YS2C organizing committee warmly thanks the keynote speakers who agreed to take part in this second edition.

Prof. Frances Balkwill, PhD

Barts Cancer Institute, Queen Mary University of London, United Kingdom

Prof. Frances Balkwill is a British scientist, renowned for her research on the interaction between tumors and their microenvironment, particularly in the context of cancer growth and spread. Her work has been instrumental in deciphering the connection between inflammation and cancer. She is a prominent figure in translating this understanding into innovative treatments for ovarian cancer. Prof. Balkwill has also played a pivotal role in raising public awareness of scientific advancements.

Prof. Balkwill was trained at Barts Medical Oncology Unit and the ICRF Lincoln's Inn Fields. During her postdoctoral research, she focused on studying interferons and their role in cancer therapy. Her subsequent work delved into the intricate roles of cytokines, both in cancer promotion and inhibition. This research paved the way for broader studies on the cellular and mediator components of the complex and dynamic tumor microenvironment. Currently, Prof. Balkwill's research is centered on ovarian cancer. Recently, her lab published a comprehensive profile of the human ovarian cancer microenvironment. They have also developed new mouse models and human multicellular tissue culture models, which they are using to investigate biological therapies that could prevent relapse and improve patient survival in ovarian cancer.

In addition to her scientific contributions, Prof. Balkwill is actively involved in public engagement with biomedical science. She has authored several books for children on cell and molecular biology. She is the Director of the Centre of the Cell, an informal biomedical science center for children, educational website, and outreach project in East London, which has engaged over 180,000 participants since its inception in September 2009.

Prof. Balkwill serves as a non-parliamentary board member of the Parliamentary Office of Science and Technology (POST) and is a Trustee of Bloodwise. She was awarded an OBE in the 2008 Queen's Birthday Honours list. In 2015, the University of Bristol conferred upon her a Doctor of Science honoris causa, and in 2017, she received the Cancer Research UK Inspiring Leadership in Research Engagement Prize.

Dr. Mara Sherman, PhD

Memorial Sloan Kettering Cancer Center, New York City, United States of America

Dr. Mara Sherman is an associate member of the Memorial Sloan Kettering Cancer Center at New York City. The Mara Sherman lab aims to understand the heterocellular interactions among pancreatic cancer cells and their surrounding microenvironment, and to target these networks for therapeutic benefit.

She did her PhD in the laboratory of Michael Teitell at the University of California, Los Angeles, working on the mechanisms regulating B cell lymphomagenesis. Then she moved to the laboratory of Danny Manor at Cornell University, Ithaca, working on mechanisms of cellular transformation by the Dbl oncogene.

She has prestigious recognition in the field and her laboratory is supported by grants like R01 and P01.

Dr. Nicholas McGranahan, PhD (EMBO Young Investigator Lecture)

Cancer Genome Evolution Research Group, CRUK-UCL Lung Cancer Centre of Excellence, UCL Cancer Institute, London, United Kingdom

Dr. Nicholas McGranahan is an computational geneticist based at the CRUK-UCL Lung Cancer Centre of Excellence, located in the UCL Cancer Institute. He completed his Ph.D. at the University College London under the advisory of Dr. Charles Swanton and Dr. Nicholas Luscombe. He pursued his research as a postdoctoral fellow at The Francis Crick Institute in the lab of Dr. Charles Swanton. Currently, he is leading a research group that develops computational methods to explore the cancer genome and antitumor immunity within an evolutionary framework.

Using state-of-the-art bioinformatics and evolutionary methods, his team aims at understanding how tumors are developing and how they might be treated. Dr. McGranahan's work is centered around exploring tumorigenesis as an evolutionary process. He has developed various tools to assist researchers in understanding the effect of genetic mutations accumulated during tumor development, but also use this knowledge to predict the tumor's trajectory. This comprehensive approach might help in designing more effective cancer treatments in the future. Dr. McGranahan's exceptional work in the field of cancer evolution has granted him several prestigious awards, including: Blavatnik Awards

for Young Scientists, Young Investigator EMBO Award and Sir Henry Dale Fellowship, Wellcome Trust.

Prof. Charles Herbaux, MD, PhD

CHU de Montpellier, Institut de Génétique Humaine, Université de Montpellier, Montpellier, France

Prof. Charles Herbaux is an MD PhD whose work is primarily focused on lymphoproliferative diseases. He obtained his MD in the University and CHRU of Lille in 2014. He did his PhD in 2017 working on pathophysiology of T prolymphocytic leukemia (T-PLL). In 2018, he joined the laboratory of Dr. MS Davids at Dana-Farber Cancer Institute in Boston as a postdoctoral fellow. While there, he focused on the development of a BH3 profiling method to assess the tumor cell's dependency to anti-apoptotic proteins from the Bcl-2 family. Prof. Herbaux came back to France in 2020, where he now carries out his care and clinical research activities in the department of Clinical Hematology – CHU de Montpellier. He is also leading translational research projects within Prof. Jérôme Moreaux's team at Institute of Human Genetics. He is studying how cellular signaling cascades and cell death are affected by BCR and TCR pathway inhibition, and how these results correlate with clinical outcomes in trials. Ultimately, the aims of his clinical and research efforts are to help develop novel therapies that improve outcomes for patients afflicted with lymphomas and lymphoid leukemias.

Dr. Daniela Thommen, MD, Ph.D

Precision Cancer Immunotherapy Group, Netherlands Cancer Institute in Amsterdam, Amsterdam, Netherlands

Dr. Daniela Thommen is a group leader at the Netherlands Cancer Institute in Amsterdam. Her group focuses on understanding the determinants of response to checkpoint inhibitors, taking advantage of a unique ex-vivo tumor model, the patient-derived tumor fragment platform. Through transcriptomic, spatial and multiparametric flow cytometry analysis, her lab aims to identify predictive biomarkers of response to immune-checkpoint inhibitors. She has made major contributions to the field, characterizing thoroughly the lymphoid cell's compartment in solid tumors, especially lung cancer.

Supported by a MD-PhD fellowship, she pursued both medical training and completed a PhD in T-cell immunology at the University of Basel, Switzerland, in 2010 under the supervision of Prof. Biedermann. She continued her dual activity as a clinician scientist, through training in internal medicine and medical oncology

and, in parallel, work as a research fellow in the lab of Prof. Zippelius at the Department Biomedicine in Basel.

In 2016, she joined the lab of Prof. Ton Schumacher at the Netherlands Cancer Institute with a postdoctoral fellowship from the Swiss National Science Foundation. During this time, she continued to advance understanding of the heterogeneity in intratumoral immune activity, with a focus on T cell dysfunction, which allowed her to establish her own independent research group at the Netherlands Cancer Institute in April 2020.

She is the recipient of several prestigious grants and awards, such as: the Swiss Pfizer Research Prize in oncology (2019), the Young Investigator Grant/Bas mulder award (2018) from the Dutch Cancer Society and a Melanoma Research Alliance Team Science Award, together with C. Blank and D. Peeper.

Dr. Inmaculada Martínez Reyes, Ph.D

Junior Group Leader, Max Delbrück Center for Molecular Medicine, Berlin, Germany

Dr. Inmaculada Martínez Reyes is an expert in the field of cancer metabolism, focusing on the role of mitochondria in immune suppression and exhaustion within the tumor microenvironment. She earned her Ph.D. in 2012 at University Autonoma in Madrid, Spain, and subsequently conducted postdoctoral research in Spain with Dr Marcos, in USA with Dr Chandel and finally with Prof. Blankenstein in Germany. She is an author of numerous publications in prestigious journals, including: Nature, Cell Metabolism, Molecular Cell, Nature Communications and Nature Chemical Biology. Since September 2024, she leads her own group at the Max Delbrück Center for Molecular Medicine (Berlin, Germany).

Dr. Joanne Clancy, Ph.D

Associate Editor in Nature Communications, Springer Nature, United Kingdom

Dr. Joanne Clancy joined Nature Communications in March 2022 as a Locum Associate Editor. Previous to this Joanne received a MSc(Res) in Translational Oncology at the University of Sheffield and went on to study for a PhD at University College London. Her doctoral research evaluated the effects of combining radiotherapy and a novel inhibitor of DNA damage repair on tumour

immunogenicity. Joanne is based in the London office and handles mainly cancer therapy related submissions.

ABSTRACTS OF ORAL PRESENTATIONS

Session 1: Tumor immunology

Differences in HPV-specific CD8 and CD4 T-cell responses in HPV-16 associated cervical cancer and head and neck cancer

Giulia Costanza Leonardi[1,2], Victor Sarradin[1,2], Noémie Thébault[2], Clara-Maria Scarlata[2], Marie Michelas[2], Virginie Feliu[2], Françoise Lauzéral-Vizcaïno[2], Nicolas Congy[1], Jean-Pierre Delord[1,2], Alejandra Martinez[1,3], Maha Ayyoub[2]

[1] Department of Medical Oncology, Institute Universitaire du Cancer de Toulouse, Toulouse, France
[2] T2i, Antitumor immunity and immunotherapy, Cancer Research Center Toulouse, Toulouse, France
[3] Department of Surgery, Institute Universitaire du Cancer de Toulouse, Toulouse, France

Background: Human Papillomavirus (HPV)-16 is responsible for 50% of cervical cancers (CC) and 90% of HPV+ head and neck cancers (HNC). HPV+ tumors provide the opportunity to detect and characterize tumor-reactive CD8/CD4 T-cells adopting a set of virus-derived tumor-antigens. The characterization of HPV-specific T-cell responses is of paramount importance to understand the role of the immune system and tailor immunotherapy strategies.

Aims: The aim of this study was to compare HPV16-specific CD8 and CD4 T-cell responses in CC and HNC and evaluate the HPV16-specific CD8 T-cell exhaustion status.

Methods: We collected tumor biopsies, peripheral blood mononuclear cells, and clinical data from non-metastatic HPV-16+ CC and HNC. Potential CD8 T-cell epitopes derived from the HPV proteins and presented by a reference set of 27 human leukocyte antigens were predicted using the Immune Epitope Database. CD4 T-cell responses were assessed using pools of 20 amino acid long overlapping peptides covering the full sequence of E2, E5, E6 or E7 proteins. Fluorescent HLA class I/peptide/tetramers and flow cytometry analysis were used to characterize Ag-specific T cells.

Results: 13 HPV-16+ CC and 22 HPV-16+ HNC were included in this study. We were able to detect HPV-16 specific CD8 and CD4 responses in 9 out of 13 CC and 18 out of 22 HNC. No differences in the amplitude of the HPV-16 specific response was

observed in CC versus HNC. In CC, CD8 T-cell responses were observed against E2, E5, E6 and E7 oncoproteins while no responses against E7 where observed in HNC. Using fluorescent HLA class I/peptide multimers and flow cytometry analysis, we observed that CD8 T-cells specific for the same epitope exhibited different exhaustion phenotypes in CC versus HNC.

Conclusion: Despite no quantitative differences in HPV16-specific CD8 and CD4 T-cell response in CC versus HNC, the exhaustion profile of HPV16-specific CD8 T-cells was different according to the primary tumor type.

The secret life of antigen-specific T cells in cancer

Lydia DYCK[1], Kathleen ANDERS[1], Miha MILEK[2], Elmehdi BELBARAKA[1], Maria Teresa NORCIA[1], Dieter BEULE[1,2], Inmaculada MARTINEZ-REYES[1], Thomas BLANKENSTEIN[1]

[1] Max Delbrück Center for Molecular Medicine, Berlin, Germany
[2] Core Unit Bioinformatics Berlin Institute of Health at the Charite, Berlin, Germany

Chronic antigen exposure in solid tumors leads to T cell dysfunction and cancer progression. Cancer immunotherapies have demonstrated the induction of functional T cell responses in specific tumor models and human cancer types. However, the efficacy observed in mouse models with transplanted tumors often does not translate to the same degree in humans. To address this disparity, we have developed a transgenic, autochthonous tumor model called TTC, in which the oncogene and T cell antigen SV40 large T antigen (Tag) is induced by doxycycline (dox) from birth in cells with (a history of) tyrosinase expression. This model allows us to study the interplay between tumor-reactive T cells and tumor development over time. After 4 months on dox, TTC mice failed to mount an immune response against transplanted Tag-expressing cancer cells, indicating the onset of T cell dysfunction. Between 6-12 months on dox, TTC mice developed singular tumors. Transfer of dysfunctional TTC T cells into Rag-ko mice, followed by immunization, induced polyclonal expansion of Tag-reactive T cells. However, these cells failed to reject Tag-expressing tumor cells in vivo. Single cell TCR sequencing revealed a notable proportion of Tag-specific T cells in tumor-bearing TTC donor mice that were undetectable by Tag-directed tetramer staining. This suggests that tumor-specific dysfunctional T cells downregulate their TCRs.

Consistent with this, the expression of several genes associated with the TCR complex was significantly downregulated in tumor-reactive CD8 T cells in TTC mice. Tumor-reactive CD8 T cells in autochthonous cancer-bearing mice predominantly exhibited an exhausted or progenitor-exhausted state, a phenotype they maintained after transfer and immunization. These findings indicate that, in addition to upregulation of inhibitory receptors, tumor-directed T cells downregulate TCR expression, which hampers T cell activation and potentially undermines the efficacy of immunotherapy.

Exercise-induced extracellular vesicles delay tumor development by igniting inflammation in an immunologically cold triple negative breast cancer

Agata MLYNSKA[1], Neringa DOBROVOLSKIENE[1], Karolina SUVEIZDE[1], Gabija LUKASEVICIUTE[1], Krizia SAGINI[2], Beatriz MARTIN GRACIA[2], Silvana ROMERO[2], Alizia LLORENTE[2], Aija LINE[3], Austeja BUTKUTE[1], Beatrice GUDAITE[1], Tomas VENCKUNAS[4], Vita PASUKONIENE[1]

1 National Cancer Institute, Vilnius, Lithuania
2 Oslo University Hospital, Oslo, Norway
3 Latvian Biomedical Research and Study Centre, Riga, Latvia
4 Lithuanian Sports University, Kaunas, Lithuania

Preclinical studies demonstrate that physical activity reduces tumor incidence and growth. Rapid release o extracellular vesicles (EVs) during exercise suggests their potential role as mediators of exercise-induced systemic effects and physiological adaptation. This study investigates the impact of exercise-induced plasma EVs, potential exercise mimetics, on tumor growth and the immune microenvironment in two murine models of triple-negative breast cancer (TNBC) - EO771 and 4T1. Size exclusion chromatography isolated exercise-induced EVs from plasma of healthy female mice (BALB/c and C56BL/6, n=30 per strain) that underwent ten 30-minute treadmill running sessions, classified as moderate exercise intensity. Nanoparticle tracking analysis, Western blot and electron microscopy confirmed the presence of EVs in the samples. Tumor-bearing mice (n=72 per strain) were administered with exercise-induced EVs preventively, therapeutically, or in combination. Local and systemic immune responses were assessed using flow cytometry, multiplex cytokine assay, ELISA, and qPCR. Treatment with

exercise-induced EVs reduced tumor growth by 19-57%. Notable differences in tumor-infiltrating lymphoid and myeloid cell subpopulations indicated immunomodulatory effects of exercise-induced EVs, particularly in the 4T1 model. Exercise-induced EVs, when used as preventive conditioning in EO771 or in preventive+therapeutic setting in 4T1, led to a substantial increase in intratumoral CD8+ T lymphocytes and reduction of PD-L1 expression on tumor cells. Our study demonstrates that exercise-induced EV treatment triggers a pro-inflammatory antitumor immune response, converting 'cold' TNBC tumors to 'hot', which are associated with better outcomes. The use of EVs as exercise mimetics could be a promising strategy for enhancing antitumor immune responses and sensitizing tumors to immunotherapy.

Conclusion: This rare presentation highlights the phenotypic heterogeneity of metastases after various lines of chemotherapy. With more than half of lesions with no or weak SSTR-2 expression, 177Lu-DOTATATE internal therapy was ruled out. Further investigations are needed to better comprehend this tumor mismatch and its prognostic implications. Screening of molecular targets using complementary nuclear imaging techniques is an interesting option to guide treatment of refractory neuroblastomas.

Tuft cell-dependent microenvironment remodeling promotes intestinal tumorigenesis

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Colorectal cancer (CRC) remains a major public health issue, ranking among the most prevalent malignancies and the second leading cause of cancer-related death. Initial research focused on genetic alterations of the APC gene (Adenomatous Polyposis Coli), whose loss of function was identified as the initiating event in most sporadic CRC cases. Recently, there has been growing interest in the impact of the tumor microenvironment on tumor initiation and progression. One critical component of the tumor microenvironment is the

immune system, now recognized as a major player in both preventing and promoting cancer development. Understanding the mechanisms underlying the establishment of a pro-tumoral immune microenvironment could lead to the development of new immune-based therapies and the discovery of biomarkers. We have identified that tuft cells, a rare intestinal epithelium cell type, are immunomodulatory cells and key players in tumor initiation. Our data indicate that in a mouse model of intestinal tumorigenesis (Apc $^{\Delta 14/+}$), tumor initiation is significantly decreased in the context of tuft cell deficiency (Pou2f3ko). Interestingly, this lower rate of tumor initiation was associated with a reduced infiltration of regulatory T cells (Treg) in non-tumoral mucosa, where only a single Apc allele was lost. The functional role of Treg infiltration was then assessed using a genetic mouse model (DEREG) that allowed their specific depletion during intestinal tumorigenesis. Our results showed a drastic reduction in tumor initiation following Treg depletion, confirming the critical role of tuft cell-dependent Treg infiltration at the pre-initiation stage. Overall, our findings suggest that in the context of Apc heterozygosity, tuft cells modulate the rate of tumor initiation, likely in part through the recruitment of immunosuppressive cells such as Tregs. This study highlights that tuft cells could be a potential target for CRC prevention and treatment.

Session 2: Tumor microenvironment

A single-cell resolved spatiotemporal atlas of murine triple-negative breast cancer reveals myofibroblastic CAFs as drivers of aggressive tumor phenotypes

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Triple-negative breast cancer (TNBC) presents an aggressive phenotype and high metastatic rate, posing significant challenges for targeted therapy and resulting in poor patient prognosis. To adress these challenges, novel models are needed to faithfully recapitulate the complexity of human TNBC within an autochthonous tumor micro-environment (TME). In this study, we present a new murine model of TNBC using breast-specific expression of patient-relevant gain-of-function mutations in TP53, PIK3CA combined with WNT pathway overactivation.

Our model demonstrates rapid, reproducible tumor development within weeks, mirroring human tumor progression, including metastasis. Using single-nuclei and high-resolution spatial-transcriptomics, we constructed a spatiotemporal atlas of tumor and micro-environment remodeling at cellular resolution. We characterize the reprogramming of epithelial cells into heterogeneous basal-like tumor populations within specific stromal niches. Notably, in the TME, we describe the transition of fibroblasts first towards inflammatory cancer-associated fibroblasts (iCAFs) then progressing into myofibroblastic CAFs (myCAFs) in the direct vicinity of advanced tumors. Leveraging cell-cell communication tools, we predict the role of the NCAM1-FGFR axis between myCAFs and tumor cells in promoting pre-metastatic states. Furthermore, we show using patient xenograft models, that human tumors can induce NCAM1-positive CAFs. Finally, using syngeneic transplants in immunocompetent mice and an ex vivo organoid co-culture system, we demonstrate the strong tumor-promoting capabilities of myCAFs, driving transformed cells towards an invasive phenotype.

In summary, our study provides insights into tumor and micro-environment remodeling throughout TNBC tumorigenesis, identifying CAFs that promote cancer growth. We anticipate these findings will pave the way for targeted therapeutic strategies aimed at disrupting CAF to tumor cells communication pathways.

Preclinical models for rare tumors to advance precision oncology

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Rare tumors (RT) represent over 22% of all newly diagnosed cancers in Europe. Due to the low incidence of the individual tumor types, clinical trials for specific subtypes are difficult to perform, and there is a significant lack of preclinical models available to study novel treatment approaches. Available targeted and immunotherapy are effective in only a subset of patients, and data are lacking specifically for RT types, creating an urgent need for reliable preclinical models to repurpose existing and test novel therapies. Patient-derived organoids (PDO) are a powerful tool to mimic the tumor cells' heterogeneity but lack the TME interactions, making them inadequate for preclinical evaluation of immunotherapy. There is an intrinsic need to characterize the crosstalk between

tumor cells and the tumor microenvironment (TME) to decipher mechanisms of therapy resistance, and to define biomarkers of response. Therefore, we modified the classical PDO (cPDO) protocol to maintain the immune cell population in culture (mPDO). We hypothesized that maintenance of the tumor infiltrating immune cell (TIIC) population would be superior to traditional co-culture protocols as it allows for more reliable prediction of tumor immune cell interactions. Nine RTs were processed with the mPDO protocol with two distinct observations: 1) organoids including native TIICs having a short life span without further TIIC expansion (urachal carcinoma and ameloblastoma), and 2) organoids, which were quickly overgrown by the TIIC population with continued expansion (medullary renal carcinoma). The different generated models are currently in analysis to study their phenotype using cyclic multiplex immunofluorescence, to understand the TME crosstalk and details of the antitumoral activity of TIIC population that is produced when cultured alongside the organoids using the mPDO protocol.

Role of Vps34 in pancreatic cancer initiation

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PI3K/Akt/mTORC1 pathway is among the major oncogenic signaling pathways that are activated in panvreatic pancreatic ductal adenocarcinoma (PDAC). Whether class III PI3K also tightly control mTORC1 in physiological settings remains controversial; besides, the role of class III PI3-kinase remained underexplored both in PDAC and in pancreatitis,a potential risk factor for PDAC development. In our study, we developed Vps34+/+, Vps34KI/KI(=V34), KC and KCVps34KI/KI(=KCV34) mice models to understand the role of class III of PI3-kinase in pancreatic diseases. Vps34 inactivation in exocrine pancreas resulted in fibrogenesis and lipid accumulation. In vivo and ex vivo, acinar cells had heterogeneous levels of autophagy ; Vps34 inactivation showed blocked flux of autophagy and differential expression levels of autophagy-regulating proteins compared to WT acini. Autolysosome surface decreased in acinar cells with Vps34 inactivation. Surprisingly, despite a blockage of autophagy, those cells appeared resistant to mutant Kras oncogenicity as full inactivation of Vps34 in KC mice led to absence of precancer lesions in aged mice. ScRNA-seq showed that Vps34 inactivation prompted a selective loss of a subset of acinar cells with high mitochondrial and

autophagy-related genes. Moreover, acinar cells with Vps34 inactivation showed enrichment in the expression of regenerating islet-derived 3 beta (Reg3b) gene. Acinar cells with Vps34 inactivation showed difference in Regs protein levels both at the basal levels and in response to autophagy modulators compared to WT acini. Finally, acinar cells with Vps34 inactivation showed also decreased levels of p-Akt levels, known to be necessary to pancreatic plasticity. Vps34 full inactivation may protect from initiation of precancer lesions in the context of inflammation by bypassing epithelial plasticity. These finding may be a key to understand pancreatic cancer initiation.

Session 3: Cancer treatment and clinical trials

Pioneering Cancer Therapy Through Microbiota-derived Metabolites and Synthetic Analogues

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Microbial metabolites interact with eukaryotic hosts, influencing cell physiology through various mechanisms. A notable molecular pathway involves the incorporation of the gut microbial metabolite queuine into the wobble position of host tRNAs (as a queuosine nucleotide) by the host enzyme eTGT, thereby regulating host cell translation. Microbes also produce pre-queuosine 1 (preQ1),

an intermediate in the complex queuosine biosynthesis pathway. We discovered that both preQ1 and queuine are detectable in the plasma and tissues of mice and are incorporated into host tRNAs both in vitro and in vivo. However, the impact of preQ1 on host cell biology is markedly different from that of queuine. Our study demonstrates that preQ1 incorporation into tRNA disrupts protein synthesis, significantly alters gene expression, and inhibits the proliferation of human and mouse cancer cell lines, while having no effect on non-cancerous fibroblast cell lines. Additionally, preQ1 treatment reduces tumor growth in a xenografted cancer mouse model without affecting healthy tissues. The effect of preQ1 is limited by its competition with queuine for eTGT-mediated tRNA incorporation and by a "bell-shaped" efficacy curve at higher concentrations. To address these limitations, we designed a panel of synthetic queuine analogues and identified one, STL-105, which exhibits a similar inhibitory effect on cancer cell lines but with linearly increasing competitive efficiency. This study highlights the role of the microbiome in regulating host gene expression and presents natural metabolites from our microbiota as a potential source of anti-cancer therapeutic molecules.

Deciphering the oncogenic properties of Fascin-1 in Hepatoblastoma

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Chronic antigen exposure in solid tumors leads to T cell dysfunction and cancer progression. Cancer immunotherapies have demonstrated the induction of functional T cell responses in specific tumor models and human cancer types. However, the efficacy observed in mouse models with transplanted tumors often does not translate to the same degree in humans. To address this disparity, we have developed a transgenic, autochthonous tumor model called TTC, in which the oncogene and T cell antigen SV40 large T antigen (Tag) is induced by doxycycline (dox) from birth in cells with (a history of) tyrosinase expression. This model allows us to study the interplay between tumor-reactive T cells and tumor development over time. After 4 months on dox, TTC mice failed to mount an immune response against transplanted Tag-expressing cancer cells, indicating the onset of T cell dysfunction. Between 6-12 months on dox, TTC mice developed singular tumors. Transfer of dysfunctional TTC T cells into Rag-ko mice, followed by immunization, induced polyclonal expansion of Tag-reactive T cells. However,

these cells failed to reject Tag-expressing tumor cells in vivo. Single cell TCR sequencing revealed a notable proportion of Tag-specific T cells in tumor-bearing TTC donor mice that were undetectable by Tag-directed tetramer staining. This suggests that tumor-specific dysfunctional T cells downregulate their TCRs. Consistent with this, the expression of several genes associated with the TCR complex was significantly downregulated in tumor-reactive CD8 T cells in TTC mice. Tumor-reactive CD8 T cells in autochthonous cancer-bearing mice predominantly exhibited an exhausted or progenitor-exhausted state, a phenotype they maintained after transfer and immunization. These findings indicate that, in addition to upregulation of inhibitory receptors, tumor-directed T cells downregulate TCR expression, which hampers T cell activation and potentially undermines the efficacy of immunotherapy.

Unveiling interactions between senescent tumor cells and the host immune system - Implications for senolytic immunotherapy

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Therapy-induced senescence (TIS) stops tumor growth and improves treatment outcome in many preclinical cancer models but it also alters the tumor biology and remodels the tumor environment. In this way TIS contributes to treatment resistance and tumor relapse. Several publications have described the close and intricate relationship between senescent tumors and the host immune system, but the molecular mechanism of their interaction remains unclear and its impact on treatment efficacy contingent upon cancer type. Consequently, understanding the dynamics between senescent tumor cells and the host immune system emerges as a priority target to improve cancer therapy, for example with new senolytic immunotherapies. RNA and proteome analyses as well as immunophenotyping by flow cytometry showed that both mouse lymphomas and human cancer cell lines exhibited higher expression of gene sets and surface markers associated with immune system activation upon TIS. Investigation of tumor-host immune system interactions, performed by immune mass cytometry and spatial multiplex immunofluorescence imaging MACSima, revealed that TIS promotes the infiltration of both CD4 and CD8 T cells into senescent tumor sites and their direct interaction with senescent cells. Immunophenotyping analyses of these T cells identified direct interaction with senescent cells. Immunophenotyping analyses of these T cells identified overexpression of Fas ligand as an actionable moiety that induces apoptosis in Fas receptor positive TIS cells. Finally, we employed genetic and pharmaceutical tools to modulate the binding of FasL to FasR, to show the crucial impact of this interaction for senolytic therapies and treatment outcome. This study elucidates the impact of TIS on

tumor cell immunogenicity demonstrating enhanced immune system activation and increased susceptibility to T-cell mediated apoptosis by direct interaction of both CD4 and CD8 T cells via the FasL-FasR pathway. These findings identify new actionable moieties to improve the efficacy of senescence-based immunotherapies in different cancers.

Session 4: Cancer cells, tumor heterogeneity, and microenvironment

SUMOylation controls the AML surface proteome: role

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Acute Myeloid Leukemias (AML) are severe hemato-malignancies, that affect myeloid progenitors and hematopoietic stem cells. Despite recent advances in the characterization, prognosis and therapies, AML still have a poor prognosis with frequent relapses (5-year survival <20%), highlighting the need for new therapeutic strategies. We have shown that SUMOylation plays a key role in AML response to therapies. Targeting SUMOylation with TAK-981, a first-in-class inhibitor of SUMOylation, sensitizes them to therapies and favors an anti-tumor immune response in vitro and in vivo.This largely relies on the ability of TAK-981 to induce a transcriptional reprogramming in AML, including an enrichment in signatures associated to receptors and the cell membrane. This prompted us to analyze how SUMOylation controls AML cells surface proteome and modulates their interaction with their environment. We performed a cell surface proteome analysis on three AML cell lines treated or not with TAK-981. We identified more than 450 cells surface proteins whose expression at the membrane is controlled by SUMOylation. This includes various immune-cell activating/repressing ligands. For example, we confirmed, both in vitro and in vivo, that TAK-981 induces the expression of ICAM-1, which recruits Natural Killer cells. Accordingly, TAK-981 treated AML cells are more sensitive to NK-mediated cytotoxicity. In addition, many nutrient transporters and several Receptor Tyrosine Kinases (RTKs) known to regulate cell signaling were found up- or down-regulated at AML cell surface by SUMOylation. Among then, we observed a 50% decrease in FLT3, one of the most

frequently mutated genes in AML, associated with a poor prognosis. We could confirm the decrease of FLT3 at cell surface level in cell lines and patient samples treated with TAK-981. In conclusion, SUMOylation controls AML cell surface proteome and its targeting could both favor their recognition by immune cells and limit oncogenic signaling.

MYCN-driven oncogene dosage heterogeneity determines therapy response in neuroblastoma

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Neuroblastomas pose significant clinical challenges due to their heterogeneous response to therapy, particularly in cases with MYCN amplification. Despite their sensitivity to neoadjuvant chemotherapy, these tumors frequently relapse. Thus, it is crucial to understand the underlying mechanisms of treatment resistance. In neuroblastoma, MYCN amplification can occur on a chromosome segment (HSR) or on extrachromosomal DNA (ecDNA). While linear amplified oncogenes segregate evenly onto daughter cells during cell division, ecDNAs lack centromeres and segregate unequally during mitosis. This process generates cells with variable oncogene copy numbers, increasing tumor heterogeneity. To investigate this heterogeneity, we quantified MYCN copy numbers using fluorescence in situ hybridization across primary tumors, patient-derived xenografts, and cell lines. The analysis revealed pronounced ecDNA- driven intra- and intertumor heterogeneity, supported by mathematical modeling. Colony formation experiments and spatial proteomics confirmed that ecDNA-driven genetic differences guide phenotypic diversity in MYCN-high versus MYCN-low cells. We show that ecDNA and HSR-amplified cell lines react differently to therapy. HSR cells are highly sensitive to chemotherapy, whereas ecDNA cells exhibited both apoptosis and senescence, facilitating regrowth upon drug withdrawal. Notably, ecDNA copy numbers dynamically change under therapy, shaping the tumor phenotype and treatment response. Cells with high MYCN copy numbers die in response to treatment, while cells with low MYCN copy numbers survive therapy, either persisting in a senescent state or regaining cells with low MYCN copy numbers survive therapy, either persisting in a senescent state or regaining proliferation capacity. Therefore, targeting these MYCN-low cells should overcome treatment resistance and improve overall survival. In conclusion, our integrated analyses of genetic and phenotypic heterogeneity suggest refining treatment protocols to tackle the challenges posed by neuroblastomas with extrachromosomal MYCN amplification.

Unmasking the early events of meningioma Tumorigenesis

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Meningiomas are the most frequent tumors in the central nervous system. However, the cell of origin, the timing of initiating event, or the initiation mechanisms in the 20% of meningiomas without known driver mutations are still unknown. Progestin agonists(PA) lead to a significant increase of meningioma development, with patients presenting multiple meningiomas located at the skull base. We provide new insight in meningioma initiation mechanisms through the description of the mutational landscape of normal meninges. Thanks to ultradeep targeted exome sequencing of 90 normal samples of every layers of meninges, we describe mutations in NF2 and TRA7 genes (the two main driver genes of meningiomas) at very low frequency in normal meninges. Our results suggest that mutant clones are present physiologically in the "normal" meninges without micro or macroscopic signs of meningiomas. Moreover, thanks to multiomic analyses (WES, RNA Seq and methylation study) of multiple meningiomas in 15 patients under PA, we show that PA induces a selection of specific mutational clones on a location-specific manner at the skull base, explaining the genetic background and the phenotype of these particular meningiomas. Taken together, our results are in favour of a complex initiation mechanisms, where mutant clones are under control in normal tissues, that are able to trigger tumor formation under the effect of external agent (such as high progesterone levels) or accumulation of genetic events (such as copy number variations or chromosomal abnormalities).

Identification of molecular determinates for tailored treatments in BRAF^{V600E} CRC by using preclinical models

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Background
The $BRAF^{V600E}$ mutation is present in about 8-10% of the colorectal cancer (CRC) and is associated with poor prognosis. Chemotherapy remains the mainstay of palliative treatments for $BRAF^{V600E}$ metastatic CRC (mCRC), with limited efficacy. Despite encouraging results leading to the approval of a new chemo-free regimen for 2nd and 3rd line treatment, most patients still do not respond, thus highlighting an unmet clinical need. Molecular analysis of $BRAF^{V600E}$ CRC have pointed out the heterogeneity of this disease, but still those patients are treated similarly with no stratification.

Aims
With the current study we aimed at identifying potential intrinsic vulnerabilities for each different molecular subgroup of BRAF $BRAF^{V600E}$ CRC by using preclinical models such as cell lines and patient derived organoids (PDOs).

Methods
A total of nine and ten $BRAF^{V600E}$ CRC cell lines and PDOs were treated with the following drugs in short term proliferation assays, respectively: erlotinib (OSI-744), encorafenib (LGX818), 5-flurouracil (5-FU), irinotecan (SN-38), oxaliplatin (L-OHP), OSI-744+LGX818, OSI-744+SN-38, 5-FU+SN-38, 5-FU+L-OHP, 5-FU+SN-38+L-OHP, vinorelbine. Sensitivity was being defined by IC50, AUC and Synergy Score. All models underwent baseline WES, RNAseq, and Mass spectrometry. Models were classified in different molecular subgroups and prediction to drug response was investigated.
Results

Dose response data and molecular data confirmed the heterogeneity of $BRAF^{V600E}$ CRC. Molecular subgroups of response and mechanisms of intrinsic resistance to the above mentioned combinations were identified.

Conclusions

$BRAF^{V600E}$ mutation alone is not a sufficient marker for responsiveness to conventional treatment in CRC. Molecular stratification based on gene expression and mutation analysis is required to better predict response to conventional treatment. Validation of our in vitro data with clinical datasets is ongoing.

LIST OF POSTER PRESENTATIONS

P-1: Identification of mechanisms of primary resistance to CHK1 inhibitors in BRAFV600E CRC PDOs *Diogo ABREU Session 1*

P-2: A potent agonist-based PROTAC targeting Pregnane X Receptor that delays colon cancer relapse *Lucile BANSARD Session 2*

P-3: Targeting pre-leukemic cells in B-acute lymphoblastic leukemia *Jeremy BIGOT Session 1*

P-4: Role of enterotropic CD8+ T cells in the control of colorectal cancer metastases *Ylan BLANCHARD Session 2*

P-5: Dual role of mir-125b in progression and response to chemotherapy in breast carcinoma *Verorina BOUŠKOVÁ Session 1*

P-6: CD38, CD39, and BCL2 differentiate disseminated forms of high-grade B-cell lymphomas in biological fluids from Burkitt lymphoma and diffuse large B-cell lymphoma *Caroline BRET Session 2*

P-7: Optimized NGS-based de novo MET amplification detection for improved lung cancer patient management *Simon CABELLO-AGUILAR Session 1*

P-8: Replication stress associated micronucleation of extrachromosomal DNA *Lotte BRÜCKNER Session 2*

P-9: Intra-tumor heterogeneity in core-binding factor acute myeloid leukemia *Raphael HABLESREITER Session 1*

P-10: Expression of transcriptional repressors of the ZBTB family in Acute Lymphoblastic Leukemia cell lines treated with Prednisolone *Gabriela María CÁLIX RODRÍGUEZ Session 2*

P-11: Plasma proteome and metabolome signatures to better understand, monitor and predict neuroblastoma evolution *Roza Suerme MIZRAK Session 1*

P-12: UNRAVELING THE CLINICAL IMPACT OF INTRATUMORAL HETEROGENEITY IN HEAD AND NECK SQUAMOUS CELL CARCINOMA *Anastasia DIELMANN Session 2*

P-13: Senescence as an antitumor mechanism in the therapy of the hepatoblastoma *Lara FANKHÄNEL Session 1*

P-14: IMGT/mAb-DB: Unraveling Mechanisms of Action in Oncology mAbs *Taciana MANSO Session 2*

P-15: Identification of specific peptides encoded by lncRNAs and presented on HLA-I in cancer *Apollinaire ROUBERT Session 1*

P-16: Lipid-mediated modulation of DNA damage signaling as a therapeutic and prognostic approach against Multiple Myeloma *Elvira GARCÍA DE PACO Session 2*

P-17: Analysis of microRNA and mRNA co-sequencing data at the single-cell level *Louise VELUT Session 1*

P-18: Impact of 5-Fluorouracil on cell plasticity *Laura JENTSCHEL Session 2*

P-19: Exploring Galectin-9 isoforms in triple negative breast cancer after irradiation. *Nour Kotaich Session 1*

P-20: Enhancing CAR T Cell Efficacy in B-NHL Through the Use of Therapy-Naive T Cells *Charlotte JUNKUHN Session 2*

P-21: Investigating a PTBP1-mediated post-transcriptional network in AML metabolism and therapy resistance *Yann Aubert Session 1*

P-22: Sodium Butyrate and Sodium Propionate modulate Cell Migration, Invasion, and Epithelial-to-Mesenchymal Transition (EMT) in Breast Cancer models *Louna KARAM Session 2*

P-23: Dynamics of clonal hematopoiesis under DNA-damaging treatment in patients with ovarian cancer *Klara KOPP Session 1*

P-24: Unraveling causes of drug resistance in BRAFV600E Colorectal Cancer using single-cell mRNA sequencing of preclinical models *Anna KOTARAC Session 2*

P-25: Impact of ecDNA re-integration on Chromatin: Insights into TOP2 and Supercoiling *Varvara-Rigina LOUMA Session 1*

P-26: Dissecting The Role of Uncontrolled Neural Stem Cell Division in Cortical Neurogenesis and Glioma Genesis: The role of DIAPH3 *Asma MAHDI Session 2*

P-27: Age-Dependent Differences in Neuroblastoma Microenvironment: Integrative Analysis of Human and Mouse Single-Cell RNA Sequencing Data *Ekaterina PETRENKO Session 1*

P-28: Deciphering FGFR1-Mediated Molecular Mechanisms Underlying Endocrine Resistance in Breast Cancer through Single Cell Multi-omics *Sofya MARCHENKO Session 2*

P-29: Oncogene inactivation-induced senescence facilitates tumor relapse *Maria Teresa NORCIA Session 1*

P-30: Cyclotron-Based new Hadron Center in Kutaisi, Georgia *Mariam OSEPASHVILI Session 2*

P-31: The role of the glutamylase TTLL6 in colon homeostasis and pathology *Vanessa PIRES Session 1*

P-32: Developing circular RNA as biomarker for therapy resistance in liquid biopsies of patients with neuroblastoma *Johannes RIEPL Session 2*

P-33: Evaluation of Cancer Adaptive Therapy in Preclinical Models *Mohamad SKAYNI Session 1*

P-34: Clonal Dynamics and Cellular Responses to Stress-Induced Toxicity in Autologous Stem Cell Transplantation *Catarina M. STEIN Session 2*

P-35: Generating a colorectal cancer specific oncogenic stress signature by integrating transcriptomic (bulk/single-cell) and proteomic datasets *Iliana Karina TRISTAN MORENO Session 1*

P-36: Deep characterization of cisplatin-induced mutations in hepatoblastoma for early detection of chemoresistance *Mallaury VIÉ Session 2*

P-37: Tracing Clonal Hematopoiesis and Tumor-Specific Mutations in Hematopoietic Progenitors of B Cell Non-Hodgkin Lymphoma (NHL) Patients *Laura WIEGAND Session 1*

P-38: Fibrillarin contributes to the oncogenic characteristics and metastasis of colorectal cancer cells and reduces sensitivity to 5-Fluorouracil *Ting WU Session 2*

P-39: Nucleic Acid Immunity and Replication Stress in Cancer Treatment *Chun-Yen YANG Session 1*

P-40: Cancer Cell Clonal Evolution in vitro Alters Ovarian Cancer Cell Line Model Resulting in Drug Response Changes *Eglė ŽYMANTAITĖ Session 2*

P-41: Prediction of metastatic melanoma response to kinase inhibitor therapies: a point of view of heterogeneity through mechanistic modeling *Sarah DANDOU Session 1*

P-42: MAPK-mediated translational control in ovarian cancers : role of the glutaminyl-tRNA synthetase QARS in DNA damage response *Martina SERAFINI Session 2*

P-43: Study of a new variant of FLT3 in Acute Myeloid Leukaemia *Claire ROUY Session 1*

P-44: Exploring Nat8L involvement in HSC self-renewal/expansion processes upon stress *Arthur POULET Session 2*

P-45: Cancer cells transfer invasive properties through collagen-tracks *Lucile ROUYER Session 1*

P-46: Primary cilia regulate colon homeostasis and pathology *Maya SARIEDDINE Session 2*

P-47: Metabolomic and Transcriptomic changes in VEGFR2 mutated melanoma cells *Anna VENTURA Session 1*

P-48: Metabolomic and functional analysis reveal that VEGFR2 R1051Q alters cell metabolism through heterodimerization *Camilla MAGGI Session 2*

P-49: Type I conventional dendritic cells and CD8+ T cells predict favorable clinical outcome of head and neck squamous cell carcinoma patients *Rebecca ROTHE Session 1*

P-50: Circumvent TGFβ-related CAR T-cell malfunction against neuroblastoma using TGFβ-inhibitor or dominant-negative TGFβ receptor *Tabea Biereder Session 2*

P-51: Decoding Immunity: Genomic Study and Biocuration of Antigen Receptor Loci in Norway Rats – A Leap Towards Translational Cancer Research) *Chilanay ALAKBAROVA Session 1*

P-52: Implication of Supra Molecular Attack Particles (SMAPs) in cytotoxic T lymphocytes lethal activity. *Frecia RODRIGUEZ Session 2*

P-53: Mapping cellular networks by mass spectrometry-based single-cell proteomics *Pierre GIROUX Session 1*

P-54: The IFNy response in tumor/stroma interactions of a novel subtype of Pancreatic Adenocarcinoma: antigenic peptide translation and Antigen-presenting CAF *Mehdi Liauzun Session 2*

P-55: Acquired chemoresistance in Pancreatic Adenocarcinoma: Mechanisms involving a stromal ZBTB family transcription factor *Hippolyte Audureau Session 1*

P-56: Targeting Iron Homeostasis As a Therapeutic Strategy in Multiple Myeloma *Laura Alibert Session 2*

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NOTES

The YS2C organizing committee warmly thanks the academic and industrial sponsors who agreed to take part in the second edition of the congress.

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