Oxford Cancer Immuno-Oncology Network (OCION)

2026 Annual Symposium

Programme Friday 19th June 2026

08:30 Arrival and registration – Poster Setup, Coffee available

09:15-09:25 **Welcome** **Tim Elliott**

09:25-09:30 **OCION Early Career Researchers Committee- ECRs committee**

Session 1: Fundamental mechanisms in tumour recognition and immune escape (chair: Tim Elliott)

09:30-09:50 "Aspirin enhances T cell immunity to cancer metastasis" - Jie Yang (Oncology/Centre for Immuno-Oncology (CIO))

09:50-10:00 "Peptide and enzyme-level dissection of a TAP–cytosolic aminopeptidase axis of tumour immune escape" - Om H. Gandhi (Centre for Immuno-Oncology (CIO))

10:00-10:20 "The Tangled Genome: Chromosomal Instability and Immune Landscape"- Eileen Parkes (Oncology/Centre for Immuno-Oncology (CIO))

10:20-10:30 "TMEM33 deletion potentiates anti-tumour CD8+ T cell immunity"- Matthew T Jackson ((Oncology/Centre for Immuno-Oncology (CIO))

10:30-10:40 "Fine-tuning innate immunity to overcome cancer immune evasion" - Adan Pinto-Fernández (CAMS Oxford Institute (COI))

10:40 -11:00 Coffee and pastries with Poster viewing

Session 2: Tumour models for evolving cancer (chair: David Withers)

11.00-11.20 "Early transformation models of colorectal cancer to study RAS driven reprogramming" - Giulia Orlando (Oncology)

11:20-11:30 "Experimental Evaluation of Neoantigen Prediction Tools Using AI and Lab Automation" - Anthony Hsieh (Centre for Immuno-Oncology (CIO))

11:30-11:50 "Deciphering Cellular and Oncogenic Determinants of Tertiary Lymphoid Structures"- Isabela Pedroza-Pacheco (Centre for Immuno-Oncology (CIO)/ CAMS Oxford Institute (COI))

11:50-12:00 "Integrating agent-based modelling and spatial statistics to study tertiary lymphoid structures in cancer" - Nicholas Lai (Mathematical Institute (Maths))

12:00-13:15 Lunch and Poster viewing

13:15-13:45 **PPI in fundamental research** Catriona Gilmour-Hamilton/ Julliet Lwiindi/ Alex Colton

Session 3: Treatment and translational cancer research (chair: Silvia Panetti)

13:45-14:05 "Deciphering the phagocytic landscape in solid tumours"- Felipe Galvez-Cancino (Centre for Immuno-Oncology (CIO))

14:05-14:15 "Developing CAR-Macrophages as Targeted Cancer Vaccines"- Ahmet Hazini (Oncology)

14:15-14:35 "Mitochondria determine the lymphopoietic response and clinical outcomes to checkpoint blockade" - Ben Fairfax (Oncology)

14:35-14:45 "Watching the immune response unfold: How pembrolizumab treatment reshapes the tumour-reactive T cell repertoire in Triple-negative breast cancer patients"- Mariana Pereira Pinho ((CAMS Oxford Institute (COI))

14:45-15:15 Poster Session with Coffee

Session 4 (chair: Tim Elliott)

15:15-16:00 **Phoenix Pitching sessions.** Introduction to the Phoenix Scheme (Ester Gea-Mallorquí). Pitchs from: César López-Camacho, Maria Aggelakopoulou, Yimon Aye's group, Eleni Adamopoulou, Malcolm Sim, Ahmet Hazini, Karl Smith Byrne and Zinaida Dedeic.

16:00-16:45 **Keynote: "Dark genome and tumour immunity" - Sebastian Amigorena (Immunity and Cancer Unit, Institut Curie, Paris)**

16:45-17:00 **Awards and closing remarks** **Tim Elliott**

17:00-18:30 **Networking drinks reception**

Oxford Cancer Immuno-Oncology Network

Keynote Speaker

Sebastian Amigorena

Sebastian Amigorena, PhD, is a leading immunologist whose work sits at the interface of immunology and cell biology, with major contributions to the field of antigen presentation. He is **currently Directeur de Recherche de Classe Exceptionnelle at CNRS**, a senior tenured position he has held since 1990, and **Team Leader at Institut Curie**, INSERM U932, PSL University, where he has led his research group since 1995.

He completed his PhD in Biochemistry and Immunology at University Paris VII in 1990, working on Fc receptor expression in B lymphocytes at the Laboratoire d'Immunologie Cellulaire et Clinique, Institut Curie, in the Fridman lab. He followed by a three-year postdoctoral fellowship at Yale University School of Medicine in the Mellman lab, focusing on the cell biology of antigen presentation in B lymphocytes.

In 1995, he returned to Paris, where he founded an Inserm AVENIR research group with Christian Bonnerot. His early research focused on IgG receptors and the molecular basis of their inhibitory properties. He later became internationally **recognized for his work on antigen presentation and cross-presentation in dendritic cells, including the endocytic and phagocytic pathways that shape immune responses**.

In 2003, Sebastian Amigorena founded INSERM Unit 932, "Immunity and Cancer," the Immunology Department at Institut Curie, and served as its director until 2021. He is also co-founder of Mnemo Therapeutics, established in 2018, and served as its Chief Scientific Officer from January 2023 to August 2025.

Amigorena and his team have made major contributions to understanding cytotoxic T cell dynamics in vivo, both during the initiation of immune responses in lymph nodes and during the invasion and rejection of solid tumors. More recently, his group has investigated the epigenetic programming of regulatory and memory T cells. Today, his team focuses on the biology of transposable elements, their regulation, and their roles in anti-tumor immunity.

Session 1:
Fundamental mechanisms in tumour recognition and immune escape

Chair: Tim Elliott

Dr. Jie Yang

"Aspirin enhances T cell immunity to cancer metastasis"

Department of Oncology/Centre for Immuno-Oncology (CIO)

Om H. Gandhi

"Peptide and enzyme-level dissection of a TAP–cytosolic aminopeptidase axis of tumour immune escape"

Centre for Immuno-Oncology (CIO)

Poster #5

Prof. Eileen Parkes

"The Tangled Genome: Chromosomal Instability and Immune Landscape"

Department of Oncology/Centre for Immuno-Oncology (CIO)

Dr. Matthew T Jackson

"TMEM33 deletion potentiates anti-tumour CD8+ T cell immunity"

Department of Oncology/Centre for Immuno-Oncology (CIO)

Poster #14

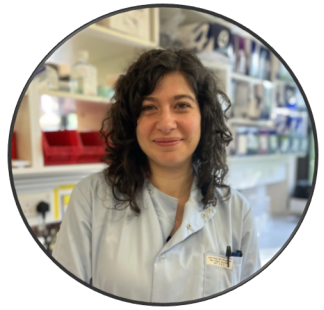
Dr. Adán Pinto-Fernández

"Fine-tuning innate immunity to overcome cancer immune evasion"

Chinese Academy for Medical Sciences Oxford Institute

Session 2:
Tumour models for evolving cancer

Chair: David Withers



Dr. Giulia Orlando

"Early transformation models of colorectal cancer to study RAS driven reprogramming"

Department of Oncology

Dr. Anthony Hsieh

"Experimental Evaluation of Neoantigen Prediction Tools Using AI and Lab Automation"

Centre for Immuno-Oncology (CIO)

Poster #13

Dr. Isabela Pedroza-Pacheco

"Deciphering Cellular and Oncogenic Determinants of Tertiary Lymphoid Structures"

Centre for Immuno-Oncology (CIO /Chinese Academy for Medical Sciences Oxford Institute

Nicholas Lai

"Integrating agent-based modelling and spatial statistics to study tertiary lymphoid structures in cancer""

Mathematical Institute

Poster #6

Session 3:
Treatment and translational cancer research

Chair: Silvia Panetti

Oxford Cancer Immuno-Oncology Network

Dr. Felipe Gálvez-Cancino

"Deciphering the phagocytic landscape in solid tumours"

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Chinese Academy for Medical Sciences Oxford Institute

Poster #9

Short talks

Om H. Gandhi

"Peptide and enzyme-level dissection of a TAP–cytosolic aminopeptidase axis of tumour immune escape"

Poster #5

Om H. Gandhi[1], Michael E. Bryan[1], Miles Weatherseed[1], Rishi Patel[1], Neil Dalchau[2], Eamonn Gaffney[3], Mark Coles[4], Malcolm JW Sim[1], Tim Elliott[1]

[1] *Centre for Immuno-Oncology, University of Oxford, Oxford, United Kingdom;* [2] *Microsoft Research, Cambridge, United Kingdom;* [3] *Wolfson Centre for Mathematical Biology, University of Oxford, Oxford, United Kingdom;* [4] *Kennedy Institute of Rheumatology, University of Oxford, Oxford, United Kingdom*

Tumors may escape $CD8^+$ surveillance by remodeling the antigen-processing machinery (APM) upstream of MHC class I. We recently-developed Predictor Of immunogenic Epitopes using Mechanism (POEM), a mechanistic ODE model of class I processing scaled by patient RNA-seq, which identifies a dominant escape trajectory in TRACERx NSCLC: coordinated TAP downregulation and cytosolic aminopeptidase (CytAmin) upregulation. The TAP–CytAmin ratio stratifies immune-checkpoint response in SU2C-MARK above PD-L1 and IFN-γ. However, these cohort-level analyses cannot predict patient-specific peptide vulnerabilities along the axis.

We extend POEM at two resolutions through a hybrid computational–in vitro system. First, peptide sensitivity to the TAP–CytAmin axis: each candidate epitope receives a six-component elasticity vector (±25%) and dynamic ranges from a 17-point dose–response sweep (0.2×–2.5×); a prioritization score then ranks pair/triplet combinations whose predicted presentation diverges maximally on TAP–CytAmin but is matched on ERAP1 and tapasin. In A375 melanoma, ICP47 plus NPEPPS co-expression produces dose-dependent reductions in surface HLA-ABC by W6/32 staining: double transfectants reduce MFI ~47% ($p<0.0001$), versus ~15% for NPEPPS alone ($p=0.004$) and ~13% for ICP47 alone ($p=0.01$). Second, peptide sensitivity to specific enzymes: in TRACERx, the five aminopeptidase genes co-vary on one transcriptional axis (within-patient r=0.86–0.96; NPEPPS tracks closest), justifying POEM's collective-species treatment.

pMHC-specific TCR-Jurkat assays will both evaluate peptide sensitivity to the TAP–CytAmin axis and identify peptides whose precursors best match aminopeptidase N-terminus specificity. This approach enables rational vaccine design by using POEM to prioritize peptide pools enriched for epitopes resistant to immune escape via TAP–CytAmin regulation.

Short talks

Matthew T Jackson

"TMEM33 deletion potentiates anti-tumour CD8+ T cell immunity"

Poster #14

Matthew T Jackson[1,2], Tianming Zhao[1,2], Gusztav Milotay, Isabela Pedroza-Pacheco, Yanshu Cai, Claire Willis, Su M Phyu, Bruno Beernaert, Jamie TW Kwon, Hala Estephan, Vinnycius Pereira Almeida, Maria Aggelakopoulou, Silvia Panetti, Christian Zierhut, Tim Elliott, Eleni Adamopoulou, Jan Rehwinkel, Malcolm JW Sim, Amit Grover, Dmitry I Gabrilovich, Benjamin P Fairfax, Eric Honoré, David R Withers, John C Christianson, Eileen E Parkes.

[1] *Centre for Immuno-Oncology, University of Oxford, Oxford, United Kingdom;* [2] *Department of Oncology, University of Oxford, Oxford, United Kingdom*

Improving responses to cancer immunotherapies requires deeper insight into the cellular mechanisms governing T cell-mediated anti-tumour immunity. TMEM33 is an endoplasmic reticulum-resident transmembrane protein enriched across multiple tumour types, with reported functions in anti-viral immunity as well as calcium and lipid homeostasis, yet its role in tumour immunosurveillance remains unknown. Using murine genetic models, we demonstrate that host TMEM33 constrains anti-tumour CD8+ T cell responses. Constitutive Tmem33-/- mice exhibited delayed melanoma tumour growth and increased CD8+ T cell infiltration. Antigen-specific CD8+ compartments in tumours of Tmem33-/- mice showed TCF-1+PD-1+ progenitor-exhausted cell (Tpex) enrichment, elevated effector function and reduced exhaustion, alongside improved effector memory expansion and T-bet expression in draining lymph nodes. We highlight that TMEM33 functions intrinsically within the T cell compartment, as TMEM33 deletion (1) enhanced polyclonal activation of naive CD8+ T cells ex vivo, (2) promoted preferential Tpex accumulation among adoptively transferred naive OT-I cells in B16F10-OVA tumours and draining lymph nodes, and (3) improved the potency of ex vivo-expanded OT-I cells in controlling tumour growth during adoptive cell therapy. Finally, in a large, prospectively recruited metastatic melanoma cohort, lower TMEM33 expression in patient CD8+ T cells significantly correlated with improved survival and elevated TCF-7 (encoding TCF-1). Collectively, our findings define TMEM33 as a formerly unrecognized intrinsic determinant of tumour-directed CD8+ T cell fate that limits Tpex maintenance, and restrains cell therapy responses, suggesting that its modulation may strengthen immunotherapeutic efficacy.

Short talks

Adán Pinto-Fernández

“Fine-tuning innate immunity to overcome cancer immune evasion”

Simeon D. Draganov[1], Haofeng Chen[1], Hedib Alrawili[1], Melisa Cetinkaya[1], Meshal Alghanem[1], Alice Beard[1], Angela Chen[1], ilknur Sur Erdem[1], and Adán Pinto-Fernández[1].

[1] *Chinese Academy for Medical Sciences Oxford Institute, University of Oxford, Oxford, United Kingdom.*

Type I interferon (IFN-I) signalling is a central regulator of anti-tumour immunity, promoting antigen presentation, chemokine secretion and immune cell activation. However, chronic or aberrant activation of this pathway can contribute to systemic inflammation, toxicity and resistance to immunotherapies, while many tumours rewire or attenuate IFN-I outputs to avoid immune recognition and killing. My research programme aims to define and therapeutically exploit tumour-intrinsic regulators that fine-tune IFN-I signalling.

We focus on a class of interferon fine-tuning modulators, including USP18 and USP24, among others. Our previous work identified USP18 as a key suppressor of IFN-I signalling, tumour antigenicity and radiosensitivity, establishing a mechanistic link between post-translational regulation, innate immunity and cancer immune evasion. Building on this, we are using functional genomics, activity-based proteomics, immunopeptidomics, lipidomics and tumour-immune functional assays to define how these modulators control antigen presentation, inflammatory tone and tumour immune recognition.

Short talks

Anthony Hsieh

“Experimental Evaluation of Neoantigen Prediction Tools Using AI and Lab Automation”

Poster #13

Anthony Hsieh[1], Lennard Lee[1].

[1] *Centre for Immuno-Oncology, University of Oxford, Oxford, United Kingdom*

Accurate prioritisation of tumour neoantigens remains a challenge for personalised cancer vaccine development and is bottlenecked by a dearth of functional immunogenicity data. We sought to evaluate and refine peptide prediction tools by testing predicted class I neoepitopes derived from melanoma, prostate cancer, and glioblastoma patients in the Cancer Genome Atlas Program. Patient tumour and germline sequencing were used to identify tumour mutations, from which candidate minimal peptides will be predicted using established prediction tools such as NetMHCPan, Stabpan, MHCflurry, PRIME, and BigMHC, in addition to a novel AI-based prediction tool CancerVaccineGPT. Patients will be HLA-matched with healthy donors used for in vitro priming assays.

Candidate peptides will be tested using cultured ELISpot. Because expected precursor frequencies for neoantigen-reactive naïve T cells are low, many designated high-priority neoantigens are unlikely to yield detectable signal. Therefore, our aim is to improve the hit-rate of algorithmic predictions through iterative improvement of CancerVaccineGPT by generating high-throughput tumourigenicity data. By including 5 patients per cancer type and 20 neoepitopes per patient, the scope of our project will be on the order of 1000 peptides. To considerably improve throughput, we are building a prototype automated lab with collaborative robotics and liquid handling, all controlled through API by our AI, CIARA (Centre for Immuno-oncology AI Research Agent).

This study will generate a systematically annotated dataset linking predicted peptide-HLA interactions to functional T-cell responses under controlled experimental conditions. This study aims to identify determinants of successful neoantigen detection and improve rational selection of vaccine candidates

Short talks

Nicholas Lai

"Integrating agent-based modelling and spatial statistics to study tertiary lymphoid structures in cancer"

Poster #6

Nicholas Lai[1,2], Fengyun Zhong[1], Joshua Bull[2], Tim Elliott[1], Isabela Pedroza-Pacheco[1,3], Helen Byrne[2]

[1] *Centre for Immuno-Oncology, University of Oxford, Oxford, United Kingdom;* [2] *Mathematical institute, University of Oxford, Oxford, United Kingdom;* [3] *Chinese Academy for Medical Sciences Oxford Institute, University of Oxford, Oxford, United Kingdom.*

Tertiary lymphoid structures (TLSs) are organised immune aggregates that form within tumours and are associated with improved patient outcomes. TLSs exhibit diverse spatial organisation, from diffuse lymphocyte aggregates to highly structured regions with distinct B- and T-cell zones. The processes driving this spatial maturation remain poorly understood. We combine spatial statistics and agent-based modelling to investigate how local cellular interactions generate the global organisation observed in TLS maturation.

We develop a spatial analysis pipeline to detect and characterise TLSs in colorectal cancer using spatial transcriptomic data. We quantify immune cell organisation using spatial statistics, enabling identification of TLS regions and characterisation of their structure. Using principal component analysis, we order TLSs along a notional maturation axis. These results suggest a maturation trajectory from well-mixed lymphocyte aggregates to spatially segregated B- and T-cell compartments.

To investigate the mechanisms underlying this progression, we develop an agent-based model of TLS formation. The model describes interactions between B-cells, T-cells, stromal cells, and diffusible chemokines—CXCL13 and CCL19. B and T cells are recruited into the tissue and migrate in response to chemokine cues produced by stromal cells. Feedback between lymphocytes and stromal cells drives changes in chemokine expression, leading to the emergence of TLS-like spatial organisation.

Finally, we integrate these approaches by applying the same spatial metrics to model simulations and map the simulated TLSs onto the experimentally derived maturation axis. This provides a direct link between the spatial structure observed in TLSs in vivo and the cellular interactions that generate them. Together, this work establishes a quantitative framework for identifying and comparing TLS organisation in tumour tissue, and linking biological observations with mechanistic models of TLS maturation

Short talks

Mariana Pereira

"Watching the immune response unfold: How pembrolizumab treatment reshapes the tumour-reactive T cell repertoire in Triple-negative breast cancer patients"

Poster #9

Mariana Pereira Pinho[1,2], Balraj Sandhar[1,2], Elie Antoun[1], Ting Shu[1], Magdalena Kielak[3], Rooman Javed[3], Aglaia Skolariki[3], Caroline Avelar[3], Emma Johnson[3,4], David Maldonado-Perez[5], Adrian L. Harris[3], Yanchun Peng[1,2,], Laura Spiers[3], Simon Lord[3], Tao Dong[1,2]

[1] CAMS Oxford Institute (COI), Nuffield Department of Medicine, University of Oxford, UK; [2] MRC Translational Immune Discovery Unit, Weatherall Institute of Molecular Medicine (WIMM), Radcliffe Department of Medicine, University of Oxford, UK; [3] Department of Oncology, University of Oxford, UK; [4] Sample Handling Lab, Oxford Cancer Trials Unit, UK;
[5] Nuffield Department of Surgical Sciences, University of Oxford, John Radcliffe Hospital, UK

Background: Immune checkpoint blockade has improved outcomes in patients with triple-negative breast cancer (TNBC), yet many patients fail to achieve durable benefit. A better understanding of treatment-induced immune changes may help identify mechanisms of response and inform biomarker development. In this study, we longitudinally profiled circulating CD8 T cell responses in TNBC patients treated with pembrolizumab. *Method:* Peripheral blood samples from TNBC patients were collected before and at different timepoints after pembrolizumab treatment. Circulating CD8 T cells were profiled using CITE-seq combined with single-cell T cell receptor sequencing (scTCR-seq) to simultaneously assess transcriptional states, surface protein expression, and TCR clonotypes. Multiparametric flow cytometry was performed to validate phenotypic changes. Tumour-reactive TCRs were identified by co-culturing patient-derived T cells with autologous dendritic cells loaded with tumour antigens, followed by TCR sequencing. Identified tumour-reactive TCRs were matched to clonotypes detected by scTCR-seq, allowing the analysis of the phenotype of tumour-reactive T cells. *Results:* Both multiparametric flow cytometry and CITE-seq revealed an increase in circulating activated CD8 T cells following pembrolizumab treatment. Ex vivo anti-IgG4 staining confirmed pembrolizumab binding to activated circulating CD8 T cells after treatment. Longitudinal TCR tracking demonstrated dynamic changes of the tumour-reactive repertoire during therapy. Early during treatment, some pre-existing tumour-reactive clonotypes declined or transiently disappeared from the circulation. At later timepoints, we observed the emergence of newly detectable tumour-reactive clonotypes as well as expansion of tumour-reactive clones that were present at baseline but increased in frequency with treatment. These changes were selective for tumour-reactive clonotypes since they were not observed among pathogen-specific TCRs. In a patient with matched tumour tissue available at surgery, TCR clonotypes present within activated circulating CD8 T cells after treatment were also detected in the tumour, supporting a link between systemic clonal expansion and intratumoral T cell responses. *Conclusions:* Our data show that PD-1 blockade in TNBC specifically reshapes the tumour-reactive circulating repertoire through selective contraction, emergence, and expansion of clonotypes. The detection of treatment-associated clones in both blood and tumour highlights the potential of circulating T cell profiling to reflect intratumoral immune dynamics.

Poster Abstracts

Poster #1 –Amy Bailiss

Exploiting the 'helper-like' functions of NK cells to enhance anti-tumour immunity

Amy Bailiss[1], Tanusya M. Murali[1], Malcolm J W Sim[1], Maria Aggelakopoulou[1]

[1]Centre for Immuno-Oncology, University of Oxford

Background: Natural killer (NK) cells are widely recognised for their cytotoxic activity against cancerous cells. In murine tumour models, activated NK cells release the mediators XC motif chemokine ligand 1 (XCL1) and Fms-like tyrosine kinase 3 ligand (FLT3L), which recruit and sustain the survival of conventional type I dendritic cells (cDC1s) within the tumour microenvironment. cDC1s are specialised for antigen cross-presentation and are required for $CD8^+$ T cell priming and effective anti-tumour immunity. In cancer patients, increased NK cell and cDC1 infiltration correlates with $CD8^+$ T cell infiltration and improved prognosis. However, the upstream stimuli that license human NK cells to produce XCL1 and FLT3L and support cDC1 recruitment remain unclear.

Methods: Human peripheral blood NK cells, isolated from healthy donors, were activated using three different stimulation conditions: cytokine stimulation, activating receptor engagement, and co-culture with the HLA-null leukaemia cell line K562. XCL1 and FLT3L secretion was quantified by ELISA 48 hours and 72 hours later.

Results: Production of XCL1 and FLT3L was minimal in response to cytokine only stimulation. Engagement of activating receptors using the defined ligands ULBP1 and CD155 induced low but detectable release of both mediators. In contrast, co-culture with K562 cells induced the strongest production of XCL1 and FLT3L, suggesting that multiple or synergistic receptor-ligand interactions may be required to license this NK cell helper programme. The receptor-ligand interactions identified will be presented.

Conclusion: These findings suggest that receptor-ligand interactions, rather than cytokine stimulation alone, are responsible for driving NK cell production of XCL1 and FLT3L. Defining these interactions will help to exploit NK cell helper-like functions to enhance anti-tumour immunity. Ongoing work will investigate how XCL1 and FLT3L shape cDC1 phenotype and downstream $CD8^+$ T cell responses.

Poster #2 –Medina Abudula

Cholangiocyte–Tumour Crosstalk Drives Inflammatory and Metabolic Reprogramming in Cholangiocarcinoma

Medina Abudula[1], Gemma Owen[1], David Kerr[1], Shijie Cai[1]

[1]Nuffield division of clinical laboratory sciences, Redcliff Department of Medicine, Medical Science Division

Background: Cholangiocarcinoma (CCA) is a highly aggressive malignancy of the bile ducts with poor prognosis and limited therapeutic options. While the tumour microenvironment is known to support CCA progression, the contribution of cholangiocytes, the biliary epithelial cells from which CCA originates, to early tumour development and inflammatory signalling remains poorly understood. Here, we investigated the early crosstalk between cholangiocytes and CCA cells and its impact on tumour cell behaviour, cytokine signalling, and metabolic adaptation.

Methods: Indirect co-culture models were established between cholangiocytes (MMNK1) and CCA cell lines. Functional assays assessing cell apoptosis, invasion, viability, and clonogenicity were combined with transcriptomic, metabolomic, cytokine secretome, and protein expression analyses to characterise tumour–cholangiocyte interactions.

Results: Co-culture with cholangiocytes increased apoptosis and reduced invasive capacity in CCA cells, accompanied by altered epithelial–mesenchymal transition (EMT)-associated protein expression consistent with a partial EMT state. Omics analyses identified metabolic rewiring in CCA cells, including altered branched-chain amino acid metabolism and accumulation of 2-oxoisopentanoate (2-OXIP), which suppressed tumour cell clonogenicity and invasion. In parallel, cholangiocytes exhibited enhanced survival, increased ATP availability, and sustained production of inflammatory cytokines, including IL-6 and CCL5, suggesting activation of stress- and inflammation-associated signalling pathways. Cytokine perturbation studies further identified IL-6 as a key survival factor for cholangiocytes during tumour interaction.

Conclusions:These findings demonstrate that bidirectional signalling between cholangiocytes and CCA cells promotes inflammatory and metabolic reprogramming prior to full malignant progression. Our data identify cholangiocytes as active regulators of the early tumour microenvironment and highlight cytokine-mediated signalling and metabolic vulnerabilities as potential targets for future therapeutic and immunomodulatory investigation in cholangiocarcinoma.

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Poster Abstracts

Poster #3 –Masha Sychevskaya

Investigating the monocyte to tumour-associated macrophage transition in real time

Masha Sychevskaya[1], Pascale Wehr[1], Paul Tarlatt[1], Nabina Pun[1], Claire Willis[1], Katie Holden[2], David Withers[1]

[1]Centre for Immuno-Oncology, University of Oxford; [2]Centre for Human Genetics, Cellular Imaging Core Facility, University of Oxford

Introduction: Tumour-associated macrophages (TAMs) are highly abundant, functionally-heterogenous and considered key drivers of cancer progression and current immunotherapy failure. TAMs are either tissue-resident or circulating monocyte-derived, with functional consequences. Clinical strategies targetting this compartment, such as blocking monocyte recruitment (e.g. anti-CCR2) or re-programming towards an anti-tumour phenotype (e.g. CSF1R inhibitors), have had minimal success to date. This likely reflects an incomplete understanding of how TAM populations form, persist and evolve, and over what time scales. Here we sought to improve understanding through dissecting how monocytes differentiated into TAMs in real time.

Methods: We performed droplet-based single-cell RNA sequencing of orthotopic YUMM1.7 melanoma tumours from Kaede photoconvertible mice, enabling transcriptomic profiling of intratumoural immune cells at 24 and 72 hours post-photolabelling. Leukocyte infiltration into the tumour was tracked with anti-CD45 intravenous labelling. TAM origin was further assessed using the Ms4a3cre × Rosa26LSLTdTomato fate-mapping mice, analysed using flow cytometry and immunofluorescence.

Results:Transcriptomically, newly-entering monocytes have a pro-inflammatory and interferon-stimulated signature, but within 24 hours of tumour residence, they acquire an angiogenic and arginine-metabolising, pro-tumour phenotype. Ligand-receptor analysis suggested that this rapid monocyte recruitment could be driven by tumour cell expression of Csf1 and Ccl2. Comparisons with the TAM compartment persisting for at least 72 hours indicated that some states are highly transient and constantly replenished. A subset of cells present in the tumour for ≥ 24 hours demonstrated a tissue-residency phenotype with a high expression of complement-associated genes. RNA velocity analysis implies that these could give rise to lipid-scavenging Trem2+ macrophages. Fate-mapping with the Ms4a3-Ai9 mouse confirmed that the macrophage compartment in YUMM1.7 tumours is predominantly monocyte-derived and densely distributed throughout the tumour.

Conclusion:These data reveal the rapid differentiation of monocytes into multiple TAM states that appear to coalesce over a time-span of days to a pro-tumour/wound healing functional state.

Poster #4 –Oluwafemi Akin-Adigun

Pre-clinical human immunogenicity assessment of a preventive lung cancer vaccine targeting shared oncogenic drivers

Robert Harris[1], Oluwafemi Akin-Adigun[1], Ava Connolly[1], Yanshu Cai[1], Navrose Singh[1], Michael Bryan[1], Adam Kelly[1], Emery Hoos[1], Tanusya M. Murali[1], Malcolm J W Sim[1], Tim Elliott[1], Maria Aggelakopoulou[1]

[1]Centre for Immuno-Oncology, University of Oxford

Preventive cancer vaccination represents a promising strategy to reduce disease recurrence and intercept tumour development before clinically significant disease emerges. LungVax is an off-the-shelf preventive cancer vaccine designed to target recurrent oncogenic drivers in NSCLC, including TP53, KRAS and EGFR hotspot mutations, together with the tumour-associated antigens NY-ESO-1 and MUC1. Two complementary clinical development programmes are currently underway using distinct delivery platforms: a ChAdOx2 adenoviral vector and an mRNA-lipid nanoparticle (mRNA-LNP) vaccine. Prior to clinical evaluation, it is essential to establish whether vaccine-encoded antigens can be processed, presented and recognised by human immune cells and to define the breadth and variability of human T cell responses against the encoded antigen repertoire. Using complementary human in vitro immunogenicity platforms, we evaluated antigen presentation and T cell responses against LungVax-encoded antigens in healthy donors and treatment-naive patients with NSCLC. Antigen presentation was assessed using monocyte-derived dendritic cells (MoDCs) pulsed with vaccine-derived antigens or exposed to the ChAdOx2-LungVax vaccine construct and subsequently co-cultured with antigen-specific TCR-expressing Jurkat reporter cells. In parallel, PBMC-based T cell assays employing overlapping peptide pools spanning the vaccine-encoded regions were used to assess antigen-specific T cell responses. To model vaccine-induced priming, we also established a human co-culture system in which MoDCs transduced with ChAdOx2-LungVax were co-cultured with naïve healthy donor T cells, followed by assessment of antigen-specific T cell priming using IFN-γ ELISpot, tetramer staining and intracellular cytokine staining.
These studies demonstrate functional activation of multiple vaccine-specific TCRs and provide evidence that LungVax antigens can be processed and presented by human antigen presenting cells. Antigen-specific T cell responses were elicited against several vaccine-derived peptide pools, including IFN-γ production measured by ELISpot and expansion of polyfunctional IFNγ+TNFα+ CD4 and CD8 T cell populations. Importantly, preliminary data from the MoDC–naïve T cell co-culture system demonstrate successful priming of antigen-specific T cell responses following exposure to the ChAdOx2-LungVax vaccine. Immune responses were observed in both healthy donors and treatment-naive patients with NSCLC, although substantial inter-individual variation in the magnitude and specificity of responses was evident. Ongoing studies aim to identify immunodominant epitopes, compare responses across vaccine delivery platforms and define determinants of human vaccine responsiveness.

Oxford Cancer Immuno-Oncology Network

Poster Abstracts

Poster #7 –Fengyun Zhong

Deciphering B-Cell-Centric Immune Hubs in Colorectal Cancer

Fengyun Zhong[1], Nicholas Lai[2], William Serkin[1,3], Zheheng Fan[1], Yanshu Cai[1], Alistair Easton4, Kezia Gaitskell[5], Maria Greco[5], Helena Coker[6], Joshua Bull[2], Marco Fritzsche[6], Helen Byrne[2], Timothy Elliott[1], Isabela Pedroza-Pacheco[1]

[1]Centre for Immuno-Oncology, Nuffield Department of Medicine, University of Oxford; [2]Mathematical Institute, University of Oxford; [3]National Institutes of Health (NIH), Bethesda, Maryland, USA; [4]Department of Oncology, University of Oxford,; [5]Radcliffe Department of Medicine, University of Oxford,; [6]Kennedy Institute of Rheumatology, University of Oxford

Background: Tumour-infiltrating B cells (TIL-Bs) have been associated with favourable prognosis in multiple cancer types and are predominantly localised within tertiary lymphoid structures (TLSs). However, the spatial organisation, phenotypic diversity, and functional roles of TIL-B subsets in colorectal cancer (CRC) remain incompletely understood.

Methods: A multi-omic strategy integrating single-cell RNA sequencing, B-cell receptor (BCR) profiling, Xenium spatial transcriptomics, high-parameter spectral flow cytometry, and immunofluorescence imaging was employed to characterise the phenotypes, clonality, spatial distribution, and functional properties of TIL-Bs in CRC at single-cell resolution.

Results: Microsatellite instability-high (MSI-H) CRC patients who responded favourably to anti-PD-1 therapy exhibited enrichment of TLS-associated transcriptional modules together with enhanced immune cell recruitment and activation-related cell–cell interactions. Spatial transcriptomic analyses revealed heterogeneity in TLS abundance and maturation states across CRC samples and identified distinct IgG^+ antibody-secreting cell (ASC)–macrophage–tumour cell triads. BCR profiling demonstrated increased somatic hypermutation and clonal expansion of IgG BCRs in tumour tissues relative to adjacent normal tissues. IgG^+ TIL-Bs represented the dominant isotype and displayed the highest mutation frequencies, predominantly localising within ASC–macrophage–tumour triads. Flow cytometry and immunofluorescence confirmed IgG binding to primary tumour cells, while patient-derived plasma from selected CRC cases also showed reactivity against CRC cell lines.

Conclusions: $CD20^+$ B cells were predominantly enriched within TLSs in CRC and were associated with favourable anti-PD-1 treatment responses. TIL-Bs exhibited evidence of antigen-driven maturation, clonal expansion, and IgG class switching within the tumour microenvironment. The identification of IgG^+ ASC–macrophage–tumour cell triads, together with tumour-reactive IgG antibodies in both tumour tissue and patient plasma, supports a potential anti-tumour role for B cells mediated through antibody-dependent effector mechanisms. These findings provide new insights into the spatial architecture, maturation, and functional dynamics of B cells in CRC and highlight their potential as therapeutic targets for next-generation immunotherapies and cancer vaccines.

Poster #8 –Emanuele Marchi

The HERVome as a Guardian of Immune Homeostasis in Cancer and Disease

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We performed a comprehensive transcriptomic analysis across diverse human tissues, immune populations, and pathological conditions to characterise the expression landscape of human endogenous retroviruses (HERVs) and investigate their functional and therapeutic relevance. Integrating bulk and single-cell RNA sequencing datasets, we identified context-specific HERV activation patterns associated with tumour progression, epigenetic remodelling, and cellular dedifferentiation. Several HERV families were enriched in cancers displaying stem-like and hypomethylated phenotypes, supporting their potential utility as biomarkers of demethylation and cancer stemness. In parallel, multiple HERV-derived transcripts encoded putative neoantigenic peptides selectively expressed in malignant tissues, highlighting their promise as preferential targets for immunotherapy.

Beyond cancer-specific activation, we identified immune cell-restricted HERV expression signatures, suggesting broader regulatory roles in immune function and homeostasis. Distinct HERV loci were preferentially expressed in specific immune populations, consistent with potential involvement in immune signalling, surveillance, and cellular identity programs. Based on these observations, we propose that the “HERVome” functions as a dynamic guardian of immune homeostasis. In conditions in which cancer or persistent pathogens induce immune escape, dormant HERV elements may be reactivated, contributing to the restoration of effective immunity through innate immune stimulation, inflammatory signalling, and enhanced antigen presentation.
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Poster Abstracts

Poster #10 –Haofeng Chen

USP18 is a cell-intrinsic, active-site–dependent suppressor of anti-tumour T-cell function and a candidate target for cancer immunotherapy

Haofeng Chen[1], Nicole Patrizia Kadzioch[2], Simeon Draganov[1], Ilknur Sur Erdem[1], Melisa Çetinkaya[1], Meshal Al-Ghanem[1], Pablo Céspedes[1], Delaney C. C. Dominey-Foy[1], Xuan Yao[1], Fei Gao[1], Elie Antoun[1], Yanchun Peng[1], Jennifer Frommer[3], Klaus-Peter Knobeloch[4], Roberto F. Speck[2], Benedikt M. Kessler[1], Tao Dong[1], Adán Pinto-Fernández[1]

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Cancer immunotherapy has transformed outcomes for some patients, yet durable responses remain limited by the immunosuppressive tumour microenvironment and the progressive exhaustion of tumour-infiltrating T cells. Type I interferon (IFN-I) signalling is a central driver of anti-tumour immunity, but sustained signalling also engages intrinsic negative-feedback brakes. Ubiquitin-specific peptidase 18 (USP18), the principal ISG15 deconjugase and a key negative regulator of IFN-I signalling, has been implicated in tumour immune evasion, yet its cell-intrinsic role in human T cells has remained poorly defined.

Across The Cancer Genome Atlas and single-cell datasets, USP18 was recurrently up-regulated in human tumours and marked interferon-rich, terminally exhausted antigen-specific CD8+ T cells. Genetic ablation of USP18 in Jurkat cells, primary human CD8+ T cells, and NY-ESO-1–specific CD8+ T cells consistently enhanced proximal T-cell-receptor signalling, increased CD69 expression and IFN-γ release, and promoted more sustained antigen-specific tumour-cell killing. Using a doxycycline-inducible rescue system, this suppression was shown to require an intact catalytic site: the catalytically inactive C64S mutant was markedly less able than wild-type USP18 to restrain T-cell activation. Immunoprecipitation–mass spectrometry identified an autophagy-related interactome preferentially associated with wild-type USP18, with ATG5 as a candidate partner, and ATG5 depletion attenuated USP18-mediated suppression. Finally, a selective covalent USP18 inhibitor raised proteome-derived T-cell activation and cytotoxicity scores and enhanced cytokine production and tumour-cell killing in activated PBMCs, while a NanoLuc reporter screen nominated a lead antisense oligonucleotide for USP18 silencing.

Collectively, these findings define USP18 as an active-site–dependent, cell-intrinsic brake on T-cell activation and cytotoxicity, acting through an ATG5-associated autophagy pathway, and establish it as a mechanistically defined target for enhancing anti-tumour T-cell responses.

Poster #11 –Samuel Martyn-Smith

Resolving spatial and temporal immune cell dynamics in response to differential αPD-1 & αCTLA4 immunotherapy combinations

Samuel Martyn-Smith[1], Charlotte Lettau[1], Stefanie Hasan[1], Paul Tarlatt[1], Pascale Wehr[1], Nabina Pun[1], Bethany Kennedy[1], Claire Willis[1], David Withers[1]

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Introduction: Immune checkpoint blockade (ICB) has redefined the therapeutic oncology landscape. Treatment with anti-PD-1 monotherapy is a well-established regimen, which is in part, driven by a subset of exhausted CD8+ T cells characterised by TCF^{+} $PD\text{-}1^{+}$ $TIM3^{-}$ expression. When administered in combination with anti-CTLA-4 therapy, enhanced tumour control has been observed, but at the cost of an increased frequency of serious immune-related adverse effects (IrAEs). This creates dosing challenges despite the clear advantage to some cancer patients Further research determining how best to combine targeting of PD-1 and CTLA-4, potentially alongside other immunotherapies, is needed to ensure more cancer patients can benefit from these breakthrough therapies.

Methods: To investigate how best to target PD-1 and CTLA-4, in vivo tumour models utilising both wildtype and photoconvertible Kaede mouse hosts have been utilised, the latter to enable consideration of immune cell dynamics in the response. Multiple tumour models were utilised to identify tumours with an appropriate responsiveness to treatment to support the study of combined targeting of PD-1 and CTLA-4. Tumour growth in vitro was carefully monitored and tissue analysed using flow cytometry.

Results: To interrogate tumour models appropriate for investigation of the response to combined ICB, colorectal (MC38) and melanoma (YUMM3.3) tumours have been characterised (MHCI/II expression) and tested in vivo with multiple administrations of anti-PD-1. Our data shows that anti-PD-1 treatment has a limited effect in MC38 tumour-bearing mice. A similarly modest effect was also seen when MC38 tumour-bearing mice are treated with a novel Receptor Inhibitor via Phosphatase Recruitment (RIPR).

Discussion: Given that anti-PD-1 monotherapy only precipitates a modest effect, immediate next steps include looking at combinatory therapy of anti-PD-1 and anti-CTLA4. Notwithstanding this, our initial finding suggests that MC38 may be a suboptimal model of ICB response, and warrant investigation into other tumour models. Therefore, ongoing work investigates how responsive the YUMM3.3 melanoma cell line is to anti-PD-1 treatment.

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Poster Abstracts

Poster #12 – Asja Puncuh

Design of HLA-E-targeted adoptive cell therapies for acute myeloid leukaemia (AML)

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Acute myeloid leukaemia (AML) is a haematological malignancy with a poor prognosis despite therapeutic advances, highlighting the need for new treatment strategies. T cells engineered with canonical T cell receptors (TCRs) have shown promise in AML and solid cancers; however, their clinical utility is limited by restriction to highly polymorphic classical human leukocyte antigen (HLA) class I and II molecules.

The non-classical HLA class I molecule HLA-E is dimorphic and may therefore represent a more universal therapeutic target. While its primary function is monitoring the integrity of antigen presentation, HLA-E can also present self- and pathogen-derived peptides to CD8+ T cells. In addition, HLA-E is frequently upregulated in cancer, whereas classical HLA class I/II expression may be lost, potentially reducing immune evasion when targeted by TCRs or chimeric antigen receptors (CARs).

Candidate HLA-E-binding peptides were identified from tumour-associated antigens (TAAs) using NetMHC 4.0/4.1pan and an in-house prediction algorithm. Binding was evaluated by nano differential scanning fluorimetry and enzyme-linked immunosorbent assay, identifying seven candidate peptides from six TAAs. Peptides derived from cyclin A1 (CCNA1), preferentially expressed antigen in melanoma (PRAME), and telomerase reverse transcriptase (TERT) showed the strongest HLA-E binding.

The strongest binder, a CCNA1-derived peptide, formed a stable complex with HLA-E in a blue native gel shift assay and increased HLA-E surface expression on β2-microglobulin (β2M)-knockout 293T cells when incorporated into a single-chain trimer (peptide–β2M–HLA-E heavy chain). Cytosolic expression of the CCNA1-derived peptide also increased HLA-E surface expression, demonstrating endogenous presentation by HLA-E.

Ongoing studies are priming T cells with CCNA1-, PRAME-, and TERT-derived peptides to generate HLA-E-restricted responses and assess their cytotoxic activity against AML in vitro and in vivo. These studies will determine the potential of HLA-E-targeted immunotherapy as a universal treatment strategy for AML.

Poster #15 –Aleksandra Dzhoneva

Proteogenomic Discovery of Unannotated Reading-Frame-Derived HLA-I Antigens in Colorectal Cancer

Aleksandra Dzhoneva[1,2], Qian Yang[1,2], Annalisa Nicrastri[1,3], Robert Parker[1,4], Iliyana Kaneva[1], Nicola Ternette[1,5], COMBATcancer Consortium[2], Timothy Elliott[1,2], Eleni Adamopoulou[1,2]

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Colorectal cancer (CRC) remains a leading cause of cancer mortality worldwide. Recent advances in immunotherapy have mainly shown benefit in the typically highly mutated microsatellite-instable (MSI) disease subtype, whereas microsatellite-stable (MSS) disease has shown poor responsiveness. Most antigen-discovery studies in CRC have examined peptides derived from annotated protein-coding genes, potentially overlooking tumour antigens arising from unannotated reading frames, including alternative ORFs, upstream ORFs, long non-coding RNAs and other cryptically translated transcripts. These non-canonical sources could expand the actionable immunopeptidome and reveal novel antigens in both MSS and MSI CRC.

We applied an integrated proteogenomic strategy combining immuno-affinity purification and LC-MS/MS acquisition of HLA class I peptides (nanoElute 2-timsTOF SCP, Bruker Daltonics) with RNA sequencing of matched tumour material from patients with MSS and MSI CRC. RNA-informed custom translation databases spanning canonical and alternative reading frames enabled identification of non-canonical peptide candidates, which were further prioritised by spectral confidence (PEAKS v13 and FragPipe v23.1), HLA-binding compatibility (NetMHCpan-4.2), transcript expression, tumour specificity, recurrence across tumour macro-regions and patients, and absence from matched non-malignant colon tissue. This workflow identified tumour-enriched non-canonical peptides linked to expressed transcripts and enabled comparison across MSS and MSI disease. PRIME2.0 further prioritised candidates with predicted T-cell recognition potential, supporting unannotated reading frames as an expanded source of actionable CRC antigens.

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Poster Abstracts

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Poster #16 – Anna Broomfield

IL7 Enhances ImmTAC-mediated Killing by T cells in Vitro and Improves T Cell Fitness in Patients

Anna Broomfield, Melanie Desbois, Des Jones, Laura Collins, Donghoon Choi, Peter Kirk, Adel Benlahrech, Koustubh Ranade, Joseph Sacco

IMMUNOCORE

Background: A blood T cell fitness (TCF) signature, reflecting properties of naïve and stem cell memory T cells, was strongly associated with clinical benefit from tebentafusp (gp100 × CD3) and brenetafusp (PRAME × CD3) ImmTAC bispecific therapies[1]. Here, we assessed whether TCF could be enhanced by IL7, a cytokine that plays a key role in T cell homeostasis and promotes the proliferation and survival of naïve and stem cell memory T cells.

Methods: T cell exhaustion was induced in vitro by four weekly ImmTAC stimulations against the Non-Small Cell Lung Cancer (NSCLC) cell line NCIH1755 with or without IL7. Tumor killing, T cell cytokine secretion, and T cell phenotype were assessed using standard methods. Data are given as mean ± SEM; groups were compared using paired t test. IL7R gene expression and TCF (mean expression of TESPA1, CD28 and GPR183) were measured in baseline whole blood from patients with unresectable or metastatic uveal melanoma (mUM) treated with tebentafusp (n=132 NCT02570308) or brenetafusp (N=37 NCT04262466) as previously described1. TCF high/low threshold was cut at the median. TCF was also assessed in baseline and on-treatment PBMC from patients with NSCLC and Triple-Negative Breast Cancer (TNBC) (n=15) receiving NTI7, a long acting recombinant human IL7 (rhIL7, NCT03752723, NCT04984811).

Results: In vitro, repeated re-direction of T cells by ImmTAC resulted in reduced tumor cell lysis from 46±9% after a single stimulation to 3±1% after 4 weekly stimulations. In contrast, T cells cultured with IL7 retained tumor cell lysis after 4 stimulations (58±2% ImmTAC-redirected cytolysis compared with 3±1% in IL7-untreated T cells, $p = 0.02$), accompanied by a 14.3-fold increase in IFNγ secretion ($p = 0.04$). The proportion of naïve/stem cell memory T cells increased in response to IL7 treatment from 37±5% to 57±8% ($p=0.009$). In mUM patients, gene expression of IL7R strongly correlated with TCF signature in peripheral blood ($R = 0.91$, $p < 0.001$). As early as 3 weeks following a single dose of NTI7, TCF increased by ~3-fold in PBMC from NSCLC and TNBC patients. The proportion of TCF high patients increased from 36% at baseline to 93% post NTI7 monotherapy. This increase in TCF signature was sustained for at least 12 weeks.

Conclusion: Despite repeated antigen stimulation in vitro, IL7 sustained naïve/memory T cells, enhanced ImmTAC-redirected T cell cytotoxicity and IFNγ production, and reduced T cell exhaustion. A single dose of rhIL7 resulted in a sustained increase in TCF signature and converted patients with low TCF signature into high TCF. These findings support combination with IL7 as a rational strategy to augment T cell fitness and potentially improve efficacy of ImmTAC bispecific T cell therapies.

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OCION Phoenix Pump-Prime

1

1. Abstract

300-word abstract
Original idea

Potential collaborators / gaps

2

2. Pitch

3 min pitch

Open session at the **OCION symposium**

Invited PIs

3

3. Rebuild

1:1s meetings

PPI involvement

Strengthen concept

Add collaborators

4

4. Phoenix panel

15 min final presentation
To the Steering Committee

Collaborators present

Novelty + feasibility

Grant readiness

If you are interested to discuss/transform the project/collaborate with any of the applicants please follow the QR code or submit your interest here

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