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# Annual Symposium

Tuesday 31st March 2026

Mathematical Institute, University of Oxford

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# About Oxford Cancer and the CRUK Oxford Centre



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**Oxford Cancer** is a city-wide network and partnership between Oxford University and Oxford University Hospitals NHS Trust, based on the University's Translational Biomedical Research Campus.

It currently comprises over 10,000 members from around 90 different Departments, Units, and Institutes of the University, as well as the NHS Trust, which invests over £55m each year into translational cancer research.

Oxford Cancer is an inclusive network of organisations in Oxford for whom cancer research is a priority focus. We support and connect people working across a range of disciplines and aim to facilitate research collaboration on a local, national and international scale to speed up translation from scientific discovery to treatments in patients.

A large, thick yellow arch graphic that spans across the bottom half of the page, partially obscuring the background image of Oxford's skyline.

**The CRUK Oxford Centre harnesses Oxford's world-leading cancer research with the core aim of facilitating collaboration to ensure rapid translation from scientific discovery to treatments for patients. The Centre is run as part of the Oxford Cancer network, with a focus on funding Cancer Research UK strategic priorities.**



# Programme



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|                                                              |  |             |                                                                                                                                                                |                                                                                                                                             |
|--------------------------------------------------------------|--|-------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------------------------------------------------------|
|                                                              |  | 8.30        | Registration open                                                                                                                                              |                                                                                                                                             |
| Session 1, Part 1:<br>New to Oxford<br>Chair: Tim Elliott    |  | 9.15-9.30   | <b>Welcome</b>                                                                                                                                                 | <b>Tim Elliott</b><br>Co-Director, Oxford Cancer                                                                                            |
|                                                              |  | 9.30-9.55   | Neurodevelopmental and injury programs shape glioblastoma and therapy response                                                                                 | <b>Lucy Brooks</b><br>Department of Oncology                                                                                                |
|                                                              |  | 9.55-10.20  | New bioengineering approaches for cancer detection and treatment                                                                                               | <b>Molly Stevens</b><br>Department of Engineering                                                                                           |
|                                                              |  | 10.20-10.45 | An RNA-dependent positive feedback loop drives malignant ribosome biogenesis and tumourigenesis downstream of RAS oncogene                                     | <b>Faraz Mardakheh</b><br>Department of Biochemistry                                                                                        |
|                                                              |  | 10.45-11.30 | <b>Coffee break and poster viewing (odd numbers), information stalls</b>                                                                                       |                                                                                                                                             |
| Session 1, Part 2:<br>New to Oxford<br>Chair: Eileen Parkes  |  | 11.30-11.55 | Understanding how myeloid cells adapt to the tumour microenvironment in real time                                                                              | <b>David Withers</b><br>Nuffield Department of Medicine                                                                                     |
|                                                              |  | 11.55-12.20 | Death and taxes: The hazards of smoking and benefits of cessation and taxing tobacco                                                                           | <b>Prabhat Jha</b><br>Nuffield Department of Population Health                                                                              |
|                                                              |  | 12.20-12.45 | Early detection and cancer evolution: From single-cell models to cohort-scale science                                                                          | <b>Philip Awadalla</b><br>Nuffield Department of Population Health                                                                          |
|                                                              |  | 12.45-13.45 | <b>Lunch, poster viewing (even numbers), information stalls</b>                                                                                                |                                                                                                                                             |
|                                                              |  | 13.00-13.15 | <b>Cancer Research Horizons information session (lecture theatre)</b>                                                                                          |                                                                                                                                             |
| Session 2:<br>Keynote<br>Chair: Mark Middleton               |  | 13.45-14.35 | Neoadjuvant therapy in melanoma – the journey from concept to approved treatment                                                                               | <b>Christian Blank</b><br>Netherlands Cancer Institute                                                                                      |
| Session 3:<br>PPI & EDI<br>Chair: Catriona Gilmour Hamilton  |  | 14.35-15.05 | Inclusive research design and PPI: What does it all mean?                                                                                                      | <b>Toral Gathani</b><br>Cancer Epidemiology Unit<br>Nuffield Department of Population Health<br><br><b>Julliet Lwiindi</b><br>Oxford Cancer |
|                                                              |  | 15.05-15.55 | <b>Coffee break and poster viewing (all), information stalls</b>                                                                                               |                                                                                                                                             |
| Session 4:<br>Flash talks<br>Chair: Isabella Pedrosa-Pacheco |  | 15.55-16.00 | Machine perfusion and spatial transcriptome mapping identify intra-tumoural endothelial anergy as an immune escape mechanism in colorectal liver metastasis    | <b>Peter Wan</b><br>Department of Oncology                                                                                                  |
|                                                              |  | 16.00-16.05 | Tubal ligation and incidence of histological types of uterine and ovarian cancers: Evidence for different origins of serous carcinomas of the ovary and uterus | <b>Kezia Gaitskell</b><br>Nuffield Department of Population Health                                                                          |
|                                                              |  | 16.05-16.10 | Oncogenic KBTBD4 mutations and the stem cell agonist UM171 converge on E3 ligase rewiring to target the LSD1-CoREST-HDAC1/2 complexes                          | <b>Zhuoyao Chen</b><br>Centre for Medicines Discovery                                                                                       |

# Programme



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Session 5:  
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announcements

|              |                                                                                                                                                           |                                                                          |
|--------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------|
| 16.10-16.15  | Enhancing solid tumor immunotherapy by combining spatially fractionated FLASH radiotherapy with mRNA-LNP-based bispecific antibody therapy                | <b>Jia-Ling Ruan</b><br>Department of Oncology                           |
| 16.15-16.20  | BRN2-ATF4 coupling rewires the integrated stress response to protect invasive melanoma from apoptosis                                                     | <b>Pakavarin Louphrasitthiphol</b><br>Ludwig Institute of Medicine       |
| 16.20-16.25  | Preoperative weight loss in patients with excess weight and colorectal cancer: The CARE feasibility randomised clinical trial                             | <b>Dimitrios Koutoukidis</b><br>Nuffield Department of Population Health |
| 16.25-16.30  | Surface nucleolin regulates $\gamma$ -irradiation-induced DNA damage repair in breast cancer cells                                                        | <b>Dongxiao Yang</b><br>Department of Oncology                           |
| 16.30-16.35  | Watching the immune response unfold: How pembrolizumab treatment reshapes the tumour-reactive T cell repertoire in triple-negative breast cancer patients | <b>Mariana Pereira Pinho</b><br>CAMS Oxford Institute                    |
| 16.35-16.45  | <b>Closing remarks</b>                                                                                                                                    | <b>Mark Middleton</b><br>Co-Director, Oxford Cancer                      |
| 16.45 -18.00 | <b>Networking reception</b>                                                                                                                               |                                                                          |



## Session 1: New to Oxford



**Dr Lucy Brooks**

**MSci PhD**

Group Leader of the  
Brooks Group in the  
Department of Oncology

### 'Neurodevelopmental and injury programs shape glioblastoma and therapy response'

Glioblastoma engages neurodevelopmental and injury-response programs that shape tumour organisation and behaviour. This talk will examine how tumour cell states emerge, how they are regulated by the microenvironment, and how these dynamics intersect with therapeutic response.

Dr Lucy Brooks received her Ph.D. from Imperial College London. She conducted her postdoctoral work with Prof. Simona Parrinello at the UCL Cancer Institute, first as a postdoc then a CRUK RadNet Senior Research Fellow. Lucy now leads the brain tumour plasticity and resistance lab at the University of Oxford studying how cellular plasticity and intratumoral heterogeneity contribute to therapy resistance in brain cancer.



**Professor Dame Molly Stevens**  
**FEng FRS**

John Black Professor of  
Bionanoscience at the  
Department of  
Physiology, Anatomy &  
Genetics

### 'New bioengineering approaches for cancer detection and treatment'

Professor Dame Molly Stevens FRS FEng FMedSci is John Black Professor of Bionanoscience at the University of Oxford (Department of Physiology, Anatomy and Genetics and Institute of Biomedical Engineering) and part-time Professor at Imperial College London and the Karolinska Institute.

Professor Stevens' multidisciplinary research balances the investigation of fundamental science with the development of technologies and designer biomaterials to address some of the major healthcare challenges across diagnostics, advanced therapeutics and regenerative medicine. The impact of her work is recognised by numerous accolades, including the 2023 Novonordisk Prize.

Professor Stevens' work has a strong translational drive, focussed on translating scientific innovation into practical solutions to benefit patients and society. She is a serial entrepreneur and the founder of five spin-off companies. She is also the Oxford University Champion for Women and Diversity in Entrepreneurships.

## 'An RNA-dependent positive feedback loop drives malignant ribosome biogenesis and tumourigenesis downstream of RAS oncogene'



**Associate Professor  
Faraz Mardakheh  
PhD FHEA**

Principal Investigator of  
the Mardakheh Lab,  
Department of  
Biochemistry

Alterations to the nucleolar morphology and function are well-established hallmarks of cancer, yet their molecular underpinnings remain poorly understood. Using a RAS-inducible mouse model of pancreatic ductal adenocarcinoma (PDAC), we performed a spatially resolved proteomic and phospho-proteomic profiling of the nucleolus, following oncogene activation.

Our analysis identified a RAS-driven phosphorylation programme, orchestrated through relocation of Casein Kinase 2 (CK2) holoenzyme to the nucleolus. This programme predominantly targets rDNA transcription and early rRNA processing factors, resulting in hyperactivation of rRNA synthesis and ribosome biogenesis.

Disrupting the nucleolar localisation of CK2 blocks RAS-induced rRNA synthesis, while constitutive trapping of CK2 in the nucleolus is sufficient to activate rRNA synthesis independent of RAS. Mechanistically, nucleolar CK2 accumulation is mediated through its direct binding to the pre-rRNA, creating a positive feedback loop that acts to amplify rRNA synthesis. Examination of tissues from diverse human cancers revealed nucleolar CK2 accumulation as a broadly conserved cancer-associated feature. Importantly, disrupting the RAS-CK2-mediated feedback loop selectively inhibits the anchorage-independent growth of RAS-expressing cells, and blocks PDAC tumourigenesis.

Our findings reveal an RNA-dependent positive feedback loop involving CK2 that is a critical driver of malignant ribosome biogenesis, and highlights a specific cancer vulnerability that could be exploited therapeutically.

Associate Professor Faraz Mardakheh received his undergraduate degree (BSc Hons) in 2006 from the University of Birmingham, before joining the laboratory of Professor John Heath to study feedback regulation of Receptor Tyrosine Kinase signalling, and its role in cancer as a tumour suppressive mechanism.

After completing his PhD, Faraz moved to London in 2010 to study cancer cell migration and invasion as a postdoctoral researcher in the laboratory of the late Professor Chris Marshall. Thanks to an unexpected discovery during his post-doc, he started working on RNA Binding Proteins (RBPs) and their role in cell migration and cancer invasion. Subsequently, in 2017 Faraz received a Medical Research Council (MRC) Career Development Award fellowship to start his own group at the Barts Cancer Institute, Queen Mary University of London, and study RBPs in the context of cancer.

In 2024, Faraz's group moved to the Department of Biochemistry at the University of Oxford, where they continue their research into RBPs and their role in cancer. By using various state-of-the-art multi-omics approaches, their aim is to identify and characterise cancer-relevant RBPs whose activities could be therapeutically targeted for combatting cancer.

## ‘Understanding how myeloid cells adapt to the tumour microenvironment in real time’



**Professor David Withers PhD**

Group Leader in  
Experimental Cancer  
Immunology, Nuffield  
Department of Medicine

Cancers not only evade but actively corrupt the immune response tasked with eliminating them. Solid tumours deploy a range of mechanisms that alter the function, behaviour, and fate of cytotoxic effector lymphocytes, including CD8+ T cells and NK cells, thereby blunting their ability to kill cancer cells. Although immune checkpoint blockade has demonstrated the therapeutic potential of restoring intratumoural effector activity, durable responses remain restricted to a subset of patients and tumour types. Broadening and sustaining clinical benefit will require therapeutic strategies that target additional suppressive pathways alongside enhancement of T cell function. However, designing such rational combination therapies demands a detailed understanding of how the tumour and the local tissue environment reprogramme cells across the immune landscape.

Our research aims to define precisely how immune cells are reshaped within tumours, when these changes occur, and how they influence anti-tumour immunity. Using tumour implantation in photoconvertible mice, we have developed novel labelling strategies that distinguish newly infiltrating cells from those retained in the tissue. This approach enables direct tracking of immune cell fate, quantification of population turnover, and real-time analysis of how immune subsets adapt to the tumour microenvironment.

Here I will discuss our new data interrogating the emergence and regulation of immuno-supportive and immuno-suppressive myeloid populations within in vivo cancer models. By resolving the kinetics and regulatory mechanisms that govern myeloid cell adaptation within tumours, our findings provide a framework for rational therapeutic design aimed at reprogramming the tumour microenvironment and enhancing durable anti-tumour immunity.

Professor David Withers studied for a PhD in Immunology at the Institute for Animal Health/University of Bristol (2000–2004), then post-doctoral studies first at the NIH, Bethesda, USA (NIAMS), then at the University of Birmingham, UK. In 2011, David was awarded a Wellcome Trust Research Career Development Fellowship investigating the role of innate lymphoid cells in regulating memory T cell responses and then a Wellcome Trust Senior Research Fellowship in 2016. Most recently, his research has developed to consider the regulation of anti-tumour immunity, exploiting novel in vivo models and supported by Cancer Research UK, the Cancer Research Institute, Worldwide Cancer Research and the MRC.

In June 2024, David moved his lab to the NDM Centre for Immuno-Oncology at the University of Oxford. As Professor of Experimental Cancer Immunology, he leads the Tumour-Immune Cell Dynamics Group, which is focused upon understanding how immune cells behave within, and respond to, the tumour microenvironment and then what this means for enhancing anti-tumour T cell responses. The lab is currently supported by a Wellcome Discovery Award and Cancer Research UK Programme grant.



## 'Death and taxes: the hazards of smoking and benefits of cessation and taxing tobacco'



**Professor Prabhat Jha**  
**OC MD DPhil**

Head of Department and  
Nuffield Professor of  
Population Health,  
Department of  
Population Health

Professor Prabhat Jha studied Medicine at the University of Manitoba and was awarded a DPhil in Epidemiology and Public Health (as a Rhodes Scholar) in the Cancer Epidemiology Unit where he was supervised by the late Professor Dame Valerie Beral.

He was the University Professor of Global Health and Epidemiology at the Dalla Lana School of Public Health and Temerty School of Medicine, University of Toronto, and Director of the Centre for Global Health Research. He has served on a number of advisory boards and has been a health adviser to several governments.

Prabhat's research focuses on nationwide epidemiological studies of the major causes of death in developing countries (including tobacco, alcohol, and cardiometabolic risk factors), and epidemiological research methods in low-resource settings, including randomised intervention studies. He is particularly interested in the use of routine mortality data for analytic epidemiology, and tobacco control policy in developed and developing countries. His research enabled a worldwide treaty on tobacco control signed now by over 180 countries. He has quantified malaria mortality in children and adults in various settings, leading to vaccine trials.

Prabhat was appointed Officer of the Order of Canada in 2012 for his contributions to global health. He has received a number of other awards, including the 2022 Award of Excellence from the American Public Health Association and the 2012 American Cancer Society's Luther L Terry Award for Outstanding Research Contribution for his globally influential research on tobacco control. He is a Fellow of the Royal Society of Canada and the US National Academy of Medicine.

## ‘Early detection and cancer evolution: from single-cell models to cohort scale science’



**Professor Philip  
Awadalla PhD**

Professor of Molecular  
Genetics and Co-  
Director, Translational  
Epidemiology Unit

My research programme aims to define early biological changes and translate them into better strategies for cancer risk prediction, early detection, and intervention. In this talk, I will highlight how we integrate large population cohorts, blood-based molecular profiling, and single-cell tumour models to study cancer evolution before diagnosis.

I will first discuss our work on clonal haematopoiesis and related forms of somatic mosaicism in blood, including autosomal mosaic chromosomal alterations. These studies show how age, sex, ancestry, and geography shape somatic clonal architecture, providing insight into early cancer-related evolution in otherwise healthy individuals and identifying blood-based markers of future disease risk. I will then present our work on pre-diagnosis profiling, showing that blood-based molecular signals of cancer evolution can be detected years before conventional diagnosis. These findings support the development of interpretable biomarkers for early detection and suggest that population-based biofluids can reveal the earliest measurable stages of malignant transformation.

Finally, I will touch on our single-cell work, including a pan-cancer compendium of intratumoural heterogeneity and ongoing studies of aggressive breast cancer. These analyses are helping define tumour cell states, stem-like programmes, and tumour-microenvironment interactions that may underlie progression, immune evasion, and therapeutic resistance. Together, this work links somatic evolution in blood, preclinical detection, and tumour cell-state biology to identify new opportunities for earlier intervention and improved cancer outcomes.

Philip Awadalla is Professor of Molecular Genetics at the Big Data Institute and the NDPH, University of Oxford. He recently joined Oxford from Toronto, where he was a Senior Investigator at the Ontario Institute for Cancer Research, Director of Computational Biology. He continues to be the National Scientific Director of CanPath, Canada's largest longitudinal population health cohort. His research spans genomics, population health, somatic mutation, ageing, clonal haematopoiesis, and early cancer detection, combining large-scale population cohorts with molecular profiling and experimental models.

## Session 2: Keynote Address

### ‘Neoadjuvant therapy in melanoma – the journey from concept to approved treatment’



**Professor Christian Blank MD PhD MBA**

Professor of Cancer Immunotherapy at the Leiden University Medical School and Group Leader at the Netherlands Cancer Institute

Christian Blank obtained his MD from the Medical School of the Technical University Munich, Germany, where he also completed his Doctoral thesis (summa cum laude) at the Department for Medical Microbiology and in 1997. He worked as Junior House Officer (1997–1998) at the University Clinic Munich, at the Royal Infirmary of Edinburgh, and the University of Birmingham. He went on to attain the position of Physician at the Department of Hematology and Oncology, University of Regensburg, Germany (1998–2001). During 2001–2003, Dr Blank held a Postdoctoral Research Fellowship at the lab of Professor Thomas Gajewski, University of Chicago, IL, USA. Subsequently he was appointed as Physician and Research Group Leader at the Department of Hematology and Oncology, University of Regensburg, Germany (2003–2007).

Dr Blank has obtained two Specialist Degrees in Internal Medicine (2007) and in Hematology/Oncology (2009). Since 2007, he has been appointed Staff Member at the Department of Medical Oncology, and a Group Leader at the Division of Molecular Oncology and Immunology, The Netherlands Cancer Institute - Antoni van Leeuwenhoek Hospital (NKI-AVL), Amsterdam. In 2010, Dr Blank became University Lecturer (Privatdozent) at the University of Regensburg.

In addition to his academic background, Dr Blank obtained his Master of Business Administration (MBA), from the University of Warwick, England, UK (2006).

Dr Blank is committed to teaching, lecturing on medical courses at the NKI-AVL and the Medical School of the University of Regensburg, where he was appointed Professor in 2015. In 2020 he was additionally appointed Professor at the Leiden University Medical Center (LUMC) in The Netherlands. Since end 2025 he is also Program Director Melanoma and Sarcoma at the European Institute Oncology (IEO) in Milano.

He is a member of several prestigious societies, including ESMO and ASCO. Dr Blank has been an invited speaker at more than 250 national and international congresses, and is the author of more than 250 publications. His research interests broadly include neoadjuvant immunotherapies, targeted and biological response modifiers, and prognostic markers for cancer immunotherapies. Dr Blank has been the principal investigator of several investigator initiate trials testing neoadjuvant immunotherapy in melanoma that are currently changing the treatment landscape.



## Session 3: PPI & EDI

### 'Inclusive research design and PPI: what does it all mean?'



**Associate Professor  
Toral Gathani MD MEd  
FRCS (Eng)**

Associate Professor at  
the Cancer Epidemiology  
Unit in the Nuffield  
Department of  
Population Health, and  
Consultant Surgeon

Inclusive research design and public and patient involvement help ensure research reflects the needs and experiences of the people it aims to benefit. Involving patients and the public throughout the research process improves relevance, fairness, and trust. It also helps researchers reach more diverse groups, reduce inequalities, and produce findings that are easier to use in real life. Working in partnership with communities means research is done with people rather than to them, leading to better quality evidence and greater impact on practice and decision-making.

Toral Gathani is an Associate Professor in the Cancer Epidemiology Unit in the Nuffield Department of Population Health, University of Oxford and Consultant Breast Surgeon at Oxford University Hospitals NHS Foundation Trust.

Her main research interests are in the intersection of ethnicity and breast cancer, the role of health systems in early cancer detection and diagnosis, and of breast cancer as a global health challenge.

She is the Principal Investigator for a programme of work on Ethnicity and Breast Cancer and was awarded the Hunterian prize by the Royal College of Surgeons of England in 2022 for her work in this area. She is the Chief Investigator of AgeX which is assessing the benefits and harms of extending the age range for

routine screening invitations, and chairs the Research, Innovation and Development Advisory Committee for the NHS Breast Screening Programme in England.

Toral has spent time working in India in a research and clinical capacity and is on the steering committee of the Kenya UK Breast Cancer Alliance.

Julliet Lwiindi is Oxford Cancer's Public Engagement and Involvement Officer, and is responsible for implementing the Oxford Cancer patient and public involvement strategy. She is interested in how organisations can develop long term strategies and processes to support, monitor and report on public involvement in research.



**Ms Julliet Lwiindi**  
Public Engagement and  
Involvement Officer at  
Oxford Cancer

## Session 4: Flash Talks

### Peter Kok-Ting Wan

- Title:** Machine perfusion and spatial transcriptome mapping identify intra-tumoural endothelial anergy as an immune escape mechanism in colorectal liver metastasis
- Author:** Peter Kok-Ting Wan, Pancreatic Cancer UK Career Foundation Fellow  
([peter.wan@oncology.ox.ac.uk](mailto:peter.wan@oncology.ox.ac.uk))
- Affiliation:** Department of Oncology, University of Oxford
- Background:** Colorectal liver metastasis (CRLM) is the terminal stage of colorectal cancer and remains largely resistant to immunotherapies, including adoptive T-cell therapy, yet the underlying immune escape mechanisms remain poorly understood. We aim to reveal spatial and molecular mechanisms underlying immune exclusion in CRLM and the surrounding liver microenvironment.
- Design:** First, we re-purposed normothermic hepatic machine perfusion as a preclinical model of CRLM, enabling delivery of labelled T-cells through physiological vascular routes into living tumour-bearing human livers. This allowed direct and simultaneous comparison of T-cell extravasation through hepatic and CRLM vasculature. Second, we spatially mapped the transcriptomes of CRLM and adjacent liver at single-cell resolution across ~1.9 million cells to dissect the endothelial biology in the liver microenvironment.
- Results:** Whilst T-cells readily extravasated within the peri-tumoural liver, they failed to extravasate into CRLM from the perfused tumour vasculature. Spatial analyses revealed a progressive shift in endothelial phenotypes across regions: from a non-inflamed state in normal liver, to a highly inflamed peri-tumoural endothelium characterised by TNF $\alpha$ , NF $\kappa$ B, STING and inflammasome signature enrichment, and finally to an anergic, angiogenic endothelial population within CRLM. This anergic endothelial state was defined by reduced expression of adhesion molecules required for T cell tethering and extravasation, including SELE, SELP, ICAM1 and VCAM1, in contrast to the more immune-permissive peri-tumoral endothelium.
- Conclusion:** We characterised changes in endothelial phenotype across different compartments of the metastatic liver microenvironment, and identified impaired T-cell extravasation as a potential mechanism of immune exclusion. These findings provided novel targets to sensitise CRLM to immunotherapy and a potential explanation for the limited efficacy of adoptive T-cell therapy in CRLM.

## Kezia Gaitskell

- Title:** Tubal ligation and incidence of histological types of uterine and ovarian cancers: Evidence for different origins of serous carcinomas of the ovary and uterus
- Authors:** Kezia Gaitskell, Sarah Floud, Gillian K Reeves; on behalf of the Million Women Study Collaborators
- Affiliation:** Cancer Epidemiology Unit, Nuffield Department of Population Health, University of Oxford
- Background:** Cancers of the ovary and uterus have analogous histological types, showing similarities in microscopic morphology and molecular characteristics. It has been hypothesised that some of these may have shared origins across the two cancer sites. For example, there is growing evidence that the majority of high-grade serous carcinomas found in the ovary in fact arise in the fimbrial end of the fallopian tubes, and it has been suggested that this may also be the origin of serous carcinomas found in the uterus. If these hypotheses of shared origins are true, we might expect similar patterns of risk factors between these analogous types. However, few epidemiological studies are large enough to explore this reliably. We investigated the association between tubal ligation (sterilization) and risk of analogous histological types of ovarian and uterine cancer in a large prospective cohort of UK women.
- Methods:** Participants in the Million Women Study completed a questionnaire on reproductive and lifestyle factors in 1996–2001, and were followed for cancer and death via national registries. Using Cox regression models, we estimated relative risks (RRs) of specific histological types of ovarian and uterine cancer, adjusted for potential confounders. Ovarian cancers were defined to include cancers of the fallopian tube and peritoneum, in line with clinical practice; borderline tumours were excluded.
- Results:** The cohort included 958,030 women without previous cancer, hysterectomy, or bilateral oophorectomy. After a mean follow-up time of 20.18 years (SD 6.47), 17,612 cancers of the uterus, and 11,325 ovarian cancers, had accrued. Overall, women with a history of tubal ligation had a 22% reduction in the relative risk of ovarian cancer (RR=0.78, 95% CI: 0.74–0.83,  $p<0.001$ ), and a 5% reduction in the risk of uterine cancer (RR=0.95, 95% CI: 0.91–0.99,  $p=0.01$ ). Previous tubal ligation was associated with a 14% reduction in the risk of ovarian serous carcinomas (RR=0.86, 95% CI 0.80–0.93), but there was no evidence of a significant association with uterine serous carcinomas (RR=1.01, 95% CI: 0.89–1.16); these associations were statistically different,  $p$ -heterogeneity=0.04.
- Conclusions:** We found evidence of different associations between tubal ligation and risk of serous carcinomas in the ovary (reduced risk) compared to serous carcinomas in the uterus (no association). This is not supportive of the hypothesis that uterine serous carcinomas have a similar tubal origin to high-grade serous ovarian carcinomas. While the analogous histological types have morphological and molecular similarities, they may have very different origins. However, this analysis requires replication in other cohorts, and triangulation with other sources of evidence regarding the origins of cancer subtypes.



## Zhuoyao Chen

- Title:** Oncogenic KBTBD4 mutations and the stem cell agonist UM171 converge on E3 ligase rewiring to target the LSD1-CoREST-HDAC1/2 complexes
- Author:** Zhuoyao Chen, Senior Scientist (zhuoyao.chen@cmd.ox.ac.uk)
- Affiliation:** Centre for Medicines Discovery, Nuffield Department of Medicine, University of Oxford
- Background:** Recurrent in-frame insertions in the E3 ubiquitin ligase KBTBD4 have been reported as putative driver mutations in groups 3/4 medulloblastomas as well as in pineal parenchymal tumours with intermediate differentiation. Our previous study showed that these cancer hotspot mutations elicit neomorphic ubiquitination that promote the degradation of the LSD1-CoREST-HDAC1/2 transcriptional repressor complex. The resulting epigenetic reprogramming enhances stemness in medulloblastoma cells, establishing a novel paradigm of tumorigenesis driven by neomorphic E3 ligase mutations. Remarkably, UM171, a clinical agent used to expand haematopoietic stem cells for transplantation, also induces degradation of the LSD1-CoREST-HDAC1/2 complex in a wild-type KBTBD4-dependent manner, suggesting a shared mechanistic basis. Here, we elucidate the convergent structural mechanisms underlying oncogenic KBTBD4 mutations and UM171 action, with implications for both cancer biology and stem cell therapeutics.
- Method:** We performed cellular and biochemical assays to identify the direct interactors mediating recruitment of LSD1-CoREST-HDAC1/2 to KBTBD4. Biophysical assays characterised the KBTBD4-neosubstrate interactions in the presence of cancer-associated mutations or UM171. Cryo-EM was used to elucidate the structural mechanisms of how the mutations or the drug remodel the substrate-binding surface of KBTBD4. Lastly, a cellular degradation assay was performed for structure-activity relationships (SAR) analysis of UM171 derivatives.
- Results:** We identified that the neomorphic interactions between KBTBD4 and LSD1-CoREST-HDAC1/2 are mediated by the HDAC deacetylase domain. Cryo-EM studies of both wild-type and mutant KBTBD4 captured KBTBD4-HDAC2 as well as KBTBD4-HDAC2-CoREST1 complexes at resolutions spanning 2.7 Å to 3.1 Å. The mutant and drug-induced complexes adopted similar structural assemblies requiring both Kelch domains in the KBTBD4 dimer for each HDAC2 interaction. UM171 was identified as a bona fide molecular glue binding across the ternary interface with KBTBD4 and HDAC2. Most strikingly, the indel mutation reshaped the same surface of KBTBD4 providing the first example of a natural mimic of a molecular glue. Lastly, structural insights rationalize SAR findings and identify S234984 as a more potent UM171 analog.
- Conclusions:** The oncogenic mutations in KBTBD4 reshape the surface substrate pocket to form a direct binding interface for the HDAC1/2 component of LSD1-CoREST-HDAC1/2 complexes. UM171, a clinically-tested stem cell expansion drug, similarly hijacks wild-type E3 ligase KBTBD4 as a molecular glue for HDAC1/2 recruitment through unexpected structural mimicry of the drug and cancer mutation. This structural mimicry is a most unexpected finding and provides an example of a convergent mechanism for enhanced stemness in brain cancer and haemopoietic stem cells.

## Jia-Ling Ruan

- Title:** Enhancing Solid Tumor Immunotherapy by Combining Spatially Fractionated FLASH Radiotherapy with mRNA-LNP–Based Bispecific Antibody Therapy
- Authors:** Jia-Ling Ruan, May Sin Ke, Kaia Cochran, Carl Lee, Iain DC Tullis, Edward Taylor, Kristoffer Petersson, Walter Bodmer
- Affiliation:** Department of Oncology, University of Oxford
- Background:** Immunotherapy can produce durable responses with reduced systemic toxicity; however, its efficacy in solid tumours is often limited by poor immune infiltration and immunosuppressive mechanisms, including PD-L1–mediated T-cell inhibition and loss of major histocompatibility complex (MHC) expression. To address these barriers, we developed a combinatorial strategy integrating Spatially Fractionated FLASH Radiotherapy (SFRT-FLASH) with mRNA-lipid nanoparticle (mRNA-LNP) delivery of an EpCAM-CD3-Fc bispecific antibody (BiMab).
- SFRT-FLASH delivers ultra-high dose-rate radiation in a spatially fractionated pattern, inducing intense local tumour damage while sparing surrounding tissue. This approach is hypothesised to enhance inflammation and promote immune cell infiltration. The EpCAM-CD3-Fc BiMab redirects T cells to EpCAM-expressing tumour cells (>90% of epithelial cancers) and activates cytotoxicity independently of MHC presentation, overcoming tumour immune evasion.
- Method:** We optimised the combination regimen by determining radiation doses compatible with efficient mRNA translation, identifying optimal timing for mRNA-LNP administration, and assessing post-irradiation BiMab expression and T-cell activation. Treatment efficacy was evaluated in vitro in EpCAM-positive tumour models using cytotoxicity and viability assays.
- Results:** Radiation doses were identified that preserved mRNA translation and enabled robust BiMab expression following SFRT-FLASH. Optimised scheduling enhanced T-cell activation. In vitro co-culture models incorporating human and murine tumour cells with PBMCs show significantly enhanced tumour cell killing following combination treatment relative to monotherapy, consistent with synergistic anti-tumour effects.
- Conclusions:** The combination of SFRT-FLASH and mRNA-LNP–delivered EpCAM-CD3-Fc BiMab represents a novel strategy to enhance immune infiltration and overcome immune evasion in solid tumours. This platform may broaden the applicability of T-cell– redirecting therapies while maintaining favourable toxicity profiles. Ongoing studies aim to validate efficacy and immune modulation in vivo to support clinical translation.

## Pakavarin Louphrasitthiphol

- Title:** BRN2–ATF4 coupling rewires the integrated stress response to protect invasive melanoma from apoptosis
- Author:** Pakavarin Louphrasitthiphol (1,2), Sarafina Otis (1), Fieke Verhaff (1), Laura Mosteo (1) and Colin R Goding (1)
- Affiliations:** (1) Ludwig Institute for Cancer Research, Nuffield Department of Clinical Medicine, University of Oxford; (2) Faculty of Medicine, University of Tsukuba, Ibaraki, Japan
- Background:** Circulating tumour cells (CTCs) experience multitude of stresses including oxidative, ER and detachment (anoikis) stress during transit. In melanoma, the proliferative-to-invasive phenotype switch—marked by reduced MITF and increased BRN2—promotes dissemination, yet how invasive CTCs avoid stress-triggered death remains unclear. The integrated stress response (ISR) integrates diverse stress cues through activation of ATF4 to orchestrate expression of stress mitigating genes. Sustained ISR, however result in ATF4 mediate activation and dimerization with its pro-apoptotic partner CHOP to induce regulated cell-death gene expression programmes.
- Method:** We hypothesised that BRN2 buffers ISR output to maintain stress signalling below the apoptotic threshold in invasive melanoma cells. We combined transcriptomics, ChIP-seq, protein–protein interaction mapping and genetic perturbation to define functional crosstalk between BRN2 and ATF4.
- Results:** We identify physical and functional BRN2–ATF4 interaction. Genetic disruption of BRN2 promotes ATF4/CHOP-dependent pro-apoptotic gene programme and selectively sensitises BRN2-high to stresses. Notably, BRN2–ATF4 interaction may also confer non-genetic therapies resistance as many targeted therapies (including MAPK inhibitors), radiotherapy and many chemotherapeutic agents induce ISR.
- Conclusion:** Together, our findings highlight the importance of BRN2–ATF4 axis as a modulator of stress response that enables invasive melanoma cells to survive in transit, and suggest that targeting this interaction could provide an antigen-independent approach to eliminate metastatic precursors by shifting ISR signalling from adaptation to cell death.



## Dimitrios Koutoukidis

- Title:** Preoperative Weight Loss in Patients With Excess Weight and Colorectal Cancer: The CARE Feasibility Randomised Clinical Trial
- Author:** Dimitrios Koutoukidis, Associate Professor on Diet and Obesity
- Affiliation:** Nuffield Department of Primary Care Health Sciences, University of Oxford
- Background:** Excess weight increases morbidity risk after colorectal cancer surgery. We aimed to assess the feasibility of a preoperative weight loss intervention.
- Method:** CARE was an assessor-blinded randomised clinical trial performed between March 27, 2023, to August 13, 2024, with a 30-day postoperative follow-up in adults with a body mass index of 28 or greater who were awaiting curative elective colorectal cancer resection at 8 hospitals across England. Participants in the intervention group received dietetic support with a low-energy (800 kcal/d, 76 g/d of protein) total diet replacement program between decision to treat and surgery. Participants in the control group received usual care. Progression criteria for a definitive trial included recruitment ( $\geq 72$  patients), engagement ( $\geq 75\%$  of dietetic telephone calls attended), adherence ( $\geq 60\%$  of intervention participants achieving  $\geq 5\%$  weight loss), and retention ( $\geq 85\%$ ). Secondary outcomes included morbidity, symptoms, and changes in weight and fat-free mass.
- Results:** Of the 150 potentially eligible participants approached, 71 eligible randomized participants (mean [SD] age, 64 [8.7] years; 43 [61%] male; mean [SD] body mass index, 35.4 [5.4]) undergoing surgery (36 in the intervention group and 35 in the usual care group) were included in the analysis. Participants were accrued at a rate of 0.57 participants per site per month compared with the anticipated rate of 0.75. Participants attended 85% of their dietetic calls. The median (IQR) period from randomization to surgery was 33 (25–43) days, during which 22 (61%) of the intervention participants and 3 (9%) of the usual care participants lost 5% or more of their weight (odds ratio, 16.8; 95% CI, 4.3–65.3). Intervention participants lost a mean (SD) of 6.1 (3.0) kg before surgery, which was 4.3 kg (95% CI, 2.7 to 5.8 kg) more than the usual care participants. Between-group adjusted change in fat-free mass was 0.1 kg (95% CI, –3.9 to 4.0 kg). There was no evidence of a difference between the 2 groups in the proportion of patients with any complications (14 [39%] vs 14 [40%]) or with Clavien–Dindo grade I, II, or III complications. Faecal incontinence (–8.6 points [95% CI, –16.7 to –0.5 points]) and sore skin (–15.9 points [95% CI, –25.3 to –6.6 points]) improved postoperatively in the intervention compared with usual care group. There were no intervention-related serious adverse events. Retention was 100%. In an exploratory observational analysis, participants who lost 3.2% or more of their body weight (above the median loss) had a 50% relative reduction in complications (95% CI, 1%–78%).
- Conclusion:** This randomised clinical trial's intervention of low-energy total diet replacement before colorectal cancer resection was feasible, well tolerated, and lacked safety concerns. The progression criteria were met, but some recruitment challenges need to be addressed.

## Dongxiao Yang

- Title:** Surface Nucleolin Regulates  $\gamma$ -Irradiation–Induced DNA Damage Repair in Breast Cancer Cells
- Authors:** Dongxiao Yang, Katherine Anne Vallis
- Affiliation:** Department of Oncology, University of Oxford
- Background:** Nucleolin (NCL) is a multifunctional nucleolar phosphoprotein involved in ribosome biogenesis, chromatin organisation, and DNA metabolism. Overexpression of NCL in human tumours correlates with poor overall survival and increased risk of recurrence, suggesting a role in tumour progression. Although NCL is primarily nucleolar, aberrant cell surface localisation has been reported in malignant cells. The functional significance of surface NCL and its contribution to tumour cell survival mechanisms, particularly in the context of genotoxic stress, remain incompletely defined. We therefore investigated the expression, localisation, and functional role of NCL in cancer cells, with a focus on ribosomal RNA (rRNA) transcription and DNA damage repair following  $\gamma$ -irradiation..
- Method:** Subcellular fractionation and Western blot analysis were performed to determine NCL expression and localisation in breast, oesophageal, pancreatic, and prostate cancer cell lines. Laser scanning confocal microscopy was used to evaluate surface NCL expression in the human breast cancer cell line MDA-MB-231 and the non-tumorigenic breast epithelial cell line MCF10A. Functional studies were conducted using siRNA-mediated knockdown of NCL. rRNA transcription was assessed using 5-ethynyl uridine (EU) incorporation and Click-iT® RNA imaging. To examine the role of NCL in DNA damage repair, cells were exposed to  $\gamma$ -ray irradiation, and DNA damage responses were analysed by immunofluorescence detection of DNA damage foci.
- Results:** NCL was highly expressed in multiple cancer cell lines and enriched in membrane-associated fractions. Confocal microscopy demonstrated prominent surface localisation of NCL in MDA-MB-231 cells, whereas minimal surface expression was observed in MCF10A cells. siRNA-mediated depletion of NCL significantly reduced rRNA transcription, as evidenced by decreased EU incorporation. Importantly, NCL knockdown impaired DNA damage repair following  $\gamma$ -ray irradiation, as demonstrated by persistent DNA damage foci compared with control cells. These findings indicate that NCL supports both ribosomal biogenesis and efficient repair of irradiation-induced DNA damage in breast cancer cells.
- Conclusion:** Our data demonstrate that NCL is aberrantly expressed on the surface of breast cancer cells and plays a critical role in maintaining tumour cell survival through regulation of rRNA transcription and  $\gamma$ -irradiation–induced DNA damage repair. Targeting NCL may enhance radiosensitivity while simultaneously disrupting ribosome biogenesis, highlighting its potential as a novel therapeutic strategy in breast cancer.

## Mariana Pereira Pinho

- Title:** Watching the immune response unfold: How pembrolizumab treatment reshapes the tumour-reactive T cell repertoire in Triple-negative breast cancer patients
- Authors:** Mariana Pereira Pinho (1,2), Balraj Sandhar (1,2), Elie Antoun (1), Ting Shu (1), Magdalena Kielak (3), Rooman Javed (3), Aglaia Skolariki (3), Emma Johnson (3,4), David Maldonado-Perez (5), Adrian L. Harris (3), Yanchun Peng (1,2), Simon Lord (3), Tao Dong (1,2)
- Affiliations:** (1) CAMS Oxford Institute (COI), Nuffield Department of Medicine, University of Oxford; (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; (4) Sample Handling Lab, Oxford Cancer Trials Unit; (5) Nuffield Department of Surgical Sciences, University of Oxford
- 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.

| Posters 2026 |                                                       |                             |                                                                            |                                                                                                                                                                               |
|--------------|-------------------------------------------------------|-----------------------------|----------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Number       | AM - Odd: 10:45 to 11:30<br>PM - Even: 12:45 to 13:45 | Author                      | Department                                                                 | Title of Poster                                                                                                                                                               |
| 1            | AM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 2            | PM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 3            | AM                                                    | Samah Alshami               | Nuffield Department of Medicine                                            | Evaluating the Usefulness of the Prognostic Nutritional Index and Controlling Nutritional Status in Stratifying Prognosis in Cancer Patients during the Gaza War (2023)       |
| 4            | PM                                                    | Jasmine Ban                 | Nuffield Department of Surgical Sciences                                   | Impact of ECM Properties on Colorectal Cancer Liver Metastasis Organoids                                                                                                      |
| 5            | AM                                                    | Joshua Bull                 | Mathematics Institute                                                      | MuSpAn: A Multiscale Spatial Analysis Toolbox                                                                                                                                 |
| 6            | PM                                                    | Julius Bullo                | Nuffield Department of Surgical Sciences                                   | TBC                                                                                                                                                                           |
| 7            | AM                                                    | Alex Bullock                | Centre for Medicines Discovery                                             | E3 ligase KLHL6 is a lymphoid-specific tumour suppressor recruiting Roquin2 through a multivalent degron mechanism                                                            |
| 8            | PM                                                    | Zhuoyao Chen                | Centre for Medicines Discovery                                             | Structural mimicry of UM171 and neomorphic cancer mutants co-opts E3 ligase KBTBD4 for HDAC1/2 recruitment'                                                                   |
| 9            | AM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 10           | PM                                                    | Heather Clark               | Department of Oncology                                                     | Investigating the role of macrophages in the intestinal tissue response to radiotherapy and C5aR1 inhibition                                                                  |
| 11           | AM                                                    | Joshua Moore                | Mathematics Institute                                                      | Towards Spatial Signatures of Treatment Response in Colorectal Cancer                                                                                                         |
| 12           | PM                                                    | Luke De Bretton-Gordon      | Mathematics Institute                                                      | Data-Driven Modelling of Cell Cycle Regulation                                                                                                                                |
| 13           | AM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 14           | PM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 15           | AM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 16           | PM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 17           | AM                                                    | Kezia Gaitskell             | Radcliffe Department of Medicine/ Nuffield Department of Population Health | Tubal ligation and incidence of histological types of uterine and ovarian cancers: Evidence for different origins of serous carcinomas of the ovary and uterus                |
| 18           | PM                                                    | Christiana Kartsonaki       | Nuffield Department of Population Health                                   | Infection with Toxoplasma gondii and risk of prostate cancer                                                                                                                  |
| 19           | AM                                                    | Dimitrios Koutoukidis       | Nuffield Department of Primary Care Health Sciences                        | Preoperative intentional weight loss to support postoperative recovery in patients with excess weight and colorectal cancer: the CARE feasibility randomised controlled trial |
| 20           | PM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 21           | AM                                                    | Martin Little               | Department of Oncology                                                     | Using cross-trait polygenic risk scores to predict toxicity and response in immune checkpoint-blockade                                                                        |
| 22           | PM                                                    | Pakavarin Louphrasitthiphol | Department of Oncology                                                     | A Novel Non-Genetic Strategy to Shift the Stress–Survival Balance Toward Cancer Cell Death                                                                                    |
| 23           | AM                                                    | David Maxen                 | Department of Oncology                                                     | Anomaly Detection Approaches for Circulating Tumour DNA Detection in Liquid Biopsies                                                                                          |
| 24           | PM                                                    | Gusztav Milotay             | Department of Oncology                                                     | Dissecting the relationship between response to ICB and CMV status in melanoma                                                                                                |
| 25           | AM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 26           | PM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 27           | AM                                                    |                             |                                                                            | Information                                                                                                                                                                   |
| 28           | PM                                                    | Victoria Perletta           | Department of Oncology                                                     | Using longitudinal linked electronic health records to investigate lifetime obesity prevalence in systemic anticancer therapy patients in England.                            |
| 29           | AM                                                    | Ryan Pink                   | Biomedical Sciences - Oxford Brookes University                            | Using uniquely stable molecular imprinted polymers to replace antibodies in cancer extracellular vesicle enrichment and detection                                             |
| 30           | PM                                                    | Adán Pinto-Fernández        | Nuffield Department of Medicine                                            | Deletion of the deISGylatingenzyme USP18 enhances tumour cell antigenicity and radiosensitivity                                                                               |
| 31           | AM                                                    | George Randall              | Centre for Medicines Discovery, Nuffield Department of Medicine            | A Pipeline Towards Rational Design of Molecular Glue Degradars                                                                                                                |
| 32           | PM                                                    | Jia-Ling Ruan               | Department of Oncology                                                     | Enhancing Solid Tumor Immunotherapy by Combining Spatially Fractionated FLASH Radiotherapy with mRNA-LNP–Based Bispecific Antibody Therapy                                    |
| 33           | AM                                                    | Dimitris Vavoulis           | Department of Oncology                                                     | Nanopore cfDNA Whole-Genome Sequencing for Multi-Cancer Early Detection                                                                                                       |
| 34           | PM                                                    | Peter Wan                   | Department of Oncology                                                     | Machine perfusion and spatial transcriptome mapping identify intra-tumoural endothelial anergy as an immune escape mechanism in colorectal liver metastasis                   |
| 35           | AM                                                    | Dongxiao Yang               | Department of Oncology                                                     | Nucleolin regulates DNA damage repair and radioresistance in breast cancer                                                                                                    |
| 36           | PM                                                    | Yuxin Zeng                  | Nuffield Department of Surgical Sciences                                   | High-Resolution Mapping of the Primary Melanoma Tumour Microenvironment Using Multimodal Spatial Profiling                                                                    |
| 37           | AM                                                    | Aihan Zhang                 | Department of Oncology                                                     | TBC                                                                                                                                                                           |
| 38           | PM                                                    | Lena Zuspänn                | Mathematical Institute                                                     | Genetic switching dynamics in a spatial branching model of cancer metastasis                                                                                                  |