BIG

BRISBANE IMMUNOLOGY GROUP

CONFERENCE 2026

Advancing knowledge.
Improving immune health.

Thursday 20 – Friday 21 August 2026

Sea World Resort, Gold Coast

ABSTRACT BOOKLET



IMMUNITY

GENOMICS

IMPACT

Bringing together researchers, clinicians, and industry leaders to drive discovery and translate immunogenomics into better health outcomes.

www.qimrb.edu.au/big

QIMR Berghofer

THE FUTURE OF HEALTH

Sanofi is an R&D-driven, AI-powered biopharma company committed to improving people's lives and delivering compelling growth. We apply our deep understanding of the immune system to invent medicines and vaccines that treat and protect millions of people around the world, with an innovative pipeline that could benefit millions more. Our team is guided by one purpose: we chase the miracles of science to improve people's lives; this inspires us to drive progress and deliver positive impact for our people and the communities we serve, by addressing the most urgent healthcare, environmental, and societal challenges of our time. www.sanofi.com.au

INTEGRA BIOSCIENCES is a leading provider of high-quality laboratory tools and consumables for liquid handling and media preparation. We are committed to fulfilling the needs of laboratory professionals in research, diagnostics, and quality control within the life sciences industry.

Millennium Science ignites life science research through revolutionary technologies. We provide workflow solutions for every stage of the scientific journey - from sample collection to translational research. Our immunology portfolio includes reagents from leading suppliers such as InvivoGen, Sino Biological, Acro Biosystems, Cusabio, Mabtech, Meridian Bioscience, Norgen Biotek, and MedChemExpress. Products span sample preparation kits, cell-based assays, cytokine detection, recombinant proteins, antibodies, and molecular biology reagents. Supporting applications from discovery and characterisation to functional studies and biomarker validation, we help researchers accelerate immunology breakthroughs. With trusted global brands and expert technical support, Millennium Science is your partner in advancing human health.

Learn more at www.mscience.com.au.

Miltenyi Biotec is a global leader in innovating technologies and services for patient-specific cell and gene therapies, transforming scientific discoveries into practical treatments for personalized medicine. With over 35 years of expertise, it supports biomedical discoveries and translates them into clinical applications, enhancing patient access to new therapies. Miltenyi Biotec focuses on offering integrated solutions for complex challenges in treating cancers, autoimmune diseases, and inherited disorders. Its Miltenyi Bioindustry division provides expert guidance to therapy developers efficiently from process development to commercialization. Headquartered in Bergisch Gladbach, it employs 4,700 people across 24 countries.

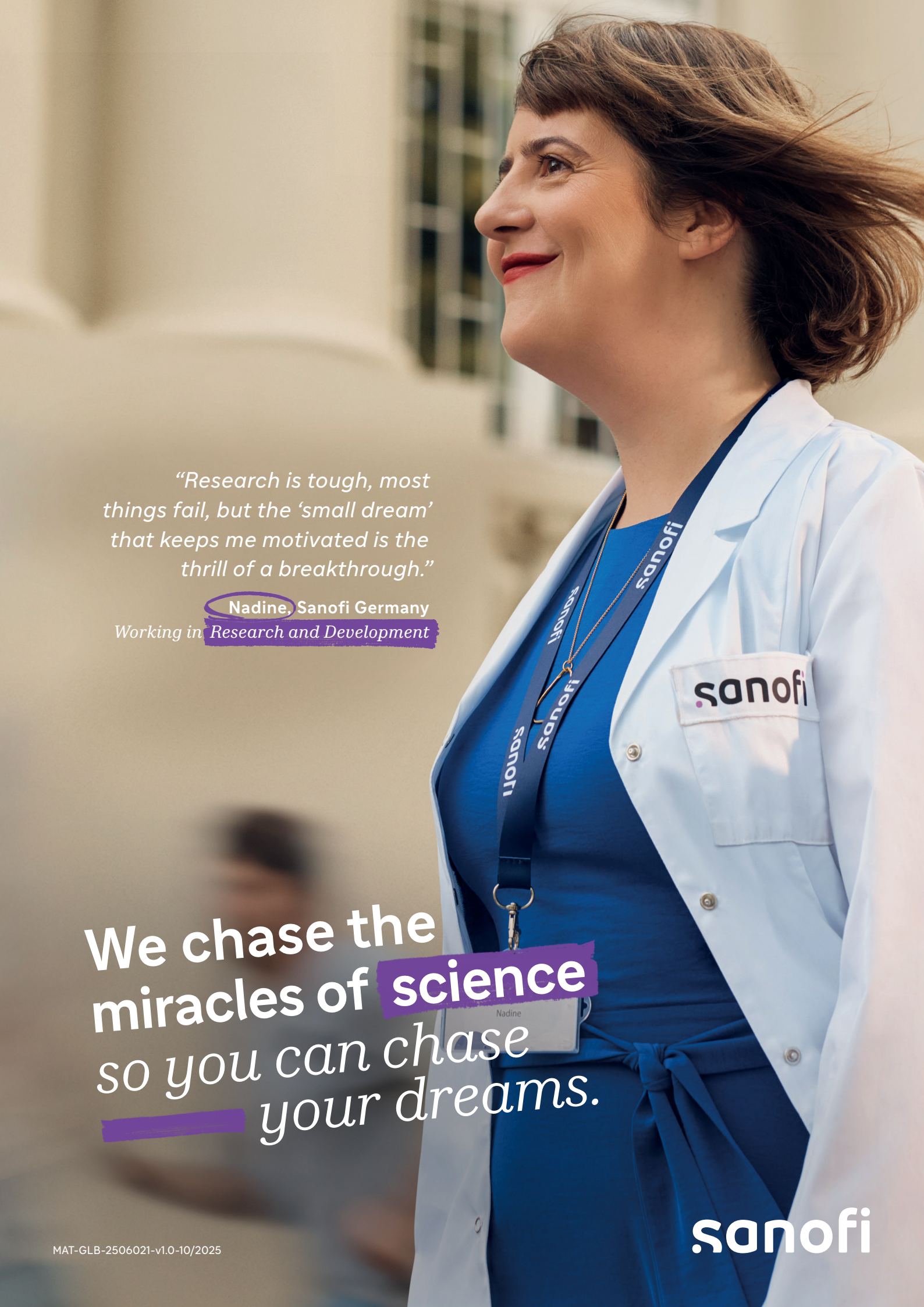
Translational Research Institute (TRI) accelerates translational research by connecting academic, clinical and industry partners to drive discoveries toward improved patient outcomes. TRI's integrated Core Facilities bring together flow cytometry, microscopy, histology, preclinical imaging, biological resources, gnotobiotic and human immune model platforms. Supported by expert technical staff, TRI enables high quality, translational data and access to advanced technologies in a collaborative environment. TRI's specialist capabilities strengthen and accelerate the development of complex immunology and biomedical projects, helping researchers advance their next research project.

The **Queensland Immunology Research Centre (QIRC)** is a multidisciplinary research network connecting researchers from The University of Queensland and QIMR Berghofer Medical Research Institute. Bringing together strengths across multiple areas of immunology research, QIRC supports collaboration to advance understanding of the immune system and support discoveries that improve health outcomes. QIRC is committed to supporting early- and mid-career researchers through initiatives including PhD scholarship top-ups, travel awards and EMCR seed grants. To stay informed about funding opportunities, events and other initiatives, subscribe to the QIRC newsletter at qirc.com.au/news/list.

Australian Biosearch proudly represents **BioLegend** in Australia and New Zealand. We distribute world-class antibodies and reagents engineered for high-impact immunology, oncology, and advanced flow cytometry. Discover more than 35,000 trusted highly validated, reproducible reagents that ensure experimental integrity. Elevate your research standard and secure reliable data with our analytical tools that can make a difference in your lab. For more information on our suppliers please visit www.aust-biosearch.com.au

The history of **Beckman Coulter** is one of innovation. Our mission today is to empower those who are seeking answers to life's important scientific and healthcare questions. Beckman Coulter Life Sciences is a leader in scientific research instruments serving the academic, biotech and pharma markets. The company also serves the clinical research and applied markets.

Waters Biosciences (formerly **BD Biosciences**) is a global leader in life sciences and diagnostics, dedicated to accelerating the benefits of pioneering science through analytical technologies, informatics, and service. With a focus on regulated, high-volume testing environments, our innovative portfolio harnesses deep scientific expertise across chemistry, physics, and biology. We collaborate with customers around the world to advance the release of effective, high-quality medicines, ensure the safety of food and water, and drive better patient outcomes by detecting diseases earlier, managing routine infections, and combating antibiotic resistance. Through a shared culture of relentless innovation, our passionate team of colleagues turn scientific challenges into breakthroughs that improve lives worldwide.


"Research is tough, most things fail, but the 'small dream' that keeps me motivated is the thrill of a breakthrough."
Nadine, Sanofi Germany
Working in Research and Development
We chase the miracles of science so you can chase your dreams.
sanofi
MAT-GLB-2506021-v1.0-10/2025



Thursday, 20th August 2026

8.00AM	Bus departs from Brisbane
10.00AM – 10.50AM	Registration, Morning Tea and Poster Setup
10.50AM – 11.00AM	**Welcome** Associate Professor Danielle Stanisic, ASI-QLD Councillor
11.00AM – 11.30AM	**BIG Plenary Lecture I** Chair: Associate Professor Danielle Stanisic Sponsored by: Millenium Science
	Professor Shalin Naik, Walter and Eliza Hall Institute *The hidden regulators of clonal destiny*
11.30AM – 12.30PM	**Postgraduate Session I** Chair: Dr Carla Proietti
	Patricia Namubiru, QIMR Berghofer *Targeting specific neutrophil populations ameliorates severe bronchiolitis in infancy and prevents the subsequent development of asthma.*
	Christopher Hunter, QIMR Berghofer *Dissecting divergent immune contributions to HFpEF using murine and human cardiac organoid models*
	Chen Wang, University of Queensland *Identification of tumor antigen specific T cells using ruthenium mediated photocatalytic proximity labelling*
	Mason Lacy, QUT *Uncovering the pathway for T cell differentiation during ex vivo expansion*

12.30PM – 1:00PM	**Hot Topics - Organoids** Chair: Professor Rajiv Khanna Sponsored by: Australian Biosearch and Biolegend
	Professor James Hudson, QIMR Berghofer *Using human cardiac organoids to dissect the role of inflammatory factors in heart failure*
	Dr Giovanni Pietrogrande, AIBN, University of Queensland *Modelling the neuroimmune system in brain organoids: how and why*
1.00PM – 2.30PM	**Lunch**
2.30PM-3.00PM	**Postgraduate Plenary Lecture I** Chair: Associate Professor Danielle Stanisic Sponsored by: Beckman Coulter
	Associate Professor Julia Ellyard, John Curtin School of Medical Research, ANU *Unravelling Lupus pathogenesis: genetics, cytokine regulation and autoantibodies*
3.00PM – 4.15PM	**Postgraduate Session II** Chair: Dr Adriana Pliego Zamora
	Vrushali Maste, University of Queensland *Differential fluid and particle uptake by cells of the bone marrow*
	Dr Shoaib Anwaar, Griffith University *Immunoediting Rewires the Tumour Microenvironment Toward Regulatory T Cell Dominance in UV-Induced Malignancies*
	Capucine Bianciotto, Mater Research *Dissecting the Role of CD169+ Macrophages in Traumatic Spinal Cord Injury Using a Multi-Omics Approach*
	Ennae Gollasch-Miller, University of Queensland *Complement dependent enhancement of Dengue virus infection*
	Thishan Illankoon, University of Queensland *The Impact of Age-Related Decline in the Unfolded Protein Response on Respiratory Infection Susceptibility and Severity*
4.15PM – 5.15 PM	**Afternoon Tea & Free time & Beach Cricket**
5.15PM – 6.30PM	**Poster Session (Light Refreshments and Drinks)**
6.30PM – 7.10PM	**Jonathan Sprent Oration** Chair: Professor Rajiv Khanna Sponsored by: Sanofi
	Professor Sharon Lewin, Doherty Institute *Towards an HIV cure: the role of anti-PD1, mRNA and other immunotherapies*
7.10PM – 9.10PM	**Conference Dinner**

Friday, 21st August 2026

7.00AM – 8.30AM	**Breakfast**
8.30AM – 9.00AM	**Postgraduate Plenary Lecture II** Chair: Associate Professor Katharine Irvine Sponsored by: Integra Biosciences
	Professor Michael Gantier, Hudson Institute of Medical Research *Harnessing a new checkpoint to control auto-immunity in lupus patients.*
9.00AM – 10.00AM	**Early/Mid Career Researcher Session** Chair: Associate Professor Kate Gartlan
	Dr Amanda Oliver, QIMR Berghofer *From First Cells to First Communities: A Multiomic Atlas of the Early-Life Gut*
	Dr Sabrina Sofia Burgener, University of Queensland *Feeding the inflammasome to damage the liver: new insights into Caspase-1 biology*
	Dr Ismail Sebina, Frazer Institute, University of Queensland *Pre-clinical assessment of mRNA vaccine-induced immunity to Group A Streptococcus*
	Dr Harindra Sathkumara, James Cook University *Mucosal vaccination with virulence-enhanced recombinant BCG protects independently of adaptive immunity and drives bone marrow myelopoiesis*
10.00AM – 10.30AM	**Morning Tea**
10.30AM – 11.00AM	**BIG Plenary Lecture II** Chair: Associate Professor Katharine Irvine Sponsored by: Translational Research Institute
	Professor Di Yu, Frazer Institute, University of Queensland *Ferroptosis: a new dimension in governing T cell function*
11.00AM – 12.00PM	**BIG Session I** Chair: Dr Adriana Pliego Zamora
	Professor Trent Woodruff, University of Queensland *Microglial-intrinsic complement signalling drives neurodegeneration*
	Associate Professor Kate Gartlan, QIMR Berghofer *Transcriptional drivers of alloreactive CD8⁺ T cell responses in donor stem cell transplantation*
	Professor Kate Seib, Griffith University *A meningococcal vaccine against gonorrhoea: Insights from recent clinical trials*

12.00PM – 1.00PM	**Lunch and QIRC-Sponsored Mentoring session**
	Mentoring Session Sponsored by: Queensland Immunology Research Centre
	Being a Woman in Immunology - Swimming Upstream (15 mins) Dr Debbie Watson, University of Wollongong
	QIRC and QIRC Opportunities (15 mins) Professor Gabrielle Belz, Frazer Institute, University of Queensland
	ASI Opportunities and awards (10 mins) Associate Professor Danielle Stanisic, Griffith University
1.00PM – 2.00PM	**BIG Session II** Chair: Dr Carla Proietti
	Professor Sumaira Hasnain, Mater Research *Interleukin-24 as a Central Regulator of Intestinal Fibrosis*
	Dr Roland Ruscher, James Cook University *Long-lived CD8aa intestinal intraepithelial T cells persist despite restrained antigen receptor signaling and are shaped by chronological aging and microbial experience*
	Dr Janin Chandra, Frazer Institute, University of Queensland *Circulating CD5⁺ dendritic cells link gut microbiome function to immunotherapy response in cancer patients*
2.00PM – 2.30PM	**BIG ICON Lecture** Chair: Professor Chris Engwerda Sponsored by: Miltenyi Biotec
	Professor Denise Doolan, IMB, University of Queensland *From Malaria Vaccines to Precision Immunology: Reflections on a Scientific Journey*
2.30PM – 3:00PM	**Prizes and Closing Remarks** Prizes sponsored by: Australian and New Zealand Society for Immunology
3.45PM	Bus departs from Surfers Paradise to Brisbane

millennium® science

At Millennium Science, we support every stage of the scientific discovery journey.
Our comprehensive product portfolio spans reagents and instruments for:



- Sample collection & preservation
- Sample extraction & purification
- Lab Automation
- Validating sample quality & integrity
- Cell Culture Reagents

SAMPLE PREPARATION



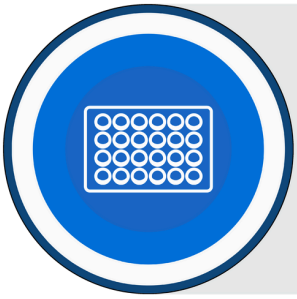
- Omics approaches
  - Single Cell, Spatial Transcriptomics, Metabolomics
- Screening
- Lab Automation
- Flow Cytometry
- Immunoassays - ELISpot, FluoroSpot, FociSpot

DISCOVERY TOOLS



- Plate readers
- Molecular Characterisation - PCR/RT-qPCR
- Accurate DNA/RNA quantification
- Expression Analysis
- Microscopy/Imaging
- Immunoassays
- Flow Cytometry

CHARACTERISATION



- Genetic Tools
- Biochemical assay kits & reagents
- Flow Cytometry
- Functional assays
  - Reporter cells, Immunoassays, Neutralising antibodies
- Pathway analysis
- Lentivirus & exosome studies

FUNCTIONAL STUDIES



- Biomarker validation
- Biologics characterisation
- Immunoassays
- Drug stability and formulation
- Extracellular vesical characterisation
- GMP nanoparticle manufacturisation
- Therapeutic release quantitation

TRANSLATIONAL

Search >920,000 products on our website today!
www.mscience.com.au

BIG Plenary Lecture I

Chair: Associate Professor Danielle Stanisic

The hidden regulators of clonal destiny

Shalin Naik

Walter and Eliza Hall Institute, Melbourne (VIC) Australia

Human haematopoietic stem and progenitor cells (HSPCs) exhibit heterogeneous lineage output, but the molecular programs underlying clonal fate remain poorly defined. To address this, we developed a human 2D haematopoietic organoid supporting differentiation into 15 cell types spanning the myeloid, lymphoid, dendritic cell and meg/erythroid lineages, called HaemaTONIC. Using this system, we barcoded and tracked HSPC clones over time. We establish that haematopoietic clonal fate is heterogeneous but stably encoded i.e. 'destined'. However, the genes and surface markers that predict clonal fate could not be found using common clustering approaches and remained hidden. Instead, by leveraging our clonal multi-omics technique SIS-seq - where clonal siblings are tested separately for fate or single cell 'omics - followed by machine learning, we uncover *clonal fate modules* that predicted clonal destiny including one for multipotency. These results have implications for haematopoiesis in health, ageing, blood and immune disorders, and leukemia.

Postgraduate Session I

Chair: Dr Carla Proietti

Targeting specific neutrophil populations ameliorates severe bronchiolitis in infancy and prevents the subsequent development of asthma.

Patricia Namubiru[1,2], Ismail Sebina[1], Tufael Ahmed[1,3], Daniel R Howard[1,2], Ridwan B. Rashid[1,2], MD Sohel Rana[1,3], Tyler Wilkins[1,2], Mariah Alorro[1], Michelle Wykes[1], and Simon Phipps[1,2,3]

[1]QIMR Berghofer Medical Research Institute, Herston, Queensland 4006, Australia; [2]School of Biomedical Sciences, The University of Queensland, Queensland 4072, Australia; [3]School of Biomedical Sciences, Queensland University of Technology, Queensland 4000, Australia.

Lifestyle factors that disturb the assembly of the gut microbiome in early life increase the risk of infant severe lower respiratory infections (sLRI), which in turn predispose to childhood asthma. To explore whether neutrophils contribute to this nexus, we employed an established mouse model whereby neonates reared to low fibre diet (LFD)-fed dams and inoculated with pneumovirus of mice (PVM) develop sLRI, characterised by an overzealous neutrophil and T cell response, mucus hyperproduction, and a high viral load, and develop experimental asthma upon allergen exposure in later-life. Here, we found that neutrophil depletion (anti- Ly6G) during the viral infection had no effect on LRI severity but prevented subsequent asthma in LFD-reared neonates. High dimensional analysis of lung neutrophils identified 8 distinct neutrophil clusters during the infection, three of which were numerically greater in LFD- compared to high fibre diet-reared neonates. PD-L1 expression was elevated on all three clusters and conventional flow cytometry confirmed that PD-L1 expression was higher on neutrophils compared to other leukocytes. Critically, anti-PD-L1 blockade in LFD-reared neonates depleted the PD-L1+ neutrophil population, ameliorated LRI severity, and prevented the subsequent development of asthma. Significantly, anti-PD-L1 decreased T cell expansion but enhanced the T cell response qualitatively as determined by increased granzyme B and perforin expression and cytokine production. This was associated with accelerated viral clearance and decreased immunopathology. Together, our findings demonstrate that specific subpopulations of neutrophils contribute to LRI severity, and that the specific targeting of PD- $L1^{+}$ neutrophils ameliorates LRI severity and prevents the later onset of asthma.

Dissecting divergent immune contributions to HFpEF using murine and human cardiac organoid models

Christopher R. Hunter[1,2], Conor J. Bloxham[1], Henrietta Cserne Szappanos[1], James E. Hudson[1]

[1]QIMR Berghofer Medical Research Institute, Brisbane, QLD 4006, Australia. [2]School of Biomedical Sciences, The University of Queensland, Brisbane, QLD 4072, Australia.

Heart failure with preserved ejection fraction (HFpEF) affects ~32 million worldwide and is associated with significant patient morbidity and a 5-year survival of ~50%. The prevalence of HFpEF is projected to increase within the coming decades due to an aging population and increased incidence of co-morbidities that drive sustained chronic inflammation. This sustained inflammatory environment promotes a shift in resident cardiac leukocytes toward pro-inflammatory phenotypes, impairing their normal tissue-protective roles. Despite this, current therapeutic strategies targeting these immune processes remain limited and largely ineffective, highlighting an urgent need for novel approaches. In our study, we aimed to characterise how the immune system shapes tissue function and HFpEF pathogenesis. We hypothesised that targeting pathways driving the altered homeostatic function of resident cardiac leukocytes may represent a next-generation therapeutic strategy. Here, we establish a novel HFpEF model in female ex-breeder mice that combines high-fat diet (obesity), angiotensin II (hypertension), and isoprenaline (adrenergic activation), and recapitulates hallmark clinical features including hypertrophy, fibrosis, and diastolic dysfunction. In this model, HFpEF drove T-cell infiltration into the cardiac parenchyma alongside a phenotypic switch from resident macrophages (TIMD4^{+}MHC-IIhi) to pro-fibrotic, bone-marrow-derived macrophages (CD206^{+}TIMD4neg). Antibody-mediated depletion identified T cells, rather than macrophages, as the primary drivers of cardiac dysfunction *in vivo*. To interrogate this axis directly, we developed a co-culture system combining stem cell-derived macrophages with human cardiac organoids. Macrophages colonised the organoids long-term and enhanced contractile force at steady state. Using IFNγ as a model of T-cell-driven inflammation, we found it drove diastolic (relaxation) dysfunction in cardiac organoids; the inclusion of macrophages protected against IFNγ-driven diastolic dysfunction. This reframes macrophages not as drivers of disease but as beneficial to cardiac function. By leveraging these models to dissect immune- driven mechanisms in HFpEF, we aim to pave the way for immune-targeted therapies that treat cardiac disease.

Identification of tumor antigen specific T cells using ruthenium mediated photocatalytic proximity labelling

Chen Wang[1,4], Hongjian Sun[1], Yunze Wu[2], Liang Zhang[3], Chunchan Li[4], Xi Jiang[2],Yaping Chen[4], Jie P. Li[2] , Mingdong Lu[3], Di Yu[1,5*]

[1]Ian Frazer for Children's Immunotherapy Research, Children's Health Research Centre, Faculty of Health, Medicine and Behaviour Sciences, The University of Queensland, Brisbane, Queensland, Australia [2]State Key Laboratory of Coordination Chemistry, Chemistry and Biomedicine Innovation Center (ChemBIC), School of Chemistry and Chemical Engineering, Nanjing University, Nanjing 210023, China [3]Department of Gastrointestinal Surgery, The Second Affiliated Hospital and Yuying Children's Hospital of Wenzhou Medical University, Wenzhou, China [4]Oujiang Laboratory, Key Laboratory of Alzheimer's Disease of Zhejiang Province, Institute of Aging, Wenzhou Medical University, Wenzhou, People's Republic of China [5]Frazer Institute, Faculty of Health, Medicine and Behaviour Sciences, The University of Queensland, Brisbane, Queensland, Australia

Tumor antigen specific T cells play a central role in anti tumor immunity, yet their identification within the tumor microenvironment remains challenging using conventional in vitro coculture systems. Here, we evaluated ruthenium mediated photocatalytic proximity labeling as an approach for identifying tumor antigen specific T cells in human colorectal cancer specimens. Two photocatalytic labeling strategies were compared: an NHS ester conjugated ruthenium catalyst (NHS-Ru), which anchors to cell surface proteins, and a GDP fucose ruthenium conjugate (Ru-G), which labels cell surface glycans through endogenous fucosyltransferases. In freshly dissociated colorectal cancer samples, NHS-Ru showed greater labeling stability than Ru-G and was therefore selected for subsequent experiments. Fresh colorectal tumors were dissociated into single cell suspensions, and tumor cells together with tumor infiltrating T cells were isolated by flow cytometry. NHS-Ru mediated photocatalytic proximity labeling was applied in both direct tumor cell T cell coculture and dendritic cell mediated antigen presentation assays using tumor lysates. Background labeling was defined using MHC blockade, allowing more accurate identification of antigen reactive T cells. In the direct tumor cell T cell coculture system, approximately 0.16% of CD4+ T cells and 0.54% of CD8+ T cells were identified as T cells capable of recognizing autologous tumor cells. In the dendritic cell mediated antigen presentation system, approximately 3.74% of CD4+ T cells were identified as T cells capable of recognizing tumor antigen presenting dendritic cells. This study establishes an experimental workflow for applying NHS-Ru mediated photocatalytic proximity labeling to freshly isolated human colorectal cancer samples. The workflow enables enrichment of tumor antigen specific T cells directly from patient specimens and provides a practical approach for investigating tumor specific immune responses in human tumors.

Uncovering the pathway for T cell differentiation during *ex vivo* expansion

Mason Lacy[1,2], Yoav Binenbaum[3,4,5], Pascal Buenzli[1,6], Adrianne Jenner[1,2]

[1]School of Mathematical Sciences, Queensland University of Technology (QUT), Brisbane, Australia; [2]Centre for Data Science, QUT, Brisbane, Australia; [3]John A. Paulson School of Engineering and Applied Sciences, Harvard University, Cambridge, MA 02138, USA; [4]The Wyss Institute for Biologically Inspired Engineering, Harvard University, Boston, MA 02138, USA; [5]Dana-Farber/Boston Children's Cancer and Blood Disorders Center, Boston, MA 02115,USA [6]ARC Centre of Excellence for the Mathematical Analysis of Cellular Systems, QUT, Brisbane, Australia

Adoptive T cell therapy is a promising immunotherapy for treating cancer, leveraging the T cell's natural ability to target and kill cancer cells. A crucial step in this therapy involves the expansion of T cells *ex vivo*, however, this process is often slow and it can be difficult to balance the desired cell counts with memory, effector and exhausted T cell phenotypes. Mathematical modelling is a powerful tool that can be used to optimise the expansion of relevant T cell phenotypes. The development of such a model requires that the pathways for differentiation between memory, effector and exhausted cells are well-understood. Unfortunately, there is a large amount of uncertainty and disagreement in the literature describing the ways in which T cells differentiate to memory and effector cells or become exhausted, limiting model-informed optimisation strategies. We conduct a large-scale mathematical investigation that aims to answer fundamental questions regarding T cell differentiation and exhaustion in the context of *ex vivo* expansion of healthy and cancer experienced T cells. By comparing thousands of hypothetical differentiation and exhaustion pathways to experimental data describing the *ex vivo* expansion of healthy donor and patient-derived T cells, we find that antigen experience likely drives changes in T cell differentiation kinetics rather than pathway structure. Our model suggests that T cells likely differentiate in a bi-directional manner, effector cell proliferation is insignificant, and differentiation to effector phenotypes does not require T cell receptor stimulation. We also propose biological research avenues that have the potential to reduce uncertainty and gain further insights under our methods. This framework allows for a deeper understanding of T cell behaviour during immune responses and paves the way for a more accurate understanding of *ex vivo* T cell expansion to improve the administration of adoptive cell therapy in a patient-specific manner.

BioLegend
From Revvity

Optimized Multicolor Flow Cytometry Panels

Our expertly designed optimized flow cytometry panels take the guesswork out of panel and experimental design, allowing you to focus on your next discovery. Our panel bundles include everything you need, fluorophore-conjugated antibodies, buffers, and a detailed protocol.

https://www.biolegend.com/en-us/flow-cytometry/optimized/large-panels

Human General Phenotyping (26c) Panel

The Human General Phenotyping (26 color) Panel has been optimized for examining the major immune cell subsets in human peripheral blood, including T, B, NK, innate lymphoid, myeloid, and dendritic cell subpopulations. This kit includes 25 reagents and a detailed, easy-to-follow protocol for maximal reproducibility. Panel optimization was performed using a 5-laser Cytek® Aurora (UV, V, B, YG, R).

https://www.biolegend.com/en-us/flow-cytometry/optimized/human-panels/hu-general-phenotyping-26c

Human Expandable Base Panel (15c)

A 15-color expandable panel for human immune cell phenotyping, enabling detailed characterization of monocytes, macrophages, NK and NKT cells, T cells, B cells, dendritic cells, basophils, and ILCs. The panel is designed to keep channels with broad conjugate availability open, allowing seamless customization. Several strategically placed drop-in/open spots enable the addition of extra markers or conjugates, supporting straightforward expansion up to 23 colors without disrupting the core panel design. Panel optimization was performed using a 5-laser Cytek Aurora.

https://www.biolegend.com/en-us/flow-cytometry/optimized/human-panels/hu-expandable-base-panel-15c

Mouse Expandable Base Panel (14c)

A 14-color expandable panel for mouse immune cell phenotyping, enabling detailed characterization of monocytes, macrophages, NK and NKT cells, T cells, B cells, dendritic cells, and neutrophils. The panel is designed to keep channels with broad conjugate availability open, allowing seamless customization. Several strategically placed drop-in/open spots enable the addition of extra markers or conjugates, supporting straightforward expansion without disrupting the core panel design. Panel optimization was performed using a 5-laser Cytek Aurora.

https://www.biolegend.com/en-us/flow-cytometry/optimized/mouse-panels/mu-expandable-base-panel-14c

AUSTRALIAN biosearch
www.aust-biosearch.com.au
customerservice@aust-biosearch.com.au

Hot Topics - Organoids

Chair: Professor Rajiv Khanna

Using human cardiac organoids to dissect the role of inflammatory factors in heart failure

James Hudson

Cardiac Bioengineering Laboratory, Infection and Inflammation Program, QIMR Berghofer Medical Research Institute, Brisbane (QLD) Australia

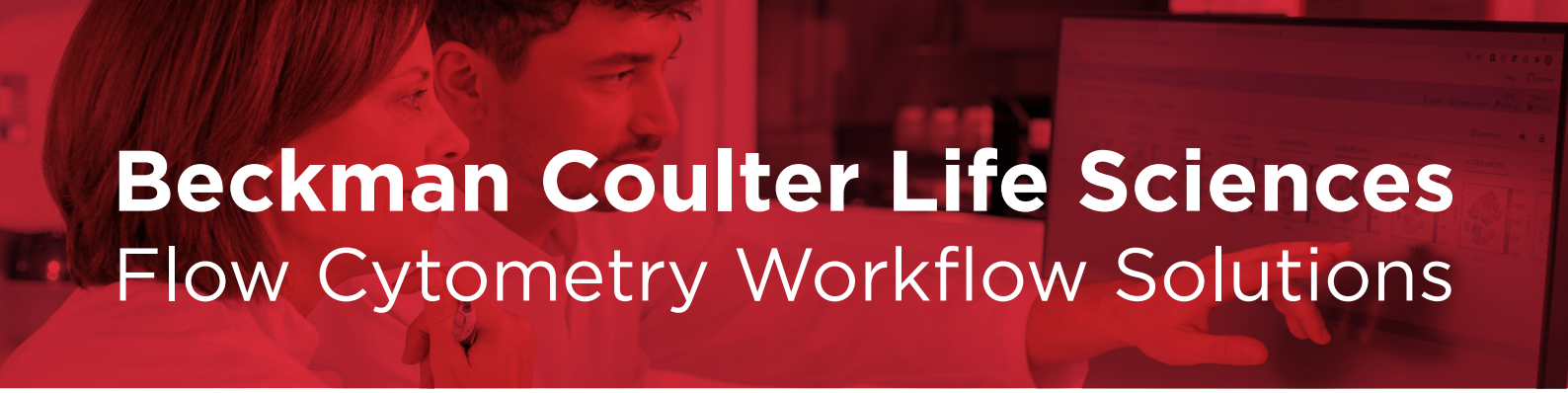
Perturbation datasets including Connectivity Map/LINCS, DepMap, and RxRx have been critical for AI-driven drug discovery and repurposing. However, these foundational resources are overwhelmingly cancer-focused. Equivalent perturbation resources for cardiac biology remain absent, creating a critical bottleneck for AI-guided cardiac drug discovery. To address this, we have completed phase 1 of the first cardiac-specific perturbation dataset designed for AI/ML applications by profiling functional contraction force and kinetics and transcriptional signatures. We targeted all 103 expressed cell surface soluble ligand receptors with available ligands in multicellular human cardiac organoids (hCOs), which covers 88% of those expressed in the human heart. We use this dataset to “fingerprint” human heart failure samples and complex responses in order to discover the core signalling at the basis of disease. This includes an interferon-gamma response that cause a paracrine IL-6 family signalling loop from fibroblasts driving dysfunction. We show the utility of Cardiopedia-Ligand, and our further larger scale datasets may transform machine learning applications in heart failure drug discovery pipelines.

Modelling the neuroimmune system in brain organoids: how and why

Giovanni Pietrogrande

Australian Institute for Bioengineering and Nanotechnology – AIBN, The University of Queensland

Brain organoids have rapidly emerged as powerful human models for studying brain development, disease mechanisms, and therapeutic responses. However, generating biologically relevant organoids remains challenging because the human brain comprises many different cell types. Among these, microglia are the resident immune cells of the central nervous system, and play essential roles in brain development, tissue homeostasis, and the initiation and resolution of inflammation. The absence of microglia from many conventional brain organoid models remains a major limitation for studying brain development and neurological disease. Recent advances have enabled the generation of immunocompetent brain organoids mainly through two approaches: the incorporation of exogenous microglia or the promotion of their endogenous development. These complementary strategies offer distinct opportunities to investigate neuroimmune interactions, although each presents unique biological and technical considerations depending on the application. We have developed several complementary strategies to generate immunocompetent brain organoids tailored to different biological questions and disease contexts. These neuroimmune models have been applied to investigate Alzheimer's disease, motor neuron disease, multiple sclerosis, and hypoxic brain injury, showcasing how the incorporation of microglia enables the study of disease mechanisms, neuroinflammatory responses, and therapeutic interventions that cannot be captured by neuron-only organoids. By recapitulating the cellular complexity of the human brain and diverse aspects of disease pathophysiology, immunocompetent organoids are emerging as a powerful platform for investigating human brain biology, identifying therapeutic targets, and accelerating translational drug discovery.



Beckman Coulter Life Sciences
Flow Cytometry Workflow Solutions

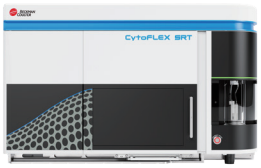
Translational Research Instrumentation and Analysis Software



Antibody Cocktailing
Biomek i3
Benchtop Liquid
Handler



Extracellular Vesicle and Nanoparticle Analysis
CytoFLEX nano
Flow Cytometer



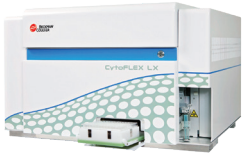
Nanoscale Sorting
CytoFLEX SRT
Benchtop Cell Sorter



Spectral Flow Cytometry
CytoFLEX mosaic
Spectral Detection Module

Cytobank
SINGLE CELL.
BIG PICTURE.

Cytobank
Gain insights from
large datasets



Conventional Flow Cytometry
CytoFLEX LX
Flow Cytometer

Kaluza Analysis Software

Translational Research Reagents



DURAClone Kits
- Immune Cell Profiling
- Immune Function
- Rare Poulation
- Stem Cell Duractiv



superRCA® technology
Advanced molecular assay
for flow cytometry

Liquid Single-Color Antibodies
Liquid Multi-Color Antibody Cocktails
Cell Health,Kits, Sample Prep



SCAN ME

superRCA® is a trademark or registered trademark of Rarity Bioscience AB
© 2026 Beckman Coulter, Inc. All rights reserved. Beckman Coulter, the stylized logo and the Beckman Coulter product and service marks mentioned herein are trademarks or registered trademarks of Beckman Coulter, Inc. in the United States and other countries. All other trademarks are the property of their respective owners.

BECKMAN COULTER
Life Sciences

For Beckman Coulter's worldwide office locations and phone numbers, please visit Contact Us at beckman.com

2026-GBL-EN-109906-V1

danaher

Postgraduate Plenary Lecture I

Chair: Associate Professor Danielle Stanisic

Unravelling Lupus pathogenesis: genetics, cytokine regulation and autoantibodies

Julia Ellyard

John Curtin School of Medical Research, Australian National University, Acton ACT 2601, Australia.

Systemic lupus erythematosus (SLE) is the prototypic systemic autoimmune disease, characterised by the production of autoantibodies against nuclear antigens that form immune complexes, driving tissue inflammation and organ damage. Despite its clinical heterogeneity and complex aetiology, SLE has a strong genetic basis, ranging from rare monogenic mutations to common polymorphic risk alleles. Studies of these variants have been substantially advanced our understanding of disease pathogenesis. In addition to autoantibody production, cytokine dysregulation, particularly elevated type I interferon (IFN-I), is a hallmark of SLE. However, the precise mechanisms by which IFN-I promotes autoreactive B-cell responses and autoantibody production remain incompletely understood. This talk will focus on our recent efforts to use mouse models that harbour patient-relevant point mutations in pathways that drive dysregulated IFN-I signalling to understand the genesis of autoreactive antibodies in SLE. Analysis of these models have revealed that, although spontaneous germinal centres formed, autoantibodies arose predominantly through extrafollicular responses. Furthermore, despite exhibiting broadly similar IFN-I dysregulation and autoimmune pathology, the models showed distinct requirements for IFN-I and TLR7 signalling in driving autoantibody production; finding that may have implication of targeted therapies.

Postgraduate Session II

Chair: Dr Adriana Pliego Zamora

Differential fluid and particle uptake by cells of the bone marrow

Vrushali Maste[1,2,3], Jintao Guo[2], Susan Millard[2], Allison Pettit[2], Jennifer Stow[1,3]

[1]Institute for Molecular Bioscience, The University of Queensland, Brisbane, Queensland Australia. [2]Mater Research Institute, The University of Queensland, Translational Research Institute, Brisbane, Queensland Australia. [3]ARC Centre of Excellence in Quantum Biotechnology – QUBIC, Australia.

Bone marrow (BM) is a complex tissue responsible for various critical functions including the production of blood cells, immune cells, and fat. Optical imaging of BM cells is currently thwarted by light scattering in the surrounding bone and our goal is to develop quantum-enhanced imaging of BM via near-infrared emitting nanoparticles to address that challenge. First, to optimise nanoparticle delivery to BM, we studied fluid phase uptake of fluorescent dextran by cells of the BM. We employed genetically modified murine models with CD169-expression driven fluorescence in macrophages. BM cell suspensions analysed by flow cytometry after *in vivo* or *ex vivo* incubation with TMR-dextran revealed that majority of fluid phase uptake was by reporter-positive macrophages with low levels of delivery into other haematopoietic cell types on the panel. For microscopy of dextran uptake *in vitro* we examined bone marrow derived reporter-positive macrophages confirming that dextran is internalized by macropinocytosis in these cells. To visualise dextran uptake in the BM setting, we then established extruded, intact BM plugs as a valuable *ex vivo* model for studying BM structure and function. These plugs retain morphologically intact functional niches, and microscopy reflects the dynamic and differential uptake of endocytosed dextran in cells therein, dominated by reporter- positive macrophages. T ogether these findings reveal that macrophages are the major proponents of fluid phase uptake in BM. Macrophages will thus be the targets for delivering nanoparticles into BM, ultimately providing a substantial reservoir of nanoparticles for optical imaging in deep bone environments. Improved optical imaging will enhance monitoring of BM and its niches in research and clinical settings.

Immunoediting Rewires the Tumour Microenvironment Toward Regulatory T Cell Dominance in UV-Induced Malignancies

Shoaib Anwaar[1], Hitesh Kore[2], James W Wells[1,3]

[1]Frazer Institute, Faculty of Health, Medicine and Behavioural Sciences, The University of Queensland, Brisbane, Queensland, Australia; [2]Department of Anatomy and Physiology, School of Biomedical Sciences, Faculty of Medicine, Dentistry and Health Sciences, The University of Melbourne, Parkville, VIC, Australia; [3]Dermatology Research Centre, Faculty of Health, Medicine and Behavioural Sciences, The University of Queensland, Brisbane, Queensland, Australia

Regulatory T cells (Tregs) often accumulate across malignancies to suppress anti-tumour immunity. Despite the established paradigm of cancer immunoediting which consists of elimination, equilibrium, and escape phases, the molecular cues driving Treg accumulation during tumour evolution remain unclear. UV-induced fibrosarcomas recapitulate all three stages of immunoediting in immunocompetent mice and provide a tractable and clinically relevant model to examine these alterations. Using fibrosarcoma regressor (41RE, 61RE) and their clonal progressor (41PRO, 61PRO) variants, we explored the cellular and molecular programs distinguishing immune-controlled from immune-evading
tumours. Bulk RNA-sequencing showed that PRO cells possess cell-intrinsic fitness advantage over RE cells with increased proliferative capacity and enhanced metabolic pathways such as glycolysis, oxidative phosphorylation, and hypoxia. In vivo, 41RE tumours were enriched in antigen-presentation and T-cell activation signatures. Conversely, 41PRO tumours upregulated cell-cycle and adhesion- associated programs. Critically, the transition to escape phase was accompanied by profound transcriptional rewiring dominated by retinoic acid signaling in 41PRO tumours with concurrent suppression of pro-inflammatory TNF, NF-κB, and IL-17 pathways. This tumour microenvironment remodeling promoted CD25+Foxp3+ Treg expansion and upregulated inhibitory immune checkpoints (TIGIT, PD-1, and LAG3) and key exhaustion-driving transcription factors (TOX and EOMES). Treg depletion with anti-CTLA-4 or anti-FR4 antibodies completely prevented 41PRO and 61PRO tumour
establishment. Furthermore, pairing Treg depletion with intratumoural IL-12 delivery synergistically boosted the proportion of $CD8^+$ T cells, increasing interferon-γ and TNF production and significantly reducing the tumour burden. Collectively, our findings demonstrate retinoic acid-driven reprogramming as a key mechanism of immune escape and highlight Treg-targeted strategies as promising interventions against immunoedited tumours.

Dissecting the Role of CD169+ Macrophages in Traumatic Spinal Cord Injury Using a Multi-Omics Approach

Bianciotto Capucine[1,3], Grice Laura F.[2,3], Lao Hong Wa[2], Carroll Sophia E.[1,3], Ruitenberg Marc J.[3]

[1]Faculty of Medicine, The University of Queensland, Brisbane, Australia; [2]School of Biomedical Sciences, The University of Queensland, Brisbane, Australia; [3]Translational Research Institute, Mater Research Institute, The University of Queensland, Brisbane, Australia

Traumatic spinal cord injury (SCI) triggers a complex inflammatory response that mediates both tissue damage and repair. Diverse myeloid immune cell populations dominate the spinal cord lesion site, but their individual role(s) remain poorly characterised. Here, we investigated the origin and dynamics of a subset infiltrating macrophages using a multi-modal approach. We first generated a single-cell RNA sequencing (scRNAseq) atlas of CD45+ immune cells isolated from the injured mouse spinal cord, with or without immune-modulatory therapy (IVIG). Subclustering identified 42 immune cell types, 38% of which showed significant changes in abundance in response to IVIG. Of these, CD169+ macrophages were amongst the most strongly affected, exhibiting pronounced shifts in both cell subtype abundance and gene expression. Using flow cytometry, we show that CD169+ cells are mobilised into the blood after SCI, with their numbers peaking at 1 day during the (sub)acute phase (day 0-7). Immunofluorescent staining at 1, 3, and 7 dpi further demonstrated recruitment of CD169+ macrophages to both the lesion core and penumbra regions. These results are consistent with the spatial distribution of CD169+ macrophages observed in our Visium spatial transcriptomics data. Transcriptomics data further showed that CD169+ macrophages adopt a pro-inflammatory profile and express genes associated with neuroinflammation and synaptic pruning. Together, our findings provide the first detailed characterization of CD169+ macrophages in SCI, and our ongoing experiments are now focused on delineating their functional contribution to neuroinflammation and synaptic remodeling.

Complement dependent enhancement of Dengue virus infection.

E. Gollasch-Miller [1], J. Kim [1], P. Vajjhala [1], H. Bielefeldt-Ohmann[1], T. Woodruff [2], E. A. Balmaceda [2], A. Pliego Zamora[1], K. J. Stacey [1]

[1]The University of Queensland, School of Chemistry and Molecular Biosciences and Australian Infectious Diseases Research Centre, St Lucia, Qld 4072, [2]Australia School of Biomedical Sciences, Faculty of Medicine, University of Queensland, St Lucia, QLD, 4072, Australia

Dengue virus (DENV) is an arbovirus that infects humans, and more than 50% of the world population is at risk of infection. DENV-infection can present as an asymptomatic infection, mild fever or develop into the life-threatening dengue hemorrhagic fever and/or dengue shock syndrome. The pathogenesis of severe dengue disease is complex, not well understood, and has been associated with increased activation of the complement system leading to dysregulation of its factors. However, the molecular mechanism and its involvement in pathology requires further investigation. DENV can enter and infect cells of monocytic lineage through a range of mechanisms, including receptor mediated endocytosis in which it can utilize a range of host cell receptors. Antibody dependent enhancement (ADE) is a process in which non-neutralizing antibodies will opsonise a pathogen and facilitate entry into the cell via the Fc receptor. This phenomenon is a significant issue with DENV infection and is suggested to contribute to the development of severe disease. Factors from the complement system also have opsonising functions which may be exploited for viral entry, similar to ADE. Here, using in-vitro assays of DENV infection in AG129 mouse bone marrow derived macrophages (BMMs) and human monocytes, we show that complement factors in serum significantly enhance infection. Infecting cells with DENV and non-heat inactivated serum, results in enhancement of infection in both mouse BMMs and human monocytes. Whereas heat inactivation of the serum reduces the enhancement effect. Moreover, treating the serum with a selective complement inhibitor results in reduction of the enhancement of infection. In conclusion, the data suggests that there are factors in non-heat inactivated untreated serum which enhance DENV infection. These factors are likely to be complement proteins due to their heat instability and loss of activity when treated with a selective inhibitor of complement.

The Impact of Age-Related Decline in the Unfolded Protein Response on Respiratory Infection Susceptibility and Severity

Thishan Illankoon[1,2], Kuan Yau Wong[1,2], Alexandra Mueller[1,2], Lucy Burr[1,2,3], Sumaira Hasnain[1,2,4]

[1]Mater Research Institute (Translational Research Institute), Brisbane Queensland, Australia; [2]University of Queensland, Brisbane Queensland, Australia; [3]Department of Respiratory and Sleep Medicine, Mater Health, South Brisbane, Australia; [4]Australian Infectious Diseases Research Centre, The University of Queensland, Brisbane, Australia.

Accumulation of misfolded protein induces endoplasmic reticulum (ER) stress, which is resolved by activation of the unfolded protein response (UPR). While increased protein misfolding is recognised as a feature of aging, how age-associated decline in UPR function contributes to respiratory epithelial dysfunction remains unclear. We hypothesise that age-related UPR senescence contributes to increased susceptibility and severity of respiratory infections in the elderly population. To investigate this, we used Human Bronchial Epithelial Cells (HBECs) from individuals aged between 25-66 years old, and utilised mouse models aged 4-96 weeks old. Additionally, lung organoids from mice of various ages were established to explore ER stress responses and their role in infection management across aging. Our results revealed significant downregulation of various key UPR factors throughout aging in murine RECs. Lung organoids from 8- and 60-week-old mice were stimulated with tunicamycin or IFNy+TNFa which displayed a reduced ability to activate the UPR to manage ER stress in older age. Additionally, IFNy stimulation in RECs increased intracellular MHC-II expression, however, surface expression required co-stimulation with both IFNy and TNFa. Using DQ-Ova (model antigen), we observed that RECs can uptake, process, and transfer antigen to immune cells, in human cell lines and murine lung organoids. Lung organoids from aged mice had an elevated uptake and processing of DQ-Ova which was mediated by ER stress and the UPR. Cytokine stimulation in HBECs from patients of various ages also indicated a hyperactivation of MHC-II expression in older age, potentially contributing to inappropriate infection responses. Our findings reveal that aging diminishes the capacity of RECs to activate the UPR and alters antigen presentation capacity. Given their ability to process and transfer antigens, RECs present as key players in immunity, and their impairment with age may contribute to the increased infection susceptibility and severity in older individuals.



Jonathan Sprent Oration

Chair: Professor Rajiv Khanna

Sponsored by Sanofi

sanofi

Towards an HIV cure: the role of anti-PD1, mRNA and other immunotherapies

Sharon Lewin

The Peter Doherty Institute for Infection and Immunity, The University of Melbourne, Victorian Infectious Diseases Service, The Royal Melbourne Hospital and Department of Infectious Diseases, Alfred Hospital and Monash University, Melbourne, Australia

Despite the great success of antiviral therapy (ART), treatment is lifelong for the majority of people living with HIV (PLWH). The main reason ART is unable to cure HIV infection is the persistence of long lived proliferating latently infected cells and impaired clearance of HIV-infected cells as a result of T-cell exhaustion. One strategy to allow safe cessation of ART is to 'reduce and control' the reservoir through boosting HIV-specific immunity to both clear latently infected cells and maintain long term control of any residual virus. Over the last 15 years, my team have explored a range of strategies to boost HIV-specific immunity including immune modulating agents such as pomalidomide, broadly neutralising antibodies and immune checkpoint blockade, specifically anti-PD1. Using single cell RNAseq of both total and HIV-specific CD8+ T-cells collected from people with HIV on ART and cancer who were participating in two separate prospective clinical trials of anti-PD1 (prembrolizumab or nivolumab), we showed that anti-PD-1 significantly reshapes the T-cell landscape. In HIV-specific tetramer positive CD8+ T-cells, following nivolumab, we saw the emergence of a distinct central memory cluster characterized by an interferon-stimulated gene signature together with an increase in expression of Granzyme-H in expanded clones with identical T-cell receptors, highlighting a potential pathway to reinvigoration of cytotoxic function. We are now recruiting healthy people with HIV on ART in Melbourne and in Singapore to a clinical trial of a single low dose of nivolumab to minimise severe immune related adverse events and enhance immune-mediated contol. To allow for sufficient antigen presentation while on ART, we have also recently developed two novel approaches to reverse HIV latency using mRNA delivered by a highly T-cell tropic lipid nanoparticle. We used mRNA to deliver either a viral protein called Tat or HIV-specific CRISPR activation with high potency in vitro and in animal models and demonstrated efficient expression of viral protein. Our long term goal is to identify a combination of interventions that allows for a person to safely stop ART through leveraging exciting advances in other fields, including cancer immunotherapy and mRNA therapeutics.



The FIRST manual pipette that repeat dispenses.

This familiar lab tool is anything but ordinary. With INTEGRA's SWITCH you choose; pipette manually or 'SWITCH' to repeat dispense mode and gain productivity while enjoying the lowest plunger force possible.

INTEGRA SWITCH

SEE IT IN ACTION >



Postgraduate Plenary Lecture II

Chair: Associate Professor Katharine Irvine

INTEGRA
BIOSCIENCES

Harnessing a new checkpoint to control auto-immunity in lupus patients.

Michael Gantier

Nucleic Acids and Innate Immunity Laboratory, Hudson Institute of Medical Research, Monash University, Melbourne (VIC) Australia

While Toll-like receptors 7 and 8 (TLR7/8) are critical for initiating innate immunity against pathogens, how they avoid activation by the abundance of host RNAs generated during routine apoptotic clearance has long remained an enigma. We recently solved this mystery by demonstrating that TLR7/8 are naturally antagonized by endogenous ribosomal RNA fragments containing 2'-O-methyl (2'-OMe) guanosine (Alharbi et al., Nature Immunology 2026). Structural determination of TLR7 complexed with these natural antagonists revealed F507 as the key gatekeeper residue governing this interaction, a finding underscored by our discovery that F507 mutation drives systemic lupus in human patients. Building on this natural immune checkpoint, we developed a 3-base therapeutic oligonucleotide that potently antagonises TLR7 and TLR8, that successfully blunts inflammation in preclinical skin lupus models, and has recently completed a Phase 1 clinical trial for topical skin lupus management. Together, our work redefines the paradigm of self-RNA recognition and provides a direct, structurally-guided therapeutic framework for the clinical translation of novel autoimmune therapeutics.

BD



COMING SOON!

BD FACSDiscover™ A7 Cell Analyser

The new standard of spectral flow cytometry for all.

LEARN MORE

bdbiosciences.com/en-au

BD is a trademark of Becton, Dickinson and Company. Waters is a trademark of Waters Corporation or its affiliates. All other marks are property of their respective owners. © 2026 BD. All rights reserved.

Waters Corporation has acquired BD Biosciences. BD remains the legal manufacturer until all required regulatory transfers are completed. Product and product information provided here is supplied under regulatory authority of BD. Learn more: www.waters.com/bdtransaction

Early/Mid Career Researcher Session

Chair: Associate Professor Kate Gartlan

Waters™

Biosciences

Formerly BD Biosciences

First Cells to First Communities: A Multiomic Atlas of the Early-Life Gut

Amanda Oliver

Barrier Immunity Laboratory, Infection and Inflammation Program, QIMR Berghofer Medical Research Institute, Brisbane (QLD) Australia

Bio: Dr. Amanda Oliver is Team Head of the Barrier Immunity Lab, at QIMR Berghofer. She completed her PhD in tumour immunology at the Peter MacCallum Cancer Centre and was a postdoctoral fellow in Professor Sarah Teichmann's lab at the Wellcome Sanger Institute, supported by a Marie Skłodowska-Curie fellowship. Her research focuses on mucosal immunity, with publications in *Nature* and *Nature Genetics* highlighting epithelial-driven immune niches and non-traditional roles of epithelial cells in adaptive immunity. Her lab uses single-cell and spatial genomics alongside patient-derived systems to study inflammatory diseases of mucosal tissues.

Abstract: Early life represents a critical period for intestinal development, shaped by extreme dietary changes, microbiome establishment, and rapid tissue maturation. We are generating a multiomic atlas of the human early-life intestine by integrating single-cell and spatial transcriptomics, metagenomics, and organoid models. Our dataset comprises 34 newly profiled donors under five years of age, including rare neonatal samples, together with 11 published datasets, creating a reference atlas of approximately 1.2 million cells spanning foetal to paediatric development. This ongoing work seeks to define the cellular programs underlying epithelial and immune maturation, characterise early-life host-microbe interactions, and establish a foundational resource for studying intestinal development and disease susceptibility.

Feeding the inflammasome to damage the liver: new insights into Caspase-1 biology

Sabrina Sofia Burgener[1], Mark Thomas Milner [1], Shoumit Dey [2], Pooranee K. Morgan[3], Daniel G Blackmore [5], Emmanuelle Frampton[1], Gregory Miller[6,7], Monalisa Duarte de Oliveira[1], Kirsten Myra Kenney[1], Rinie Bajracharya[1], Ulrich Baumgartner[8], Thomas J.C Collins[3], Zherui Xiong[1], Quan Nguyen[1], Peter J. Meikle[3,4], Liviu-Gabriel Bodea[5,8], Andrew Clouston[6,7], Dave Boucher[9], Jürgen Götz[5], Andrew J. Murphy[3], Paul M Kaye[2], Kate Schroder[1]

[1]Institute for Molecular Bioscience and IMB Centre for Inflammation and Disease Research, The University of Queensland, St Lucia 4072, Australia; [2]Hull York Medical School and York Biomedical Research Institute, University of York, York, UK; [3]Baker Heart and Diabetes Institute, 75 Commercial Road, Melbourne, VIC 3004, Australia; [4]Baker Department of Cardiovascular Research, Translation and Implementation, La Trobe University, Bundoora, VIC, 3086, Australia; [5]Clem Jones Centre for Aging and Dementia Research, Queensland Brain Institute, Faculty of Health, Medicine and Behavioural Sciences, The University of Queensland, St. Lucia, 4072, Australia; [6]Envoi Specialist Pathologists, Kelvin Grove 4059, Australia; [7]Health, Medicine and Behavioural Sciences, The University of Queensland, Herston 4006, Australia; [8]School of Biomedical Sciences, Faculty of Health, Medicine and Behavioural Sciences, The University of Queensland, St Lucia 4072, AustraliaDepartment of Biology, York Biomedical Research Institute, University of York

Caspases act as sharp scissors that cut protein substrates, modulating protein-protein interactions, creating new bioactive molecules and inducing inflammation. Such critical reactions must be strictly regulated. To date, the molecular mechanisms that silence proteases to prevent disease progression are poorly understood. Active caspase-1 (CASP1) cleaves IL-1 family cytokines (e.g., IL-1β, IL-18) into their bioactive forms, thereby inducing inflammation. Over the last two decades, most research has defined activation signals for CASP1 and its catalytic function in substrate processing. Little is known about how CASP1 regulates its activity to ensure timely signal inhibition. Here, we show that autoproteolysis terminates endogenous CASP1 activity in vivo. We generated a knock-in mouse (Casp1.CDL) in which the caspase-1 CARD domain linker (CDL) is mutated to prevent self-cleavage and examined these mice under steady-state conditions and during major physiological challenges. Stimulation of macrophages from Casp1.CDL mice confirmed heightened CASP1 activity and substrate processing. Similarly, in a mild endotoxic challenge model, Casp1.CDL mice exhibit increased circulating cytokine levels and heightened CASP1 activity at the injection site. In a diet-induced liver disease model, the CASP1 CDL mutation accelerated liver steatohepatitis and damage by upregulating IL-1R signalling pathways. Importantly, CASP1 CDL mutation delayed disease resolution during liver healing, and people living with chronic liver disease presented with higher circulating inflammatory cytokine levels. Thus, CASP1 CDL self-cleavage terminates CASP1 inflammatory programs, maintaining steady-state tissue homeostasis, suppressing inflammasome-driven diseases, and restoring homeostasis after a major challenge to organ function in mice and humans. Revealing when and how CASP1 CDL cleavage occurs is pivotal for our understanding of the molecular mechanism by which this protease is silenced.

Pre-clinical assessment of mRNA vaccine-induced immunity to Group A *Streptococcus*

Ismail Sebina[1], Rhiannon A. Bolton[1], Isabel G. Courtney[1], Nichaela Harbison-Price[2] ,Meredith Finn[3], Amanda J. Cork[2], Johanna Richter[2], Laura Davis[2], Brody Pullinger[2], Nia Bickham[2], Miguel Aguirre[2], Obadiah Plante[3], Christina Dold[3], Mark J. Walker[2], Gabrielle T. Belz[1]

[1]Frazer Institute, The University of Queensland, Brisbane, Queensland, Australia. [2]Centre for Superbug Solutions, Institute for Molecular Bioscience, The University of Queensland, Brisbane, Queensland, Australia. [3]Moderna TX, 200 Technology Square, Cambridge, Massachusetts, USA.

Developing an effective vaccine against Group A *Streptococcus* (Strep A), the pathogen that causes rheumatic heart disease, could prevent over 500,000 deaths worldwide each year. However, progress to vaccine development is hampered by safety concerns surrounding antigen targets, a lack of validated preclinical models, and an incomplete understanding of immune correlates of protection. To address these barriers, we developed Combo#5-TM, a multivant mRNA vaccine encoding five conserved Strep A antigens (SLO, SCPA, SpyCEP, TF, and ADI) and recently demonstrated its protective efficacy in mice. Here, we define the phenotypic profile, temporal dynamics and durability of Combo#5-TM-induced B and T cell responses using high-dimensional flow cytometry and Luminex-based serology. Following intramuscular immunisation (days 0, 21, 28), Combo#5-TM elicited diverse memory B and T cell populations in the spleen and draining lymph nodes, which persisted for at least 16 weeks. Memory B cells included both IgM+ and class-switched subsets, localised within follicular and marginal zones. Functionally relevant memory B cell subsets, PDL2+, CD80+, double-positive (PDL2+CD80+), and double-negative (PDL2-CD80-), were induced, with the CD80+ and PDL2-CD80- subsets dominating over time. Combo#5 also generated long-lived plasma cell responses, evidenced by sustained antigen-specific antibody titres. T cell responses were dominated by CD8+ effector memory cells, including CD127hiKLRG1hi effectors and CD127hiKLRG1lo memory precursor cells, both of which produced IFNγ and TNFα upon antigen restimulation. Together, these findings demonstrate that Combo#5-TM mRNA vaccination induces durable, phenotypically diverse, and functionally competent B and T cell memory, supporting its further development as a next-generation vaccine against Strep A.

Mucosal recombinant BCG vaccination overcomes safety barriers in immunocompromised hosts and protects against TB through innate immune priming.

Harindra Sathkumara[1], Guangzu Zhao[1], Munish Puri[1], Julia Seifert[1], Andrew Calcino[1,2], Socorro Miranda-Hernandez[1], Yide Wong[1], Matt Field[1,2], Roland Brosch[3] and Andreas Kupz[1]

[1]Australian Institute of Tropical Health and Medicine, James Cook University, Cairns and Townsville, Queensland, Australia.[2]Centre for Tropical Bioinformatics and Molecular Biology, James Cook University, Townsville, Queensland, Australia. [3]Unit for Integrated Mycobacterial Pathogenomics, Institut Pasteur, Paris, France.

Tuberculosis (TB) remains one of the leading infectious causes of death worldwide, with immunocompromised individuals particularly susceptible to severe disease and poor vaccine-mediated protection. Bacillus Calmette-Guérin (BCG), the only licensed TB vaccine, provides inconsistent protection against pulmonary TB in adults and offers limited efficacy in vulnerable populations. Although recombinant BCG (rBCG) vaccines engineered to express immunodominant *Mycobacterium tuberculosis* (*Mtb*) antigens such as ESAT-6 have demonstrated superior protection in experimental models, their clinical development has been constrained by safety concerns. In highly sensitive immunodeficient mouse models, high-dose intravenous administration of these recombinant strains caused extensive bacterial dissemination, severe pathology, and mortality. In this study, we demonstrate that the route of vaccine administration fundamentally reshapes both the safety and efficacy of ESAT-6-expressing rBCG vaccines. Using multiple immunocompromised mouse models, including adaptive-deficient $Rag1^{-}/^{-}$ and $Rag2^{-}/^{-}\gamma c^{-}/^{-}$ mice, we directly compared traditional intravenous vaccination with mucosal intratracheal delivery. In striking contrast to intravenous administration, high-dose mucosal vaccination was markedly better tolerated, significantly improved survival, and greatly reduced systemic dissemination and brain pathology, even under profound immunodeficiency. Importantly, mucosal vaccination not only improved safety but also enhanced protection against aerosol *Mtb* infection compared with standard BCG vaccination. ESAT-6-expressing rBCG strains, particularly BCG::RD1, induced heightened innate immune activation within the respiratory tract and promoted expansion of myeloid progenitors in the bone marrow. Vaccinated mice also exhibited enhanced antimycobacterial activity in bone marrow-derived macrophages, consistent with trained innate immunity. Together, these findings demonstrate that mucosal delivery of ESAT-6-secreting BCG strains safely induces durable innate immune training, enabling effective protection against *Mtb* even in the complete absence of adaptive immunity. These findings suggest that prior intravenous models may prematurely exclude otherwise promising vaccine candidates, and challenges prevailing safety assumptions surrounding ESAT-6-expressing vaccines and positions rBCG candidates as promising strategies for protecting vulnerable populations where TB risk is highest.



Take your research to the next level







Translational Research Institute (TRI) is a leading Australian medical research, development and translation facility. Our commitment to innovation and emerging technologies provides researchers with access to world-class capabilities and support through our highly specialised Core Facilities and expert technical teams.

TRI's specialist teams provide tailored solutions across our integrated **Core Facilities**: Gnotobiotics, Human Immune Model, Flow Cytometry, Histology, Microscopy, Preclinical Imaging and T3 Cleanrooms, supporting researchers from experimental design through to analysis and translation.

TRI's **Gnotobiotics and Human Immune Model Facilities** are the first of their kind in Australia, providing ready access to gnotobiotic and humanised mice to support reliable, clinically relevant research outcomes.

Access a versatile platform capable of supporting innovative research across vaccine platforms, infectious diseases, autoimmunity, oncology, and the microbiome.

Contact us to discuss TRI's full range of capabilities and support services.

- **Humanised models:** Gold-standard NSG strains engrafted with a functional human immune system.
- **Gnotobiotic strains:** Colonised with defined microbiota and customised to experimental requirements.
- Experimental design and bespoke research services.
- Specialised technical services, support, and expertise.

AUTHORIZED DISTRIBUTOR OF

The Jackson Laboratory

Kelly-Anne Masterman, PhD
Core Facilities Engagement Officer
kellyanne.masterman@tri.edu.au
+61 7 3443 7002

Want to know more about TRI's Core Facilities? Visit

tri.edu.au/facilities

BIG Plenary Lecture II

Chair: Associate Professor Katharine Irvine

Ferroptosis: a new dimension in governing T cell function

Di Yu

[1]Ian Frazer for Children's Immunotherapy Research, Children's Health Research Centre, Faculty of Health, Medicine and Behaviour Sciences, The University of Queensland, Brisbane, Queensland, Australia; [5]Frazer Institute, Faculty of Health, Medicine and Behaviour Sciences, The University of Queensland, Brisbane, Queensland, Australia

Abstract:
Ferroptosis, a major mechanism of non-apoptotic programmed cell death, critically regulates the homeostasis and functionality of peripheral $CD4^+$ and $CD8^+$ T cells, thus controlling follicular helper T (T_{FH}) cell-dependent humoral immunity and $CD8^+$ T cell-mediated anti-tumour immunity. Resistance to ferroptosis in T cells is strongly influenced by dietary composition in mice and systemic metabolism in humans. These dietary effects on ferroptosis (DEF) in T cells shape both humoral immunity (e.g., vaccination) and cellular immunity (e.g., cancer immunotherapy). Mechanistically, DEFs are microbiota-independent and are driven by variations in dietary polyunsaturated and monounsaturated fatty acids (PUFAs and MUFAs) that lead to variations in abundance of lipid species in lymphoid tissues and T cells. These findings highlight the physiological importance of metabolic regulation in T cell ferroptosis and suggest new strategies to enhance vaccine efficacy and T cell-based immunotherapies by targeting ferroptosis and metabolic pathways.

BIG Session I

Chair: Dr Adriana Pliego Zamora

Microglial-intrinsic complement signalling drives neurodegeneration

Trent Woodruff

School of Biomedical Sciences, University of Queensland, Brisbane (QLD) Australia

Neuroinflammation is increasingly recognised as a key driver of neurodegenerative disease progression. While the complement system is traditionally viewed as an extracellular mediator of innate immunity, emerging evidence suggests that complement receptors can also regulate cell-intrinsic inflammatory pathways within the central nervous system. Our work has identified the complement receptor C5aR1 as a critical regulator of microglial activation in neurodegenerative disease. Using primary microglia, mouse models, human tissues and stem-cell-derived organoid systems, we have demonstrated that C5aR1 signalling is required for full activation of the NLRP3 inflammasome in response to disease-relevant stimuli, including α-synuclein aggregates. Genetic and pharmacological inhibition of C5aR1 suppresses inflammasome activation, reduces neuroinflammation and protects against neurodegeneration in preclinical models of Parkinson's disease. These findings reveal a previously unrecognised complement–inflammasome signalling axis that links innate immune activation to neuronal injury. This presentation will discuss the emerging role of microglial-intrinsic complement signalling in neurodegeneration and its implications for understanding how innate immune pathways drive disease progression.

Transcriptional drivers of alloreactive CD8⁺ T cell responses in donor stem cell transplantation

Amir Shamshirian,[1, 2] Karshing Chang,[1] Graham Magor,[1] Yumy Zeng,[1] Jessica Engel,[1] Christian Engwerda,[1] Katie E. Lineburg,[1] **Kate H. Gartlan[1,2]**

[1]Infection & Inflammation Program, QIMR Berghofer, 300 Herston Road, Herston QLD 4006; [2]School of Biomedical Sciences, The University of Queensland St Lucia QLD 4072

Immune checkpoint pathways are widely accepted to limit T cell function during persistent inflammation and antigen exposure. However, we have identified a distinct subset of alloreactive CD8⁺ T cells in graft-versus-host disease (GVHD) that appears to defy this paradigm. In murine allogeneic stem cell transplantation (alloSCT), we observe that the most phenotypically exhausted CD8⁺ T cells co-express the highest levels of effector molecules and inflammatory cytokines. Single-cell RNA sequencing (scRNA-seq) identified Batf and Bhlhe40 as highly expressed in this population, implicating these factors in the maintenance of pathogenic effector activity despite inhibitory signalling. Parallel scRNA-seq analysis of human CD8⁺ T cells demonstrated high BHLHE40 expression in cells undergoing proliferative bursts coincident with GVHD onset post-transplant. Genetic deletion of Bhlhe40 in donor CD8⁺ T cells profoundly reprogrammed their differentiation trajectory, limiting progression into terminal effector states and enriching TCF1⁺ progenitor-like phenotypes. Bhlhe40-deficient CD8⁺ T cells produced more IL-10 and fewer pro-inflammatory cytokines, consistent with a less pro-inflammatory state. Functionally, Bhlhe40 deficiency reduced GVHD severity despite sustained donor T-cell expansion. Consistently, CRISPR-mediated Bhlhe40 editing of the donor graft significantly reduced lethal GVHD in vivo. Importantly, preliminary data indicate donor Bhlhe40 deficiency preserved GVL activity against primary MLL-AF9 acute myeloid leukaemia in vivo, suggesting Bhlhe40 may be selectively required for GVHD-driving inflammatory differentiation but dispensable for anti-leukaemic function. Together, these data identify Bhlhe40 as a transcriptional checkpoint integrating CD8⁺ T-cell fate determination, cytokine programming, and pathogenicity in alloimmunity. Targeting Bhlhe40 may therefore offer a strategy to reduce GVHD while preserving GVL activity.

A meningococcal vaccine against gonorrhoea: Insights from recent clinical trials

Kate L Seib[1], Basil Donovan[2], Fengyi Jin[2], Caroline Thng[1,3], Barbara Yeung[2], Nicholas Comninos[4], Rohan I Bopage[4], Anik Ray[5], Alison Mahony[5], Eric PF Chow[6,7,8], Marcus Y Chen[6,7], Kate Maddaford[6], Helen Lau[9], Portia Westall[2], Dan Lu[2], Benjamin R Bavinton[2], Kathy Petoumenos[2], David M Whiley[10], Michael P Jennings[1], Amy V Jennison[11], John Kaldor[2], Rebecca Guy[2], Monica M Lahra[12,13], Jane Costello[14], Brent Mackie[15], David J Templeton[2,16], Beng Eu[9], Mark O'Reilly[17], Christopher K Fairley[6,7], Anna McNulty[5,18], Rick Varma[2,5], David A Lewis[4,19], Andrew E Grulich[2]

[1]Institute for Biomedicine and Glycomics, Griffith University, Gold Coast, QLD, Australia [2]The Kirby Institute, University of New South Wales, Sydney, NSW, Australia [3]Gold Coast Sexual Health Service, Southport, QLD, Australia [4]Western Sydney Sexual Health Centre, Parramatta, NSW, Australia [5]Sydney Sexual Health Centre, Sydney, NSW, Australia [6]Melbourne Sexual Health Centre, Alfred Health, Melbourne, VIC, Australia [7]School of Translational Medicine, Faculty of Medicine, Nursing and Health Sciences, Monash University, Melbourne, VIC, Australia [8]Melbourne School of Population and Global Health, The University of Melbourne, Melbourne, VIC, Australia [9]Prahran Market Clinic, Melbourne, VIC, Australia [10]UQ Centre for Clinical Research, Faculty of Medicine, The University of Queensland, Brisbane, QLD, Australia [11]Public and Environmental Health, Pathology Queensland, Queensland Health, Brisbane, QLD, Australia [12]WHO Collaborating Centre for STI and AMR, NSW Health Pathology Microbiology, The Prince of Wales Hospital, Sydney, NSW, Australia [13]The University of New South Wales, Sydney, NSW, Australia [14]Positive Life NSW, Sydney, NSW, Australia [15]ACON Health, Sydney, NSW, Australia [16]RPA Sexual Health, Department of Sexual Health Medicine, Community Health, Sydney Local Health District and Central Clinical School, Faculty of Medicine and Health, University of Sydney, Sydney, NSW, Australia [17]Taylor Square Private Clinic, Sydney, NSW, Australia [18]School of Population Health, University of New South Wales, Sydney, NSW, Australia [19]Sydney Medical School-Westmead, Faculty of Medicine and Health and Sydney Infectious Diseases Institute, University of Sydney, Westmead, NSW, Australia

Background: No vaccines are licensed for *Neisseria gonorrhoeae* prevention. However, a meta-analysis of observational studies reported that a meningococcal B vaccine, 4CMenB, is associated with a 38% reduced risk of incident *N. gonorrhoeae* infection. **Methods:** We conducted a multicenter, randomised, double-blind, placebo-controlled trial in men who have sex with men (MSM) aged 18–50 years. Participants were either HIV-negative and taking HIV PrEP, or living with HIV, and had a documented diagnosis of gonorrhoea or infectious syphilis within the preceding 18 months. They were randomly assigned 1:1 to receive two doses 3 months apart of 4CMenB or placebo. STI screening including *N. gonorrhoeae* nucleic acid amplification tests on specimens from urogenital, anorectal, and oropharyngeal sites were performed quarterly over two-years. The primary outcome was incidence of a first-episode *N. gonorrhoeae* infection in the per-protocol population, occurring at least four weeks after administration of the second dose. Secondary outcomes included symptomatic, asymptomatic, and site-specific infections. **Results:** During 2021-23, 654 participants were randomised, with 587 included in the primary analysis. The incidence of first-episode *N. gonorrhoeae* was 48.1 per 100 person-years (160 events among 296 participants) in the 4CMenB group and 47.8 per 100 person-years (155 events among 291 participants) in the placebo group (incidence rate ratio 1.01, 95% confidence interval [CI] 0.80-1.26, p=0.97), for a vaccine efficacy of –0.5% (95% CI -26.16%-19.93%). Vaccine efficacy for symptomatic infection was 5.50% (95% CI -56.02%-42.82%); asymptomatic infection, -6.36% (95% CI -47.47%-23.19%); and urogenital, anorectal, and oropharyngeal infections, -20.00% (95% CI -

90.24%-23.86%), -1.18% (95% CI -32.95-22.97%), and 2.58% (95% CI -27.67% to 25.65%), respectively. The incidence of serious adverse events did not differ significantly between groups. **Conclusion:** 4CMenB vaccine did not reduce *N. gonorrhoeae* incidence among MSM at high risk of gonorrhoea.



QUEENSLAND IMMUNOLOGY RESEARCH CENTRE

Pfizer

Australian and New Zealand SOCIETY FOR IMMUNOLOGY INC.

QIAGEN

10X GENOMICS

Distributed by millennium® science

UNITED BIORESEARCH

QIMR Berghofer

THE UNIVERSITY OF QUEENSLAND AUSTRALIA

Miltenyi Biotec

Lunch and QIRC-Sponsored Mentoring session

Mentoring Session

Sponsored by: QIRC

Being a Women in Immunology - Swimming Upstream (15 mins)

Dr Debbie Watson, University of Wollongong

QIRC and QIRC Opportunities (15 mins)

Professor Gabrielle Belz, Frazer Institute, University of Queensland

ASI Opportunities and awards (10 mins)

A/Prof Danielle Stanisic, Griffith University

Being a woman in immunology – swimming upstream

Debbie Watson[1]

[1]School of Science and Molecular Horizons, Faculty of Science Medicine and Health, University of Wollongong

Being a woman had a major impact on my career development and research progress in immunology. I will discuss the impacts of being a woman and gender equity, the impact of career disruption and working part-time, and other impacts while talking about my career and research into blood cancer. My research focuses on blood cancer and donor stem cell transplantation. The goal of donor stem cell transplantation is to reconstitute the immune system and control any residual cancer cells in the recipient, which is known as the graft-versus-leukaemia (GVL) response. However, the donor cells (graft) can also mount a response against the recipient (host) and attack different organs including the skin, gut and liver, which is known as graft-versus-host disease (GVHD). My career led to me becoming a group leader of the Genetics and Immunology Research laboratory (GIRL) and establishing preclinical humanised mouse models of GVHD and GVL to target immune cell pathways, including specifically targeting proliferating T cells, using purinergic signalling (P2X7) blockade and targeting proinflammatory cytokines. I will briefly discuss my research, while I focus on my journey of being a woman while building my research career in immunology. Finally I will discuss the impact of being a carer and the role of gender equity in research funding and career development and acknowledge women who have inspired me in my career and my life.



BIG Session II

Chair: Dr Carla Proietti

Interleukin-24 as a Central Regulator of Intestinal Fibrosis

Sumaira Hasnain

Translational Research Institute, University of Queensland, Brisbane (QLD) Australia

Abstract: Chronic inflammatory bowel disease (IBD) is characterised by persistent immune activation and progressive tissue remodelling that can culminate in intestinal fibrosis. Although current therapies effectively suppress inflammation, they have had limited success in preventing fibrosis, suggesting that distinct immune mechanisms drive tissue remodelling. We investigated the role of the IL-20 family cytokine interleukin-24 (IL-24) in regulating immune-stromal interactions during intestinal fibrosis. Using multiple preclinical models of IBD and human patient samples, we found that IL-24 expression increased with disease progression and correlated with fibrosis severity. Neutralisation of IL-24 consistently reduced collagen deposition and fibrosis, with only modest effects on inflammation. Mechanistically, IL-24 produced by fibroblasts and macrophages reprogrammed fibroblasts into a secretory state that promoted epithelial activation. Rather than directly activating transforming growth factor-β (TGF-β), IL-24 amplified TGF-β-dependent tissue remodelling through reactive oxygen species, establishing a feed-forward circuit between immune, stromal, and epithelial cells. These findings identify IL-24 as an upstream regulator of tissue immunity and epithelial-stromal communication, highlighting a potential therapeutic strategy for preventing fibrosis in chronic intestinal inflammation.

Long-lived CD8αα intestinal intraepithelial T cells persist despite restrained antigen receptor signaling and are shaped by chronological aging and microbial experience

K. Miles[1], M. Smith[1], MS. Strine[2], SD. O'Flanagan[3], E. Gonzales Santiago[2], S. Gillert[1], M. Puri[1], A. Kupz[1], L. Mackay[4], L. Konnikova[2], and **R. Ruscher[1]**

[1]Australian Institute of Tropical Health and Medicine (AITHM), James Cook University, Cairns&Townsville, QLD, Australia; [2]Yale School of Medicine, New Haven, CT, USA
[3]Center for Immunology, Department of Laboratory Medicine and Pathology, University of Minnesota, MN, USA

CD8αα intestinal intraepithelial lymphocytes (IELs) are unconventional cytotoxic T cells implicated in gut homeostasis, yet their long-term maintenance and relevance in humans remain incompletely understood. To further define this lineage, we conducted high-parameter machine learning-assisted surface protein profiling and single cell transcriptomics, which suggested restrained TCR signalling compared with conventional CD8 T cells. Consistently, in *Nr4a1*^{eGFP} reporter mice, antigen receptor engagement robustly increased TCR signal strength in conventional T cells but not in CD8αα IELs. Despite restrained TCR signalling profile, fate mapping demonstrated maintenance of early life generated CD8αα IELs into old age, while parabiosis studies confirmed sustained tissue residency. Functionally, aging selectively reduced IFNγ production and diminished in vitro killing of MC38 colorectal cancer cells by CD8αα IELs, indicating constrained effector potency in late life. Guided by our murine profiling, we identified a CD8αα-like IEL population in the human ileum that is abundant in neonates, declines proportionally during early childhood, but remains detectable in adults. A similar pattern was observed in our Normalised Microbial Experience ("dirty") mice – a model that more closely models adult human immunity. In these mice, CD8αα IELs were proportionally reduced – driven by expansion of conventional T cells – yet remained numerically stable, when compared to conventional SPF laboratory mice. Importantly, CD8αα IELs in "dirty" mice displayed a phenotypic shift suggestive of enhanced activation, implying functional adjustments in microbially rich environments. Together, our findings suggest that CD8αα IELs constitute a long-lived lineage that exists in humans and whose effector capacity is impacted by aging and microbial experience, despite exhibiting restrained TCR signal transduction.

Circulating CD5⁺ dendritic cells link gut microbiome function to immunotherapy response in cancer patients

Janin Chandra

Translational Research Institute, University of Queensland, Brisbane (QLD) Australia

While immune checkpoint inhibitors (ICIs) have transformed oncology, the determinants of response beyond tumour-intrinsic features remain incompletely understood, particularly the contributions of systemic immunity and the microbiome. Here, in a prospective study of 82 immunotherapy-naïve patients across five solid tumour types, we integrated longitudinal deep immunophenotyping of peripheral blood with gut metagenomic and oral 16S rRNA sequencing. Elevated baseline frequencies of CD5⁺ conventional dendritic cells type 2 (cDC2) and attenuated B cell activation independently predicted clinical benefit, with sustained DC enrichment further associated with durable disease control. Gut metagenomic analysis identified an 11-species responder-associated signature and congruent enrichment of *Faecalibacterium* MIC7145 at both taxonomic and functional levels. Community-level microbial pathways, including peptidoglycan, UMP, and aromatic compound biosynthesis, correlated with CD5⁺ cDC2 abundance but not with individual species, suggesting that the microbiome's collective metabolic output may help maintain a priming-competent immune state. Although the oral microbiome was not associated with response, a distinct periodontopathogen signature predicted severe immune-related adverse events. These findings identify CD5⁺ cDC2 as a cornerstone of the antigen-presenting checkpoint and reveal a functional gut microbiome–immune axis that may be exploited to enhance immunotherapy.





START SMART

2026

Start smart with the new gentleMACS™ Octo Dissociator accessory kit promotion.

For the month of August order a gentleMACS Octo with Heaters Dissociator and receive a free accessory bundle of either 130-128-752 gentleMACS Perfusion sleeves (4 pieces) valued at $1289.00 or 130-130-533 gentleMACS Octo coolers (4 pieces) valued at $1581.00.
Visit us at our table and speak to Azadeh Seidi to receive this offer.

▶ **miltenyibiotec.com**

Miltenyi Biotec Australia Pty. Ltd. | Unit 11, 2 Eden Park Drive | Macquarie Park, NSW 2113 | Australia | Phone +61 2 8877 7400 | Fax +61 2 9889 5044 | macsau@miltenyi.com | **www.miltenyibiotec.com**

Miltenyi Biotec provides products and services worldwide. Visit **www.miltenyibiotec.com/local** to find your nearest Miltenyi Biotec contact.

Unless otherwise specifically indicated, Miltenyi Biotec products and services are for research use only and not for therapeutic or diagnostic use. gentleMACS, MACS, and the Miltenyi Biotec logo are registered trademarks or trademarks of Miltenyi Biotec and/or its affiliates in various countries worldwide. Copyright © 2020 Miltenyi Biotec and/or its affiliates. All rights reserved.

Miltenyi Biotec



BIG ICON Lecture

Chair: Professor Christian Engwerda



Miltenyi Biotec

From Malaria Vaccines to Precision Immunology: Reflections on a Scientific Journey

Denise Doolan

Institute of Molecular Biosciences. University of Queensland, Brisbane (QLD) Australia

Over the past three decades, advances in genomics, computational biology and high-throughput technologies have transformed how human immunity can be studied. My research has evolved alongside these developments, beginning with malaria immunity and vaccine development and expanding into genome-scale antigen discovery, systems immunology and biomarker research. A central focus throughout has been understanding how human immune responses can be measured and interpreted to identify targets for intervention and signatures associated with protection, disease susceptibility and clinical outcome. This work has progressed from the study of individual malaria antigens and immune mechanisms to proteome-wide approaches capable of interrogating responses across entire pathogen proteomes. In this BIG ICON lecture, I will reflect on the scientific questions, technologies and collaborations that have shaped this progression. Drawing on examples from malaria vaccinology and more recent studies of Epstein–Barr virus-associated cancers and autoimmune diseases, including multiple sclerosis, I will discuss how emerging technologies have changed both the questions we ask and the scale at which they can be addressed. The lecture will provide a perspective on the evolution of modern immunology and on building a research program spanning discovery, translation and global health.



BIG 2026 POSTERS

POSTER 1

Discovery of Marine-Derived Saponins as Sustainable Vaccine Adjuvant Candidates

Yezhou Yu[1,2], Krupa Shah[1,2], Mariusz Skwarczynski[2,3], Jack Ryan[2,4], Craig Morton[2,4], Rohan Davis[1,2,5], Danielle Stanisic[1,2*], Katherine Andrews[1,2*]

[1]Institute for Biomedicine and Glycomics, Griffith University [2]NHMRC Centre for Research Excellent in Vaccine Adjuvant Discovery and Development (Vaccine-ADD; *co-Directors) [3]School of Chemistry and Molecular Biosciences, University of Queensland [4]Commonwealth Scientific and Industrial Research Organisation (CSIRO) [5]School of Environment and Sciences, Griffith University

Saponin-based adjuvants, such as QS-21, play an important role in improving immune responses in modern vaccines. However, a challenge is that saponins are derived from the bark of the slow growing South American tree, *Quillaja saponaria*, and commercial sources are limited, with competition also arising from use by food, agricultural and other industries. As the demand for vaccine adjuvants increases, reliable sources of saponins are critical, especially given potential risks to saponin harvest by environmental change, natural disasters, or geopolitical policies. Several approaches have been explored to address this issue, such as the use of tree-lets or yeast-based production of QS-21, however to date none have proven commercially viable in terms of yield and cost. To address this, we are exploring a strategy to discover new saponins or alternative sources from natural products. This project aims to identify immunostimulatory saponins from sea cucumbers which are more widely distributed and can be easier to farm or harvest than trees. The sea cucumber library was derived from Griffith University's Nature Bank, Australia's largest natural product library for therapeutic screening. Candidate samples were selected by literature searching and database indexing, with a focus on organism type, reported saponin content and sample availability. A set of 27 Holothuroidea class samples from several genera have been selected. Extracts will be prepared and separated by HPLC to obtain saponin-enriched fractions. These fractions will be tested for immune marker stimulation using primary human cells (Peripheral Blood Mononuclear Cells, PBMCs) via flow cytometry. Hit samples will be further purified, retested and activity validated before structure elucidation and additional studies to determine in vivo immunogenicity in vaccine adjuvant formulations in mice. The goal of this work is to discover an alternative source of QS21 and/or novel saponin-based vaccine adjuvant candidates from a commercially sustainable source.

POSTER 2

Homologous Hook-related proteins exhibit coordinated spatial organisation and specialised trafficking functions

Kehuan Wang[1], Blerida Banushi[2], Liwang Zhang[1], Shannon R. Joseph[1] and Fiona Simpson[1]

[1]Frazer Institute, The University of Queensland, Brisbane, QLD, Australia, [2]School of Indigenous Studies, The University of Western Australia, Perth, WA, Australia

Polarised membrane trafficking underpins essential immune processes, including immune synapse assembly, receptor recycling and directed secretion. The Hook-related protein (HkRP)family comprises three highly homologous proteins: Girdin (HkRP1), Daple (HkRP2) and Gipie(HkRP3). Individual HkRP family members have been implicated in receptor trafficking, regulated secretion, immune synapse formation and cancer progression, yet despite their close structural similarity they have almost exclusively been studied independently. Previous work from our laboratory demonstrated that HkRP proteins influence one another through changes in expression and localisation, suggesting that HkRP biology should be interpretedin the context of the entire family. We therefore performed a comparative analysis across cancer and immune cell models. Endogenous HkRP family members occupied distinct but spatially related trafficking domains. Girdin was associated with recycling endosomal and peripheral membrane compartments, Daple was enriched within nuclear and perinuclear regions, whereas Gipie predominantly localised to the microtubule organising centre (MTOC). Co-expression of the Girdin C-terminal region redistributed the Daple C-terminal region from the nucleus to the cytoplasm, supporting functional interdependence within the HkRP family. Further analyses revealed that GFP-Gipie partially colocalised with endogenous Girdin at peripheral membrane protrusions and exhibited a ring-like distribution surrounding MHC-I-positive puncta, while mCherry-Daple was positioned adjacent to Rab8-, CASC4- and pericentrin-associated structures. Preliminary observations suggest altered Rab8 and CASC4 organisation following GFP-Gipie expression. HkRP family members also displayed distinct signalling outputs following EGF stimulation. Expression of the Girdin C-terminal region preferentially enhanced AKT phosphorylation, whereas the Daple and Gipie C-terminal regions prolonged EGFR phosphorylation, suggesting functional specialisation in receptor trafficking-associated signalling. Together, these findings support a family-level model in which HkRP proteins exhibit coordinated spatial organisation and functional specialisation, providing a framework for understanding how HkRP family members collectively regulate membrane trafficking in immune and cancer cells.

POSTER 3

Co-culture platform of patient derived organoids and autologous T cells to discover novel immunotherapeutic targets

Simon Manuel Tria[1, 2], Gregory Kelly[1, 2], Catherine Bond[1, 2], Kelly Olsen[1], Prerna Tarun Gupta[1], Terufumi Yoshida[1, 3], David Cavalucci[1, 4], Matthew Burge[1, 5], David Clark[1, 4, 6], Di Yu [1, 7], Vicki Whitehall[1, 2, 8]

[1]University of Queensland, Faculty of Health, Medicine and Behavioural Science, [2]QIMR Berghofer, Conjoint Gastroenterology Laboratory, [3]Kansai Medical University, Colorectal Surgery, Osaka, Japan, [4]Royal Brisbane and Women's Hospital, Department of Surgery [5]Royal Brisbane and Women's Hospital, Department of Medical Oncology [6]St Vincent's Private Hospital, Department of Surgery [7]University of Queensland, Frazer Institute [8]Queensland Health, Pathology Queensland

Immune checkpoint inhibition has shown limited efficacy for colorectal cancer. The liver is the primary site of colorectal cancer metastasis, where T cell function is suppressed that further exacerbates immunotherapy resistance. Historically, immunotherapy studies have required immunocompetent animal models, usually with a limited number of murine cancer cell lines. To study patient-specific tumour-immune interactions, we are using patient-derived tumour organoids co-cultured with autologous immune cells. These organoids recapitulate the originating tissue biology in both morphology and molecular heterogeneity. Systemic blood from these patients provides a pool of autologous immune cells. To understand how tumours can resist T cell mediated killing, we have optimized a protocol to generate tumour reactive T cells. This protocol generates antigen presenting cells (APCs) - namely macrophages and dendritic cells - from the patient's own PBMCs. We found the addition of these APCs significantly enhances the quality and quantity of the T cells. These T cells not only have 10-fold higher proliferative activity, but also more strongly upregulate key activation markers such as CD69, HLA-DR and CD45RO. Upon re-stimulation with organoids, the T cells readily produce functional markers CD107a, IFNɣ, GZMB, TNFα, CD69 and CD137, confirming successful reactivity. Interactions between the trained T cells and patient-matched organoids, with or without the presence of anti- PD1 immunotherapy, were studied by live cell immunofluorescence microscopy. This provides us real-time evaluation of patient specific responses to immunotherapy. To identify molecular drivers of resistance to T cell mediated killing, we will conduct a genome-wide CRISPR screen using this co-culture platform. We hypothesize that the screen will identify resistance pathways and novel immunotherapeutic targets that can be developed to enhance immunotherapy sensitivity. The proposed study has the addresses the dismal prognosis of patients with metastatic colorectal cancer, reducing the overall mortality rate.

POSTER 4

An enhanced platform for multiepitope mRNA cancer vaccines

Hannah Tompkins[1], Liam O'Brien[1], Seth Cheetham[1,2]

[1]Australian Institute for Bioengineering and Nanotechnology, University of Queensland, Brisbane, Australia
[2]National Biologics Facility, University of Queensland, Brisbane, Australia

Despite decades of extensive research effort, previous therapeutic cancer vaccines have shown modest clinical benefit to patients. Recent advances in immunotherapy, next generation sequencing, and mRNA technology have enabled the development of several personalised neoantigen mRNA vaccines which have shown early clinical efficacy in many cancers including melanoma, pancreatic cancer, and triple negative breast cancer. However, to maximise breadth and magnitude of induced T cell responses, multiepitope vaccines must be optimally designed to enhance MHC presentation and prevent immunodominance between epitopes. We therefore compared various components of vaccine design to define an optimal mRNA cancer vaccine platform. To optimise MHC processing and presentation of mRNA vaccines, we examined the impact of linker sequence *in vitro* using TCR-transgenic Jurkat cells specific for encoded vaccine epitopes. We showed that using a linker sequence optimised for immunoproteasomal cleavage (AAY) resulted in significantly enhanced T cell responses to each epitope compared to a GS-based linker. Surprisingly, we found that including signals for secretion and MHC trafficking of antigens reduced the effector T cell response. We designed three different vaccines encoding five known epitopes from B16-OVA to compare immunogenicity *in vivo*. We found that encoding epitopes on separate mRNA strands and combining before encapsulation results in significantly improved epitope-specific CD8 T cell expansion and CD4 T cell proliferation compared to a polyepitope vaccine design. A vaccine encoding long epitopes (18- or 19-mer) resulted in substantially fewer circulating epitope-specific CD8 T cells compared to minimal epitope vaccines (8- or 9-mer). Overall, encoding minimal epitopes and encapsulating them using a "combined monotopes" strategy can reduce immunodominance of epitopes and improve breadth and intensity of CD8 T cell activation in response to an mRNA cancer vaccine. This research offers an optimised engineering approach for mRNA cancer vaccines to overcome therapeutic vaccine resistance and improve patient outcomes.

POSTER 5

Adoptive transfer of wild-type bone marrow cells mitigates age-dependent neuropathology in microglia-deficient mice

Isis Taylor[1], Omkar L. Patkar[1], Yajun Liu1, Sebastien Jacquelin[1], Ginell Ranpura[1], Dylan Carter-Cusack[1], Nikita Rosendahl[1], Adam Ewing[1], Nyoman D. Kurniawan[2], Mosi Li[3,4], Deepali Vasoya[3,4], Xin He[3,4,5], Owen R. Dando[3,4,5], Peter Kind[4,5], Giles E. Hardingham[3,4,5], Barry M. Bradford[6], Neil A. Mabbott[6], Lucas LeFevre[7], Clare Pridans[5,7], Kim M. Summers[1], Katharine M. Irvine[1] and David A. Hume[1]

[1]Mater Research Institute-University of Queensland, Translational Research Institute, [2]Australian Institute for Bioengineering and Nanotechnology, Centre for Advanced Imaging, The University of Queensland [3]UK Dementia Research Institute at the University of Edinburgh, Chancellor's Building, Edinburgh Medical School [4]Centre for Discovery Brain Sciences, University of Edinburgh, Hugh Robson Building, George Square [5]Simons Initiative for the Developing Brain, University of Edinburgh, Hugh Robson Building, George Square [6]The Roslin Institute, University of Edinburgh, [7]Centre for Inflammation Research, Institute for Regeneration and Repair. University of Edinburgh

Germ-line deletion of a conserved enhancer (FIRE) in the mouse *Csf1r* locus causes congenital absence of microglia. In total RNA-seq data from multiple brain regions of juvenile wild-type and *Fireko* mice, the loss of microglia-specific transcripts and increased expression of astrocyte activation markers were the only significant changes. Transcripts strongly enriched in isolated microglia (e.g. *Tspo, Nrros, Igf1*) were not affected indicating compensatory expression by other cell types. The *Fireko* mutation on a C57BL/6J background led to perinatal lethality and hydrocephalus in surviving pups that was overcome in F2 intercross with CBA or BALB/c. Microglia deficiency in this cohort had no effect on the abundance of perineuronal nets or parvalbumin neurons, previously inferred to be regulated by microglia. Microglial deficiency also did not affect motor activity, behaviour, or myelination but led to age-dependent astrocytosis and severe calcification in the thalamus. Intraperitoneal transfer of WT bone marrow cells (BMT) in a viable congenic C57BL/6J line fully repopulated the brain with microglia-like cells that expressed damage-associated microglia transcripts and proteins (e.g. *Adgre1/*F4/80; *Lgals3/*Mac2). BMT prevented thalamic calcification, but neither increased nor decreased markers of astrocyte or oligodendrocyte activation including *C4b*, *Serpina3n* and *Gfap.* We suggest that the damage-associated microglial phenotype is a protective response to injury rather than a cause of neuropathology, and that recruited cells are as effective as transplanted microglia in mitigating the effects of microglial deficiency.

POSTER 6

Vaccine-ADD - An NHMRC Centre for Research Excellence in Vaccine Adjuvant Discovery and Development.

Krupa Shah[1,2], Yezhou Yu[1,2], Danielle Stanisic[1,2]* and Katherine Andrews[1,2]*

1. Institute for Biomedicine and Glycomics, Griffith University
2. NHMRC Centre for Research Excellence in Vaccine Adjuvant Discovery and Development (Vaccine-ADD; *co-Directors)

Vaccines are the most effective public health intervention, offering long-term protection against infectious diseases and addressing emerging global health challenges. Adjuvants are key components of some vaccines that enhance vaccine-induced immune responses and improve vaccine efficacy. Although the use of adjuvants in some licensed vaccines has significantly improved vaccine efficacy, their broader use is limited by incompletely understood mechanisms of action, restricted immunostimulatory capacity, sustainability, manufacturing complexity and stability challenges. Furthermore, new adjuvants discovery remains slow due to continued reliance on empirical, trial-and-error screening approaches, which are labour- intensive, time-consuming, and often inadequate for discovering adjuvant candidates suitable for next-generation vaccines. These limitation underscores the urgent need for safer, more effective, and mechanistically defined adjuvants, supported by integrated discovery pipelines for rapid identification and validation. To address these challenges, the NHMRC Centre for Research Excellence in Vaccine Adjuvant Discovery and Development (Vaccine-ADD) is utilising a pipeline integrating in vitro and in vivo immune profiling, in silico modelling and multi-omics analyses to accelerate the adjuvant discovery and development. Chemically diverse libraries comprising synthetic and natural product compounds derived from the Griffith University NatureBank will be screened using in vitro assays with peripheral blood mononuclear cells (PBMCs) from diverse donors to assess cytotoxicity, cellular activation and cytokine production as measured by multiplex immunoassays and flow cytometry. Prioritised leads will be further characterised by CytoF, RNA sequencing, integrated system biology analysis and reporter cell line studies to define immune activation signatures, signalling pathways, gene expression profiles and mechanisms of actions. Hits will be subjected to medicinal chemistry optimisation and iterative biological evaluation before assessment in pre-clinical vaccine models to determine their immunogenicity and protective efficacy. By integrating this pipeline, we enable mechanism- guided prioritization of adjuvant candidates, accelerating the development of safer, more effective, and precisely targeted next-generation vaccines to address future global health challenges.

POSTER 7

IFN-γ is dispensable for epithelial MHC-II induction but shapes the immune landscape of HPV16 E7-driven hyperplasia

Lakshita Patil[1], Joshua Kim[1], Kalani Hettiarachchi[1], Debottam Sinha[1], and Janin Chandra[1,2]

[1]Frazer Institute, Faculty of Health, Medicine and Behavioural Science, The University of Queensland, Woolloongabba, QLD, Australia. [2]Dermatology Research Centre, The University of Queensland, Brisbane, QLD, Australia.

Background: Oncogenic HPV-associated intraepithelial lesions are characterised by altered epithelial differentiation, immune dysregulation, and increased expression of antigen presentation markers, including MHC-II. Although IFN-γ is a well-established inducer of MHC-II expression, the mechanisms underlying epithelial MHC-II upregulation during HPV-driven epithelial transformation is unknown. **Aim:** Our study aimed to determine the role of IFN-γ in regulating epithelial MHC-II expression and shaping the immune landscape during HPV16 E7-driven epithelial hyperplasia using K14E7 transgenic mice deficient for IFN-γ. **Methods:** 13-colour flow cytometry was used to quantify MHC-II within non-immune populations isolated from murine skin epidermis. 29-colour flow cytometry combined with unsupervised t-SNE analysis was performed to characterise immune cell composition and identify IFN-γ-dependent changes within the cutaneous immune microenvironment. **Results:** Genetic deletion of IFN-γ did not alter MHC-II expression within total non-immune populations or within individual epithelial subsets, including $EpCAM^+$ keratinocytes, $EpCAM^+CD90^+$ basal epithelial cells, and $EpCAM^-$ stromal cells. This demonstrates that HPV16 E7-driven epithelial MHC-II induction occurs independently of IFN-γ. In contrast, loss of IFN-γ significantly altered the immune composition of hyperplastic skin epidermis. HPV16 E7-driven intraepithelial hyperplasia was associated with increased immune infiltration, which was significantly reduced following IFN-γ deletion, indicating that IFN-γ contributes to immune recruitment and maintenance within the hyperplastic microenvironment. Immune profiling further revealed cell-type-specific alterations following IFN-γ deficiency, including γδ T-cell depletion, reduced infiltration of Tregs, and extensive remodelling of Langerhans cell-like and monocyte-like populations. **Conclusion:** These findings identify distinct roles for IFN-γ in HPV16 E7-driven epithelial hyperplasia. While epithelial MHC-II is induced independently of IFN-γ, it is still a critical regulator of immune cell recruitment and composition within the hyperplastic skin microenvironment. This demonstrates that epithelial antigen presentation and immune landscape remodelling during HPV-associated epithelial disease are regulated through partially independent inflammatory pathways.

POSTER 8

Targeting the Tumour Microenvironment Through Eosinophil Enhancement in Humanised Adenoid Cystic Carcinoma

Lixue Li[1], Marion F. Barros[1], Debottam Sinha[1], Benedict Lum[2], Lilia Merida de Long[1], Liwang Zhang[1], Jason J. Lee[1], Kehuan Wang[1], Shannon R. Joseph[1,] and Fiona Simpson[1].

[1]Frazer Institute, The University of Queensland, Brisbane, QLD, Australia, [2]Australian Institute for Bioengineering and Nanotechnology, The University of Queensland, Brisbane, QLD, Australia

Adenoid cystic carcinoma (ACC) is a rare malignancy distinguished by an immunologically cold tumour microenvironment and restricted responsiveness to existing immunotherapies, underlining the need for novel therapeutic strategies. As eosinophils have been implicated in promoting antitumour immunity in several solid tumours, we investigated whether increasing intratumoural eosinophil accumulation could improve immune responses in ACC. Human peripheral blood mononuclear cells (PBMCs) and purified human eosinophils were engrafted into NSG-MHC I/II double-knockout (DKO) mice with ACCx11 tumours to establish a humanised ACC patient-derived xenograft (PDX) model. IL-5 and Eotaxin were administered to stimulate eosinophil accumulation. Peripheral blood, spleen, and tumour tissues were analysed using a newly developed multi-fluorescent flow cytometry panel capable of concurrently recognising human and mouse immune cells. Human PBMC engraftment was successfully achieved in the majority of mice. While adoptively transferred human eosinophils were not detected at the study endpoint, IL-5- and Eotaxin-treated mice demonstrated increased murine eosinophil accumulation, accompanied by early tumour-growth suppression and exploratory immune changes, including increased tumour NK-cell frequencies and tumour-cell PD-L1 expression. These findings establish a humanised ACC-PDX platform for investigating eosinophil-based immunotherapy and provide preliminary evidence that IL-5 and Eotaxin can enhance eosinophil responses within the ACC tumour microenvironment. Ongoing research focuses on optimising eosinophil persistence and testing combination immunotherapy methods.

POSTER 9

A dual-targeting CAR T-cell strategy for breast cancer incorporating viral and tumour antigens

Xiang Li[1, 2], Paulo Martins[1], Kum Kum Khanna[2, 3], Rajiv Khanna[1, 2]

[1]Tumour Immunology Laboratory, QIMR Berghofer Medical Research Institute, QLD Australia [2]The University of Queensland, QLD Australia, [3]Tumour Biology and Therapeutics Research, Mater Research Institute, QLD Australia

Breast cancer remains one of the most prevalent malignancies affecting women worldwide and continues to pose significant clinical challenges due to high rates of recurrence and limited therapeutic options for advanced disease, particularly in triple-negative breast cancer (TNBC). Emerging evidence suggests that infectious agents may contribute to breast cancer pathogenesis, with human cytomegalovirus (CMV), a ubiquitous double-stranded DNA virus infecting more than 90% of the global population, being increasingly implicated. CMV-positive cells have been detected in breast tumours and are frequently associated with malignant epithelial cells, where they are thought to modulate the tumour microenvironment by promoting immune evasion, angiogenesis, and metastatic progression. Consistent with these observations, immunohistochemical analysis of a 100-core breast cancer tissue microarray revealed the presence of CMV-positive cells across multiple tumour samples, supporting a potential role for CMV in breast cancer biology. Chimeric antigen receptor (CAR) T-cell therapy is a rapidly evolving form of personalized immunotherapy that involves the isolation of patient-derived T cells, their genetic modification to express tumour-targeting receptors, and subsequent reinfusion to mediate antigen-specific cytotoxicity. While CAR T-cell therapies have demonstrated remarkable success in haematological malignancies, their application in solid tumours such as breast cancer remains limited by tumour heterogeneity and an immunosuppressive microenvironment. My PhD project is aiming to develop and optimize CAR T-cell therapies targeting CMV-derived antigens, exploiting viral antigen expression within tumours, alongside established breast cancer-associated targets such as TROP2. By integrating viral and tumour-specific targeting strategies, this work seeks to establish a robust, multifaceted CAR T-cell immunotherapy platform with the potential to improve therapeutic outcomes for patients with breast cancer.

POSTER 10

Combinatorial IFNβ and CD40 Stimulation Enhances Human cDC1 Activation and Metabolic Reprogramming to enhance CD8+ T Cell Priming

Liam O'Brien[1], **Ingrid Leal Rojas[1]**, Nikita Rosendahl[1], Irina Buckle[1], Jennifer Loaiza Naranjo[1], Domenica McCarthy[1], Josephine Forbes[1], Kristen Radford[1].

Mater Research, The University of Queensland, Woolloongabba, QLD, Australia

Tumoral immune responses are primarily mediated by CD8+ cytotoxic T cells. Conventional type 1 dendritic cells (cDC1) are specialized in priming CD8+ cytotoxic T cells and are essential for cancer immunotherapy response. Optimal CD8+ T cell priming in mice is facilitated by CD4+ T cell help that is received and transmitted to CD8+ T cells via the same cDC1 in cellular triads. This help signal is primarily mediated by ligation of CD40L on the CD4+ T cell with CD40 on a mature DC1. Although innate signals including TLR ligation are known to initiate human DC1 maturation, IFN secretion and CD40 upregulation, the 'optimal' combinatorial stimulation enabling cDC1 to elicit maximal CD8+ T cell priming is unknown, along with the transcriptional and metabolic responses induced in cDC1 following exposure to varying innate signals. Here we investigated the functional and transcriptional phenotype of human cDC1 following stimulation with Type I-III IFNs and CD40 agonistic antibody. IFNβ followed by CD40 ligation induced high levels of cDC1 activation in terms of cytokine production and costimulatory molecule expression without significantly reducing survival in vitro. cDC1 activation was accompanied by upregulation of sterol and glycan biosynthetic pathways and an increase in total glycolytic and aerobic metabolism. CD40 ligation on cDC1 resulted in decreased mitochondrial efficiency, but combinatorial activation with IFNβ protected mitochondrial membrane integrity. These data highlight novel mechanisms which facilitate enhanced priming of T cells by cDC1 post-activation.

POSTER 11

Interrogating MYC-driven immune suppression in acute myeloid leukemia using an *in vitro* co-culture model.

Benjamin Hansen[1,2], Stuart Olver[1], Sophie Hamann[1], Marcela Montes de Oca[1], Steven Lane[3,4], Megan Bywater[3], Antiopi Varelias[1,2,4].

[1]Transplant Immunology Laboratory, QIMR Berghofer, Herston, QLD, 4006, Australia. [2]School of Biomedical Sciences, Queensland University of Technology, Brisbane, QLD, 4059, Australia; [3]Gilmour Leukaemia Research Laboratory, QIMR Berghofer, Herston, QLD, 4006, Australia. [4]Royal Brisbane Clinical Unit, Faculty of Health, Medicine and Behavioural Sciences, The University of Queensland, Herston, QLD, Australia.

Allogeneic stem cell transplantation (alloHSCT) is a curative treatment for high-risk acute myeloid leukaemia (AML), mediated by donor T-cell recognition of antigenic incompatibility on leukaemic cells to initiate the graft-versus-leukaemia (GVL) effect. Relapse remains a major cause of mortality post-alloHSCT and is often the result of immune evasion by leukaemic cells. Whether an AML provokes or escapes an immune response is unclear however this is dependent on the nature of the oncogenic drivers. Haematopoietic stem cells expressing mutations in the NRAS gene (Neuroblastoma RAS viral oncogene; NRAS-G12D), a common oncogenic driver in AML, display increased surface MHC and costimulatory molecule expression. MYC, a master regulatory transcription factor, has been shown to reduce AML immunogenicity by downregulating surface MHC expression and repressing interferon signalling to limit leukaemia recognition. However, MYC also drives the expression of inhibitory ligands such as PD-L1 and CD47, raising the possibility that MYC not only renders AML less visible but actively suppresses T-cell responses. To test this hypothesis, we initially developed *in vitro* co-culture assays using murine *Npm1*c/*Nras*G12D-driven (NR) AML with and without Myc overexpression as stimulators in comparison to *Npm1*c/*FLT3*ITD-driven (NF) AML. These were co-cultured with $CD3^+$ responder T-cells derived from major MHC-mismatched (BALB/c), minor antigen-mismatched (BALB.B) and syngeneic (C57BL/6) donor mice in the presence of anti-CD3/CD28 co-stimulation. Preliminary analysis revealed a reduction in $CD8^+$ T-cell proliferation and elevated expression of T-cell exhaustion markers PD-1 and TIM-3 on $CD4^+$ and $CD8^+$ T cells in the presence of Myc-overexpressing NR AML compared to empty vector (EV) control NR AML. Concomitantly, Myc-overexpressing NR AML co-cultures displayed a dysregulation in cytokine production, demonstrating a functional role for Myc-overexpressing AML in modulating T-cell responses. Ongoing work focuses on extensively characterising the T-cell phenotype to guide the development of preclinical murine transplant models to interrogate mechanisms of leukaemia control *in vivo*.

POSTER 12

Bispecific antibodies enable precision mRNA-LNP delivery

Bettina Dietmair[1,2], Liam O'Brien[1,2], James Humphries[1,3], Timothy R. Mercer[1,4], Kristofer J. Thurecht[1,3], Christopher B. Howard [1,2] and Seth W. Cheetham[1,2]

[1]Australian Institute for Bioengineering and Nanotechnology, The University of Queensland, Brisbane, QLD, Australia, [2]National Biologics Facility, The University of Queensland, Brisbane, QLD, Australia. [3]Centre for Advanced Imaging, ARC Research Hub for Advanced Manufacture of Targeted Radiopharmaceuticals, The University of Queensland, Brisbane, QLD, Australia. [4]BASE facility, The University of Queensland, Brisbane, QLD, Australia.

Messenger RNA (mRNA) therapeutics are transforming the treatment for cancers, immunological conditions, and genetic disorders. Formulated into lipid nanoparticles (LNPs), systemically administered mRNA accumulates primarily in the liver. Efficient delivery of mRNA to specific cell types remains a major challenge for clinical translation. Efforts to retarget LNPs, by altering lipid composition or adding surface ligands, have proven effective in isolated cases. However, these approaches complicate manufacturing, modify particle size and charge, and risk triggering immune recognition. We introduce a generalisable targeted delivery solution using bispecific antibodies (BsAbs) to tether unmodified mRNA-LNPs to defined cell surface markers. Each BsAb consists of an anti- PEG binding arm and an arm recognising a receptor expressed specifically on target cells. BsAbs can be combined with LNPs before injection or be administered first, bind to surface proteins on target cells, and later retain LNPs in affected tissues. Substitution of the cell-binding domain enables retargeting of various polyethylene glycol (PEG)-containing LNP formulations to different cell types. The modular BsAb platform achieves efficient, cell-specific mRNA delivery beyond the liver, targeting mRNA to a range of primary human cell targets *in vitro* and *in vivo*, and reducing off-target accumulation compared to untargeted mRNA-LNPs. BsAbs enable mRNA delivery to previously inaccessible tissues, broadening the scope of conditions addressable with mRNA therapeutics. Reduced uptake in off-target organs, including the liver and spleen, can improve the safety profile and therapeutic window of precision mRNA medicines. Non-covalent binding decouples manufacture from targeting, streamlining independent scaling of existing antibody and LNP production processes compared to functionalised mRNA-LNPs. The well-established clinical use and safety of mRNA-LNPs and BsAbs provide strong precedence for a streamlined clinical translation of this technology. Efficient and adaptable targeting of mRNA-LNPs supports the rapid development of next-generation mRNA drugs.

POSTER 13

Influence of baseline CD4$^+$ T cell structure on the anti-malaria response in naïve adults

Benjamin Crawford[1,2], Jessica Engel[2], Fabian De Labastida Rivera[2], Christian Engwerda[2]

[1]Centre for Clinical Research, The University of Queensland. [2]Immunology and Infection Lab, QIMR Berghofer Medical Research Institute.

Malaria remains a global public health challenge. It is a vector-borne disease caused by *Plasmodium* parasites that are transmitted to human via infected female *Anopheles* mosquitoes. In 2024, the World Health Organization reported an estimate of 282 million malaria cases and 610,000 deaths globally, with children under the age of five residing in Africa accounting for the majority of deaths. Effective parasite clearance and the development of protective immunity depend on a careful balance between the inflammatory and regulatory activities of CD4$^+$ T cells. There is a growing appreciation for how an individual's immune status prior to infection and vaccination (i.e., baseline) could influence the heterogeneity of responses that we typically observe. However, the impact of baseline immunity on malaria disease and vaccination outcomes remains poorly understood. Given the importance of CD4$^+$ T cells to anti-parasitic response to malaria, I hypothesize that the baseline composition of CD4$^+$ T cells may also contribute. For my PhD project, I will be utilizing large proteomic and single cell transcriptomic datasets, generated from samples collected as part of a controlled human malaria infection trial, to investigate the relationship between baseline CD4$^+$ T cell composition and the responses to *P. falciparum* infection in malaria naïve adults. This project may unveil new predictors of malaria disease outcome and identify potential immunomodulatory targets to enhance malaria treatment and vaccination.

POSTER 14

Comprehensive Phenotypic Profiling of Dendritic cells in Ovarian Cancer

Luzia Bukali[1], Ingrid Leal Rojas[1], Nikita Rosendahl[1], Kristen Radford[1].

Mater Research Institute, The University of Queensland, Translational Research Institute, Woolloongabba, QLD, Australia.

Mature regulatory dendritic cells (mregDCs) are a recently defined activation state arising from both cDC1 and cDC2 lineages, characterised by the co-expression of immunostimulatory and immunoregulatory molecules. While cDC1s are critical for anti-tumour immunity through efficient cross-presentation, tumour antigen uptake can drive their transition into an mregDC phenotype. Ovarian cancer remains one of the most lethal gynaecological malignancies and while mregDC signatures have been associated with improved patient outcomes, their precise functional role remains unclear. Here, we investigated whether tumour antigen-positive DCs promote immunogenic or regulatory immune responses. Using an *in vitro* differentiation system, cDC1s were generated from expanded cord blood-derived $CD34^+$ haematopoietic stem cells in serum-free conditions. These cells were subsequently co-cultured with ovarian cancer cells to model tumour antigen uptake and mregDC induction. To examine phenotypic changes in protein expression on the DCs, we utilised the BioLegend's LegendScreen ™, a flow cytometry-based kit that enables the screening of over 300 different surface proteins. This approach revealed phenotypic differences in tumour antigen-positive cDC1s compared to their antigen-negative counterparts, including differential expression of immune checkpoint ligands, co-stimulatory molecules and antigen presentation markers. Importantly, this broad screening strategy identified multiple previously unreported surface proteins associated with tumour-exposed cDC1s and highly activated cDC1s. These findings provide new insight into the complexity of tumour-induced DC phenotypes and establish a resource for identifying candidate regulators of anti-tumour immunity.

POSTER 15

Changing drug response in Adenoid Cystic Carcinoma through eosinophil-mediated immune recruitment

Marion Barros[1], Lixue Li[1], Debottam Sinha[1], Lilia Merida de Long[1], Shannon R. Joseph[1], Gabrielle Belz[1], Fiona Simpson[1]

Frazer Institute, The University of Queensland.

Adenoid Cystic Carcinoma (ACC) is a rare, sporadic cancer of the salivary glands with an incidence of 3 – 4.5 per million. Despite an initially indolent progression, late-stage tumours demonstrate aggressive metastasis, therapy resistance, and recurrence following surgical resection. While expressing targetable oncogenic markers, clinical response remains insufficient with currently no approved therapies. Immunologically, ACC is a cold tumour. It's limited immune infiltration and high immune checkpoint expression, particularly of B7-H4 and PD-L2, correlate with worsened prognosis. Antibody drug conjugates targeting B7-H4 show the highest overall response of 55.6% in ACC clinical trials. Targeting the immune landscape is therefore a promising therapeutic strategy. ACC tumours are specifically depleted of eosinophils, which initiate tissue inflammation and are necessary biomarkers of cancer immunotherapy response. As an innate immune granulocyte and microenvironment regulator, they are shown to normalise tumour vasculature and secrete chemical mediators to induce cytotoxic immune infiltration. We aim to therapeutically recruit eosinophils into ACC tumours to improve immune infiltration and drug response. We show that administration of IL-5 and Eotaxin, as major eosinophil recruiting molecules, upregulate mouse eosinophils in ACC tumour, spleen, and blood regions in vivo with low toxicity. For clinical translatability and to test human specific phenomena, we are optimising human eosinophil engraftment in mice. The functionality of eosinophils isolated from human whole blood by either negative immunomagnetic selection or density gradient centrifugation will be compared in vivo. We will use these novel humanised models to test the IL-5 and Eotaxin eosinophil induction strategy with ACC targeted drugs to enhance their efficacy. This research aims to uncover a new layer of granulocyte biology, specifically in the context of cancer and clinical translation. Furthermore, it aims to improve humanisation models by incorporating granulocyte populations, which are currently lacking. This enhances the clinical translatability of immunological research models.

POSTER 16

Tumour-Specific Gipie Reprogrammes Intracellular Trafficking to Promote Immune Evasion

Liwang Zhang[1], Alexandre Libert[2], Samantha Stehbens[2], Shannon R. Joseph[1], Fiona Simpson[1]

[1]Frazer Institute, Faculty of Medicine, The University of Queensland, Brisbane, Australia. [2]Institute for Molecular Bioscience, The University of Queensland, Brisbane, Australia

Antibody-dependent cellular cytotoxicity (ADCC) is a major mechanism underlying the efficacy of therapeutic antibodies, yet many tumours evade immune destruction through poorly understood intracellular mechanisms. Gipie (CCDC88B) is an immune cell trafficking protein required for immune function, but its role when ectopically expressed in cancer cells and its contribution to tumour immune evasion are unknown. Complementary cell biology, proteomic and functional immunology approaches were used to investigate the role of Gipie in immune synapse organisation, intracellular trafficking and tumour susceptibility to antibody-mediated immune killing. Gipie, a microtubule-to-vesicle linker complex protein, dynamically redistributed during immune synapse formation and accumulated at the tumour–immune interface following therapeutic antibody-mediated immune activation. Ectopic Gipie expression significantly reduced antibody-dependent tumour killing, demonstrating a functional role in tumour immune evasion. Proteomic analyses identified a distinct tumour-associated Gipie interaction network enriched for trafficking, immune regulatory and signalling proteins, supporting the hypothesis that Gipie reprogrammes intracellular trafficking rather than regulating a single downstream target. Our findings identify Gipie as a previously unrecognised regulator of tumour immune evasion. We propose that tumour cells hijack this immune cell trafficking protein to establish a tumour-specific intracellular trafficking programme that suppresses anti-tumour immunity. These findings provide a new mechanistic framework for understanding resistance to antibody-based immunotherapy and identify Gipie-associated trafficking pathways as promising therapeutic targets.

BIG-2026 Delegate List

Name	Company Name
Danielle Stanisic	IBG, Griffith University
Giovanni Pietrogrande	AIBN, The University of Queensland
Harindra Sathkumara	AITHM, James Cook University
Roland Ruscher	James Cook University
Kathryn Friend	Chameleon Science
Ken Beagley	Queensland University of Technology (QUT)
Kate Seib	IBG, Griffith University
Sumaira Hasnain	Mater Research
Rajiv Khanna	QIMR Berghofer
Vijayendra Dasari	QIMR Berghofer
Paulo Martins	QIMR Berghofer
Pauline Crooks	QIMR Berghofer
Archana Panikkar	QIMR Berghofer
Michelle Neller	QIMR Berghofer
Hilary Reddiex	QIMR Berghofer
Sweera Rehan	QIMR Berghofer
Shachi Rana	QIMR Berghofer
Leone Beagley	QIMR Berghofer
Xiang Li	QIMR Berghofer
Thuy Le	QIMR Berghofer
Shannon Flynn	QIMR Berghofer
Jyothy Raju	QIMR Berghofer
Matthew Solomon	QIMR Berghofer
Ben Leung	QIMR Berghofer
Sabine Smythe	QIMR Berghofer
Kaylah Perske	QIMR Berghofer
Christian Engwarda	QIMR Berghofer
Jessica Engel	QIMR Berghofer
Fabian De Labastida Rivera	QIMR Berghofer
Benjamin Crawford	QIMR Berghofer
James Hudson	QIMR Berghofer
Christopher Hunter	QIMR Berghofer
Mika Casey	Stemcell
Laurence Macia	Sanofi
Kimberley Nelson	Sanofi
Sharni Hardcastle	Miltenyi Biotec
TBC	Australian Bioresearch
Corey Smith	QIMR Berghofer

Oyime Poise Aula	QIMR Berghofer
Panteha Khaledi	QIMR Berghofer
Ennae Gollasch-Miller	The University of Queensland
Ismail Sebina	The University of Queensland
Denise Doolan	The University of Queensland
Gabrielle Belz	The University of Queensland
Chen Wang	The University of Queensland
Simon Phipps	QIMR Berghofer
Zeeshan Chaudhry	The University of Queensland
Hock Tay	QIMR Berghofer
Yezhou Yu	IBG, Griffith University
Katharine Irvine	Mater Research - UQ
Amanda Oliver	QIMR Berghofer
Krupa Shah	IBG, Griffith University
Shoaib Anwaar	IBG, Griffith University
Karthik Pullela	Millenium Bioscience
Patricia Namubiru	QIMR Berghofer
Kate Gartlan	QIMR Berghofer
Mason Lacy	Queensland University of Technology (QUT)
Michelle Wykes	QIMR Berghofer
Vrusali Maste	The University of Queensland
Adrianne Jenner	Queensland University of Technology (QUT)
Thishan Illankoon	The University of Queensland
Simon Tria	QIMR Berghofer
Luzia Bukali	Mater Research - UQ
Benjamin Hansen	QIMR Berghofer
Sohel Rana	QIMR Berghofer
Nazifa Tabassum	QIMR Berghofer
Carla Proietti	The University of Queensland
Di Yu	The University of Queensland
Lixue Li	Translational Research Institute
Laura Grice	Mater Research
Capucine Bianciotto	Mater Research
Liwang Zhang	Frazer Institute, the University of Queensland
Debbie Watson	University Of Wollongong
Bettina Dietmair	AIBN, The University of Queensland
Marion Barros	Frazer Institute, the University of Queensland
Ingrid Marcela Leal Rojas	Mater Research UQ / TRI
Lakshita Patil	Frazer Institute, the University of Queensland
Kehuan Wang	Frazer Institute, the University of Queensland

Trent Woodruff	The University of Queensland
Hannah Tompkins	Australian Institute for Bioengineering and Nanotechnology
Isis Taylor	Translational Research Institute
Tara Minehane	The University of Queensland
Tyler Wilkins	QIMR Berghofer
Carina Walpole	TRI/UQBR
Kelly-Anne Masterman	Translational Research Institute
Sabrina Burgener	The University of Queensland
Rajneesh Rajneesh	QIMR Berghofer
Hiu On Man	Frazer Institute, the University of Queensland
Adriana Pliego	SCMB, The University of Queensland
Kate Stacey	SCMB, The University of Queensland
Janin Chandra	The University of Queensland
Bianca Nowlan	QIMR Berghofer
Sharon Lewin	University of Melbourne
Shalin Naik	Walter and Eliza Hall Institute of Medical Research
Julia Ellyard	Australian National University
Michael Gantier	Hudson Institute of Medical Research