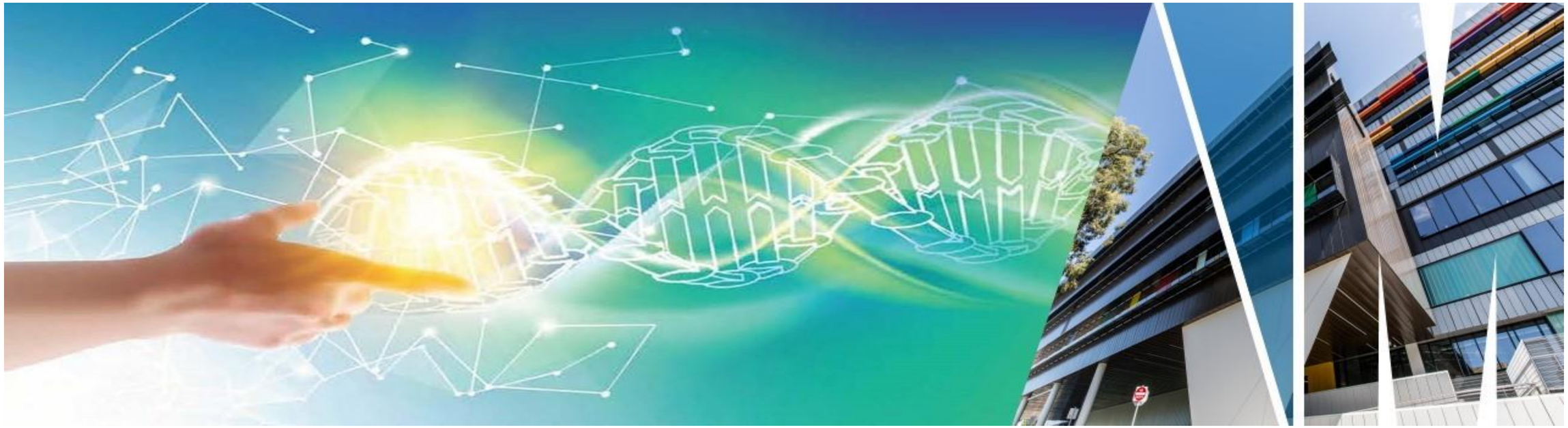
MONASH University

MONASH
CLINICAL AND
MOLECULAR MEDICINE



SF-CaMM Graduate Research Showcase Symposium 2026

10:00 a.m. to 2.30 p.m.

Program

Time	Presenter Name	Title of Presentation	Department / Institute
10:00 am Formal Opening by Head of Graduate Research, Prof. Patrick Western			
10.15 - 10.30 am	Jenna Douglas	Altered basolateral secretions of eutopic endometrial epithelial organoids reflect a dysregulated hormone response in defined clinical stages of endometriosis	Obstetrics & Gynaecology
10.30 - 10.45 am	Elly Green	'Anakinra Pilot - Safety, Feasibility, and Pharmacokinetics of Anakinra in Preterm Infants'.	Paediatrics
10.45 - 11.00 am	Andre Espiritu	Neutrophil Extracellular Trap Inhibition as a Safer Therapeutic Strategy for ANCA-Associated Vasculitis	Medicine
11.00 - 11.01 am Remembrance Day, a one-minute silence			
11.01 - 11.16am	Sheran Vasanthakumar	"Coronary artery disease in women and novel imaging risk markers"	Victorian Heart Institute
11.16 - 11.31 pm	Ruo Wang	Understanding the changes in intestinal stem cell function after enteropathogenic *Escherichia coli* infection	Hudson / Department of Molecular and Translational Sciences
11.31 - 11.46 pm	Ricki Marzan	Human Cerebral Organoids Reveal Timing-Dependent Inflammatory and Reparative Mechanisms of UCB Therapy in Perinatal Neuroinflammation.	Obstetrics & Gynaecology
11.46 – 12.01 pm	Sasan Amanat	Diet and post-stroke cognition	Nutrition Science and Dietetics
30 mins Lunch Break with Light Entertainment			
12.30 - 12.45 pm	Chathuri Abeyakoon	Improving Outcomes in Peripheral T-cell Lymphoma: From real world evidence to molecular studies and clinical trials	Medicine
12.45 - 1.00 pm	Nigus Yimer	Advancing Precision Medicine in Women's Health Through Personalised Risk Prediction and Causal Inference	Monash Centre for Health Research and Implementation (MCHRI)
1.00 - 1.15 pm	Yaoxi Wu	Targeting HSP90 suppresses bladder cancer cells and reduces tumour burden in an orthotopic NMIBC model	Surgery
1.15 - 1. 30 pm	Barnaby Kelly	Custom CRISPR screening and multi-omic data identify targetable drivers of cisplatin resistance in paediatric osteosarcoma.	Hudson / Department of Molecular and Translational Sciences
1.30 - 1.45 pm	Linda Hayes	Designing a model of care in cervical cancer prevention with and for women with Severe Mental Illness (SMI)	Psychiatry
2.00 - 2.30, Formal Close by CaMMunity and Prof. John Carroll, Interim Executive Dean, photo opportunity for the Comms teams and Winners Announcement!			

MONASH University

MONASH
CLINICAL AND
MOLECULAR MEDICINE



First Place - $500



Second Place - $300



People’s Choice - $100

Jenna Douglas, Department of Obstetrics & Gynaecology

Altered basolateral secretions of eutopic endometrial epithelial organoids reflect a dysregulated hormone response in defined clinical stages of endometriosis



Jenna Douglas is a second-year PhD student under the supervision of Dr Harriet Fitzgerald, Professor Beverley Vollenhoven and Professor Caroline Gargett in The Ritchie Centre, Hudson Institute of Medical Research and Department of Obstetrics and Gynaecology at Monash University, as well as the newly formed Ainsworth Endometriosis Research Institute (AERI). Jenna's PhD focuses on understanding endometrial dysfunction and infertility in endometriosis using patient-derived human endometrial epithelial organoids (EEO), proteomics and developing a 3D co-culture model of the endometrium to examine cell-cell interactions and the impact of endometriosis on this. She is passionate about improving reproductive and gynaecological health outcomes for all people with endometriosis. Jenna is also the co-president of the Hudson Institute Student Society (HISS), a student-led community aimed at elevating social wellbeing and career development for graduate research students.

Abstract

Background

The inner uterine lining, the endometrium, completely remodels in response to menstrual cycle hormones. Endometriosis is a chronic, debilitating gynaecological disorder that affects more than 10% of women, with infertility affecting 30-50% of cases likely due to disruptions in hormone-driven processes critical for endometrial receptivity and embryo implantation. The underlying mechanisms of endometriosis-associated infertility remain largely unknown. We aimed to investigate the impact of endometriosis on the hormonal regulation of endometrial epithelia and their secreted products using endometrial epithelial organoids (EEO).

Methods

Eutopic EEO from patients diagnosed with minimal-mild (stages I-II, n=7), moderate-severe (stages III-IV, n=5) and without endometriosis (control, n=7) were treated with either vehicle control, 17β-estradiol (E2) (proliferative phase mimic), or E2 and medroxyprogesterone acetate (MPA) (secretory phase mimic). Hormone receptors PGR, ESR1 and ESR2 in EEO were examined by RT-qPCR and in full-thickness hysterectomy tissue by immunofluorescence. The basolateral secreted proteome of hormone-treated EEO was analysed by mass spectrometry.

Results

PGR expression in EEO resembled in vivo cycle phase-dependent patterns from controls ($p<0.05$) and stage I-II endometriosis ($p<0.01$), but not stage III-IV endometriosis. Glandular PGR and ESR1 localisation were sustained in secretory phase endometrium from endometriosis patients but not control patients. 167 significantly ($P<0.05$, Fold Change >2.0, <-2.0) differentially abundant secreted proteins were identified overall across hormone treatments and groups of endometriosis severity, including RARRES2 (FC=2.64, $p<0.05$), ANXA1 (FC=1.7, $p<0.05$) and PAMR1 (FC=-6.78, $p<0.05$).

Conclusion

EEO from endometriosis patients demonstrate an impaired hormone response, leading to altered steroid hormone receptor expression and localisation and an aberrant secreted proteome in endometriosis patients, with stage-specific differences. These findings provide new insights into the hormonal regulation of eutopic endometrial epithelia across stages of endometriosis severity, facilitating the future development of treatments for endometriosis-associated infertility.

Dr Elly Green, Department of Paediatrics

'Anakinra Pilot - Safety, Feasibility, and Pharmacokinetics of Anakinra in Preterm Infants'



Dr Elly Green is a clinician researcher in the final year of her PhD with the Ritchie Centre. Elly obtained her fellowship with RACP as a neonatologist in 2020, completing her training in Victoria, mostly at Monash Newborn. Elly has worked as a consultant neonatologist at the Royal Children's Hospital, PIPER, and Royal Darwin Hospital. Elly also has training in sports medicine and is an officer in the Australian Army, where she is currently working. Outside of work, Elly is a passionate dog mama to Oscar and likes to spend her time at the park playing ball.

Abstract

Background

Improved survival of extremely premature infants has incurred the price of increased morbidity. Inflammation driven by the pro-inflammatory cytokine IL-1 (interleukin-1) plays a central pathophysiological role in multiple early-life diseases, including the cardiopulmonary bronchopulmonary dysplasia, intestinal necrotizing enterocolitis and cerebral white matter injury. Blockade of IL-1 with its antagonist IL-1Ra (medication name anakinra) may reduce disease burden.

Methods

Anakinra Pilot (NCT05280340) was a first-in-population, investigator-initiated clinical trial aiming to establish safety, feasibility and pharmacokinetics of anakinra therapy in preterm infants. We recruited 25 infants born between 24^{+0} (24 weeks, 0d) and 27^{+6} weeks' gestation at Monash Children's Hospital, Melbourne, Australia and Starship Hospital, Auckland, New Zealand from 2022 to 2024. Anakinra was administered intravenously over 3 min from d1 to d21 post-birth. Stage 1 dosing was determined through population-pharmacokinetic modeling; Stage 2 dosing was based on interim analyses largely confirming initial pharmacokinetic predictions. Safety was defined as adverse events/outcomes not occurring more frequently than expected in extremely premature infants at the participating centers.

Results

We did not observe immediate, acute or sub-acute adverse events following anakinra treatment. Intravenous administration was deemed feasible by medical and nursing teams. Pharmacokinetic analysis demonstrated our models were accurate, with drug exposure in the predicted range, ensuring safety. It also revealed immaturity of anakinra clearance at birth, with maturation over week one post-birth.

Conclusion

Anakinra therapy in preterm infants according to our protocol is safe and feasible, paving the way for a large trial to determine anakinra's efficacy at ameliorating early-life inflammatory diseases.

Andre Espiritu, Department of Medicine

Neutrophil Extracellular Trap Inhibition as a Safer Therapeutic Strategy for ANCA-Associated Vasculitis



Andre Espiritu is a PhD candidate at Monash University, and part of the Translational Kidney Therapies Group within the Centre for Inflammatory Diseases department. His current research investigates the translational capacity of neutrophil extracellular traps (NETs) inhibitors as a therapeutic strategy for ANCA-associated vasculitis. Prior to his PhD, Andre completed a Science with Honours degree, during which he explored the relationship between annexin A2 and antiviral immune responses involving the pattern recognition receptor melanoma differentiation-associated protein 5. Andre's research interests span virology and immunology in autoimmune and inflammatory diseases, with a particular focus on understanding the mechanisms behind NET-mediated pathology.

Abstract

Aim: To assess the therapeutic potential of the mitochondria-specific antioxidant SkQ1 in the treatment of kidney anti-neutrophil cytoplasmic autoantibody (ANCA)-associated vasculitis (AAV) autoimmunity.

Background: Microscopic polyangiitis is a subtype of AAV and is characterised by loss of tolerance to the neutrophil protein myeloperoxidase. It frequently causes glomerulonephritis, leading to renal failure if treated inadequately. Current induction therapies such as cyclophosphamide, rituximab, and prednisolone are broadly immunosuppressive, leading to higher risk of infection; since the introduction of immunosuppressive treatments, infection has become the leading cause of mortality. Neutrophil extracellular traps (NETs) are increasingly recognised as key drivers of glomerular inflammation in AAV, and there are currently no treatments that directly target them. SkQ1 scavenges excess mitochondria-derived reactive oxygen species, leading to the reduction of NET formation, making it a promising targeted treatment strategy.

Methods: A 20-day experimental mouse model of AAV was used to compare the efficacy of SkQ1 with cyclophosphamide *in vivo*. Renal injury and leukocyte recruitment were assessed histologically, and serum ANCA levels were ascertained using an ELISA. The effects on neutrophil functions were also examined using human neutrophils *in vitro*. To assess the safety of SkQ1 and its immunosuppressive effects, a mouse influenza model was used to evaluate viral clearance and lung inflammation following prophylactic treatment.

Results: *In vivo*, SkQ1 reduced glomerular injury, leukocyte infiltration, and circulating ANCA levels (all $p < 0.05$). In vitro, SkQ1 inhibited NET formation of stimulated, but not resting, neutrophils, and it did not impair neutrophil phagocytic function. In influenza models, SkQ1 did not impair viral clearance or exacerbate lung inflammation, in contrast to cyclophosphamide.

Conclusion: SkQ1 alleviated structural glomerular damage in experimental AAV while preserving host defence against influenza. These findings support SkQ1 as a novel, NET-targeted induction therapy for AAV, with the potential to reduce infection-associated morbidity that is frequently present in current treatment regimes.

REMEMBRANCE DAY

ONE-MINUTE SILENCE, 11.00 am - 11.01 am

Dr Sheran Vasanthakumar, Victorian Heart Institute

"Coronary artery disease in women and novel imaging risk markers"



Dr Sheran Vasanthakumar is a cardiologist and cardiac imaging fellow at Monash Health and a PhD candidate at Monash University. His research focuses on improving cardiovascular risk prediction and prevention in women through novel imaging biomarkers. His PhD explores sex-specific differences in coronary atherosclerosis, the role of CT coronary plaque quantification, and the potential of breast arterial calcification on mammography to identify women at increased cardiovascular risk. His work aims to translate advanced imaging techniques into more personalised approaches to cardiovascular prevention.

Abstract

Cardiovascular disease remains the leading cause of death in women, yet many women at increased risk remain unidentified by traditional risk assessment. Breast arterial calcification (BAC), an incidental finding on routine mammography, represents a promising opportunity to identify cardiovascular risk through an existing population screening program. This PhD investigates novel imaging biomarkers to improve cardiovascular risk prediction in women, with a primary focus on the TIMTAM study, which aims to translate mammographic BAC assessment into clinical cardiovascular prevention.

TIMTAM is a multi-phase research program investigating the acceptability, feasibility and clinical utility of integrating cardiovascular screening into routine mammography. The initial co-design phase surveys women, mammography providers and clinicians to explore awareness, perceptions, potential uptake and barriers to implementing BAC-guided cardiovascular assessment. The clinical phase investigates the relationship between BAC, coronary atherosclerosis and previously unrecognised cardiovascular risk factors. Using automated BAC quantification alongside comprehensive cardiovascular assessment, the study aims to identify women who may benefit from earlier preventive intervention. A randomised clinical trial will compare BAC-guided enhanced cardiovascular risk assessment and management with standard care, evaluating whether this approach can improve cardiovascular risk identification and preventive management.

Supporting this translational program, analyses of the CAUGHT-CAD trial investigate sex-specific coronary plaque characteristics and responses to lipid-lowering therapy using serial coronary CT angiography. Findings demonstrate differences in plaque composition between women and men, with statin therapy associated with non-calcified plaque regression and increased calcium density. Complementary population-based research evaluates the prognostic significance of automated BAC measurements.

Together, these studies aim to establish the evidence and clinical pathway for incorporating cardiovascular prevention into breast screening, transforming routinely acquired mammograms into an opportunity for earlier, more personalised cardiovascular risk assessment in women.

Ruo Wang, Department of Molecular and Translational Sciences

Understanding the changes in intestinal stem cell function after enteropathogenic *Escherichia coli* infection



Ruo Wang completed her Master's degree at the Medical School of Zhejiang University, China, where she investigated microbiota-based therapies for inflammatory bowel disease (IBD) using human intestinal organoids. This experience sparked her interest in host-microbe interactions and their impact on intestinal health. In 2024, she joined Professor Elizabeth Hartland's laboratory at the Hudson Institute of Medical Research as a PhD student. Her current research focuses on understanding how enteropathogenic *escherichia coli* (EPEC) infection affects intestinal stem cell function in human intestinal organoid models.

Abstract

Ruo Wang[1, 2], Jiaxing Yang[1, 2], Cristina Giogha[1, 2], Eva Chan[1, 2], and Elizabeth Hartland[1, 2]

[1]Centre for Innate Immunity and Infectious Diseases, Hudson Institute of Medical Research, Clayton, Victoria, Australia.

[2]Department of Molecular and Translational Science, Monash University, Clayton, Victoria, Australia.

The intestinal epithelium is a primary interface for interactions with harmful agents and pathogens. Within this layer of cells, intestinal stem cells (ISCs) play a critical role in maintaining epithelial homeostasis and driving regeneration following damage. Enteropathogenic *Escherichia coli* (EPEC), a major cause of infant diarrhoeal disease in low- and middle-income countries, employs a type 3 secretion system (T3SS) to inject effector proteins that manipulate host signalling pathways and cause intestinal damage. However, the effects of EPEC infection on distinct intestinal epithelial cell populations, particularly ISCs, remain poorly understood, largely due to the limitations of current experimental models. Here, human intestinal organoids (HIOs) were used to characterise the epithelial transcriptional response to EPEC infection and determine its effects on intestinal stem cell function.

METHODS: HIO-derived monolayers enriched for stem/proliferative cells or differentiated cells were infected with wild-type EPEC O127:H6 strain E2348/69 (WT) or the isogenic mutants ΔPP4/IE6 (lacking seven effectors) and *ΔescN* (lacking a functional T3SS) for 3 hours, followed by antibiotic treatment. Bulk RNA-sequencing was performed at 3 and 27 hours post-infection to study transcriptional changes. To evaluate the proliferative capacity of stem cells following infection, infected stem monolayers were placed into 3D culture to study their ability to re-establish as organoids, where cell viability was quantified over time. These re-established organoids were then differentiated to assess the expression of markers of intestinal epithelial cell types.

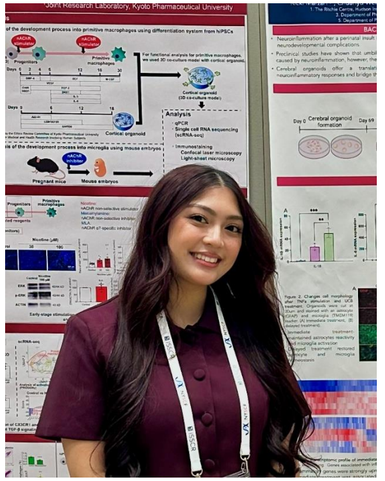
RESULTS: EPEC infection induced inflammatory responses in both organoid-derived monolayer culture systems. Transcriptomic analysis showed EPEC infection downregulated FOXM1-regulated G2/M checkpoint genes, with the strongest decrease observed with the ΔPP4/IE6 mutant, followed by WT EPEC and the *ΔescN* mutant. Organoid formation assays also showed a corresponding reduction in capacity of the cells to form organoids. Organoids from *ΔescN* and ΔPP4/IE6 groups showed increased expression of secretory lineage markers.

CONCLUSION: Human intestinal epithelial cells had different transcriptional responses to WT EPEC and the mutant derivatives. Beyond inducing inflammatory responses, EPEC infection downregulated the proliferative ability of intestinal stem cells and affected subsequent differentiation, highlighting potential mechanisms by which bacterial infection influences stem cell function.

Ricki Marzan, Department of Obstetrics & Gynaecology

Human Cerebral Organoids Reveal Timing-Dependent Inflammatory and Reparative Mechanisms of UCB Therapy in Perinatal Neuroinflammation.

Ricki Anne Solis Marzan is a third-year PhD candidate at The Ritchie Centre, supervised by A. Prof. Courtney McDonald. Using a cerebral organoid model of perinatal neuroinflammation, she investigates the molecular mechanisms of umbilical cord blood. She helped establish the use of cerebral organoids within her lab group, optimising culture conditions and workflows for downstream analysis. Her expertise includes immunofluorescence staining, qPCR, and functional assays such as multi electrode array analysis. Her research aims to uncover the mechanisms underlying the neuroprotective effects of umbilical cord blood, with particular focus on identifying cellular targets, affected signalling pathways and the impact of treatment timing on therapeutic responses.



Abstract

Background: Umbilical cord blood (UCB) cell therapy has demonstrated anti-inflammatory and neuroreparative effects in preclinical models of perinatal brain injury, but the mechanisms driving these benefits remain poorly understood. Human cerebral organoids (CO) offer a translational platform to investigate human-specific neuroinflammatory responses and bridge this mechanistic gap. This study aims to use a human CO model of perinatal neuroinflammation to investigate the cellular and molecular effects of UCB therapy and determine how treatment timing influences inflammatory and recovery pathways.

Methods: COs were generated from human induced pluripotent stem cells and co-cultured with human microglia (HMC3) from day (D)69. At D90, COs were assigned to control, TNF-a stimulated (50ng/mL) or TNF-a + UCB treatment groups receiving either immediate or delayed (72h) UCB administration ($5x10^6$ cells/CO). COs were analysed by immunofluorescence, qPCR and bulk-RNA sequencing.

Results: TNF-a stimulation induced activated microglial morphology, astrocyte hypertrophy and dendritic fragmentation. Immediate UCB treatment exacerbated the inflammatory response, maintaining activated glial morphology and significantly increasing IL-1B ($p=0.0053$) and IL-6 ($p=0.0066$) mRNA expression. In contrast, delayed UCB treatment attenuated inflammation, reducing IL-1B ($p=0.0541$) and significantly decreasing IL-6 ($p=0.0163$) relative to stimulated COs. Delayed treatment also restored control-like morphology, with ramified microglia and astrocytes, and neurons with extended dendrites. Transcriptomic analysis revealed enhanced cytokine- and interferon-driven inflammatory signalling following immediate treatment. Conversely, delayed treatment promoted homeostatic recovery, with activation of oxidative/cellular stress regulators and neuronal/synaptic remodeling pathways.

Conclusion: Human cerebral organoids provide a robust translational model of perinatal neuroinflammation and bridge mechanistic gaps left by preclinical studies. Our findings identify treatment timing as a critical determinant of UCB efficacy, with immediate treatment amplifying inflammatory signalling and delayed treatment promoting inflammation resolution, cellular recovery, and activation of repair pathways. Future studies will utilise microglia-free organoids and multi-omics, functional, and mitochondrial analyses to further define the pathways driving recovery.

Sasan Amanat, Department of Nutrition, Dietetics and Food

Diet and post-stroke cognition



Sasan is a third-year PhD candidate in nutrition and dietetics, with expertise spanning research, teaching and clinical practice. His research aims to identify dietary strategies that help stroke survivors preserve cognitive function and improve long-term health outcomes.
Using data from large cohort studies, population surveys and clinical trials, he investigates how nutrients, foods and dietary patterns influence cognition and brain health. With years of experience across multiple research disciplines, he is passionate about applying a multidisciplinary perspective to nutrition research.

Abstract

Background: Post-stroke cognitive impairment (PSCI) is a long-term disability affecting approximately 40% of stroke survivors. As the global incidence of stroke continues to rise, the number of people living with PSCI is expected to increase. Although diet is a well-established modifiable risk factor for stroke and cognitive decline, its role in cognitive recovery after stroke remains poorly understood. This PhD investigates the relationship between dietary factors and cognitive function in stroke survivors.

Method: This research comprises four studies. First, a systematic review and meta-analysis examined the associations between nutritional factors and cognitive outcomes among stroke survivors. Second, data from a cohort study were analysed to investigate the association between nut intake, cognition and markers of brain health, a relationship that has not previously been examined in stroke survivors. Third, baseline data from a clinical trial of stroke survivors in the sub-acute recovery phase were analysed to explore associations between diet, cognition, and gut microbiota composition. Finally, data from the US national survey will be used to examine dietary factors and cognitive function among stroke survivors in the chronic recovery phase (still underway).

Results: The meta-analysis suggested that energy-protein supplementation may improve cognitive outcomes, whereas B-vitamin supplementation showed no cognitive benefit. Nut intake was associated with better cognitive function in the stroke-free population; however, findings in stroke survivors were inconclusive. Moreover, in the sub-acute recovery phase, higher dietary fibre intake was associated with better cognitive function, and both fibre intake and cognition were related to gut microbiota composition.

Conclusion: These findings suggest that energy, protein, and fibre intake may be important components of post-stroke dietary strategies to preserve cognitive function. Future clinical trials are needed to determine whether optimising these dietary factors can causally improve cognitive outcomes after stroke.

Lunch Break

*Please return by 12:30 p.m. for the other presentations

Dr Chathuri Abeyakoon, Department of Medicine

Improving Outcomes in Peripheral T-cell Lymphoma: From real world evidence to molecular studies and clinical trials



Dr Chathuri Abeyakoon is a dual-trained clinical and laboratory haematologist at Monash Health specialising in lymphoid malignancies and novel therapeutics. She graduated with Honours from the University of New South Wales and holds fellowships from both the Royal Australasian College of Physicians (FRACP) and the Royal College of Pathologists of Australasia (FRCPA). She completed a two-year clinical and research fellowship at Princess Margaret Cancer Centre, Toronto, Canada, serving as Chief Fellow in lymphoma, myeloma and autologous transplantation. Her PhD focuses on improving outcomes in rare and challenging to treat lymphoid malignancies through clinical research, with an emphasis on developing more effective treatment strategies.

Abstract

Peripheral T-cell lymphomas (PTCLs) are biologically heterogeneous lymphoid malignancies characterised by poor survival and limited therapeutic options. This research program integrates international cohort and registry analyses, molecular profiling and prospective clinical investigation to improve prognostication and outcomes. The broader program also examines aggressive B-cell lymphoid malignancies; however, the work presented here focuses on the PTCL component.

An Australasian–Canadian cohort of 451 patients was established using data from the Australasian Lymphoma and Related Diseases Registry and the Princess Margaret Cancer Centre lymphoma database. Analyses characterised treatment patterns, responses and survival across major nodal PTCL subtypes. Three-year progression-free and overall survival were approximately 26% and 55%, respectively, demonstrating substantial unmet need. Additional analyses examined outcomes in older patients and treatment heterogeneity.

Within this cohort, end-of-treatment PET/CT complete remission was strongly associated with improved progression-free and overall survival outcomes, supporting its prognostic value. Further work proposes a definition of ‘primary refractory nodal PTCL’ encompassing stable or progressive disease following frontline treatment or relapse within six months of treatment completion. External validation using the International T-cell Project 2.0 cohort will assess its reproducibility and potential utility as a clinical trial endpoint.

Complementary genomic and transcriptomic profiling studies investigate the molecular landscape of PTCL to identify therapeutic targets and explore treatment sensitivities. Integrating molecular findings with clinical outcomes aims to support biologically informed patient selection and personalised therapeutic strategies.

Finally, the investigator-initiated ZiVLen prospective phase II trial, which I am leading as a co-chief investigator, is due to open to recruitment to evaluate zilovertamab vedotin, a ROR1-directed antibody–drug conjugate, combined with lenalidomide in relapsed or refractory PTCL. Embedded translational studies will examine tumour biomarkers, the immune microenvironment and dynamic treatment responses to identify predictors of benefit and inform future treatment selection. Together, these studies connect real-world evidence with molecular discovery and prospective therapeutic evaluation, providing a framework for improving risk stratification, defining treatment failure and advancing personalised care for patients with rare and challenging PTCLs.

Nigus Yimer, Monash Centre for Health Research and Implementation (MCHRI)

Advancing Precision Medicine in Women's Health Through Personalised Risk Prediction and Causal Inference



Nigus Yimer is a PhD candidate at Monash Centre for Health Research and Implementation, Monash University. His research focuses on advancing precision medicine in women's health through clinical prediction modelling, machine learning, and causal inference. He holds a Bachelor's degree in Midwifery, a Master's degree in Clinical Midwifery, and a Master's degree in Epidemiology. His PhD aims to improve personalised prevention and clinical decision-making for conditions affecting women across the reproductive lifespan, including type 2 diabetes after gestational diabetes, premature ovarian insufficiency, and early menopause. Nigus has published extensively in maternal, fetal, and women's health and has been awarded competitive scholarships in both Belgium and Australia.

Abstract

Gestational diabetes mellitus (GDM), premature ovarian insufficiency (POI), and early menopause are associated with substantial long-term health consequences in women, including an increased risk of type 2 diabetes (T2D) and cardiovascular incidents. Despite growing interest in precision medicine, the availability of robust, clinically meaningful tools to identify women at risk remains scarce due to methodological limitations in existing clinical prediction models and challenges in estimating causal effects from observational data. This PhD program aims to advance precision medicine in women's health through integrating contemporary prediction modelling, machine learning, and causal inference approaches to improve personalised prevention and clinical decision-making.

The PhD research comprises four interconnected streams. First, evidence synthesis to identify and critically appraise existing prediction models in reproductive and metabolic health, identifying methodological limitations and evidence gaps. Second, methodological research applies and compares sample size methods for developing clinical prediction models using large real-world perinatal datasets. Third, statistical and machine learning approaches are applied to develop, externally validate, and update prediction models for important maternal and women's health outcomes, including type 2 diabetes following gestational diabetes, premature ovarian insufficiency, early menopause, and caesarean section after induction of labour. Finally, modern causal inference methods, including double machine learning and causal forests, are employed to estimate causal effects and investigate treatment/exposure heterogeneity in perinatal and women's health outcomes.

The program has already generated key outputs, including published and accepted methodological and evidence synthesis studies, with multiple applied modelling and causal inference projects currently underway. By combining rigorous methodology with real-world clinical data, this research will produce more reliable, interpretable, and clinically relevant tools for risk prediction and causal decision-making. Ultimately, the findings aim to strengthen personalised prevention strategies, improve clinical care, and contribute to evidence-based policy for women's health.

Yaoxi Wu, Department of Surgery

Targeting HSP90 suppresses bladder cancer cells and reduces tumour burden in an orthotopic NMIBC model



Yaoxi Wu is a China-trained urologist with extensive clinical and research experience across a range of urological malignancies. He is currently a PhD candidate in Surgery Monash Health. His research focuses on immunotherapy for non-muscle-invasive bladder cancer (NMIBC), particularly the mechanisms underlying BCG treatment failure and the development of new therapeutic strategies for BCG-unresponsive disease. By integrating clinical experience with laboratory research, Yaoxi aims to develop more effective and clinically relevant immunotherapies for patients with treatment-resistant bladder cancer.

Abstract

Bladder cancer is a major global health burden, with overall survival has not improved over the past decade. Approximately 75% of patients initially present with non-muscle-invasive bladder cancer (NMIBC), which has a high recurrence rate and a risk of progression. For patients with recurrent or BCG-unresponsive NMIBC, effective bladder-preserving treatment options remain limited, while radical cystectomy carries substantial morbidity. Our previous proteomic work identified heat shock protein 90α (HSP90α) as a potential therapeutic target in NMIBC. Because intravesical therapy is central to NMIBC management, we evaluated whether a clinically relevant 1-hour exposure to the HSP90 inhibitor ganetespib (STA-9090) produces sustained antitumour activity in vitro and whether intravesical STA-9090 is active in an orthotopic NMIBC model. Human T24 and 5637 bladder cancer cells, non-malignant immortalised SV-HUC-1 urothelial cells and murine MB49-Luc bladder cancer cells were exposed to STA-9090 for 1 hour, followed by drug-free medium; viability was assessed at 72 hours and additionally at 120 hours for MB49-Luc cells. Human-cell experiments were performed independently four times. We established an orthotopic bioluminescent NMIBC mouse model using MB49-Luc cells, providing a preclinical platform for evaluating intravesical therapies. Mice bearing orthotopic MB49-Luc tumours received three intravesical instillations of vehicle (n=4), 1 μM STA-9090 (n=5) or 10 μM STA-9090 (n=5). Tumour burden was assessed using longitudinal bioluminescence imaging (BLI), overall survival and endpoint bladder weight. In vitro, mean 72-hour IC_{50} values were 284.0 ± 66.5 nM for T24, 340.1 ± 54.8 nM for 5637 and 425.6 ± 22.4 nM for SV-HUC-1 cells. Both human tumour cell lines had lower IC_{50} values than SV-HUC-1 across all experiments (overall $p=0.011$), while MB49-Luc cells showed sustained suppression at 72 and 120 hours. In vivo, longitudinal BLI differed significantly among groups ($p<0.0001$), with 10 μM STA-9090 producing the lowest post-treatment tumour signal. Median survival was 16 days with vehicle and 27 days with 1 μM STA-9090, whereas median survival was not reached by day 29 with 10 μM STA-9090. Among animals available at the research endpoint, bladder weight was lowest in the 10 μM group, although the small numbers precluded inferential analysis. These findings support further evaluation of intravesical HSP90 inhibition, including combination treatment with BCG.

Barnaby Kelly, Department of Molecular and Translational Sciences

Custom CRISPR screening and multi-omic data identify targetable drivers of cisplatin resistance in paediatric osteosarcoma



Barnaby Kelly is a 3rd-year PhD student from New Zealand with a background in pharmacology and toxicology, currently researching the molecular mechanisms that drive chemoresistance in paediatric osteosarcoma. My work has utilised whole-genome CRISPR screening to generate a custom cisplatin resistance sgRNA library that we have used to discover drivers of cisplatin resistance in OS.

Paediatric osteosarcoma is a complex disease with high inter- and intra-tumoral heterogeneity, which contributes to the lack of identifiable resistance drivers. To date, we have identified several targets and are investigating their role in increasing not only cisplatin responses but also responses to other mainline osteosarcoma chemotherapies. My research aims to understand these mechanisms and, through complex, clinically relevant in vivo models, provide a framework and rationale for translating these repurposed and novel therapies into the clinic.

Abstract

Introduction:

Osteosarcoma (OS) is the most common primary bone tumour in children and young adults. Resistance to MAP chemotherapy (methotrexate, doxorubicin, cisplatin) represents an unmet clinical need. Intrinsic resistance underlies poor neoadjuvant chemotherapy response, the strongest independent predictor of survival, while acquired resistance drives relapse. No biomarkers or effective therapies currently exist for refractory patients.

Materials & Methods:

Whole genome CRISPRko screens were performed in cisplatin-resistant OS cell lines using Humagne Set C & D libraries, with and without cisplatin (0.2µg/mL). Custom gRNA validation libraries were applied to a wider panel of OS cell lines. Resistance drivers were validated in isogenic cell lines and by pharmacological inhibition in vitro. Concurrently, in vivo resistant models were developed through repeated cisplatin treatment (6mg/kg, ip) in tumour-bearing NSG mice, and characterised by whole genome sequencing, RNA sequencing, and proteomics.

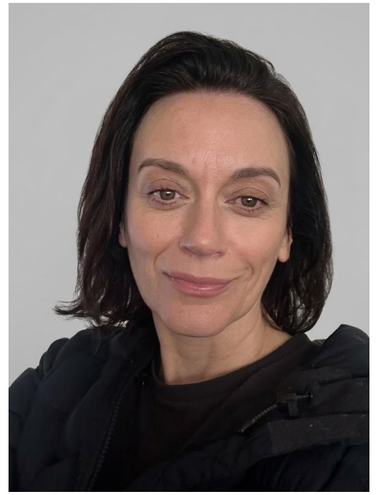
Results & Conclusions

Unbiased genetic screens revealed ATR, FAAP24 and UBE2T as potent regulators of cisplatin resistance. These targets for cisplatin resistance were validated across 4 OS cell models using a custom gRNA library. Pharmacological inhibition of FAAP24/FANCM interaction and UBE2T resulted in an additive effect. Pharmacological inhibition of ATR using berzosertib and ceralasertib synergised with cisplatin and reduced the IC50 tenfold across a panel of OS cell lines. In a cisplatin-resistant subcutaneous OS xenograft model, berzosertib potentiated cisplatin response, leading to reduced tumour volume and increased survival. Consistent with a role for ATR in OS cisplatin resistance, DNA damage repair pathways regulated by ATR, including homologous recombination (HR), Fanconi anaemia and nucleotide excision repair, were enriched in novel acquired cisplatin-resistant in vivo OS models.

ATR is a regulator of cisplatin response and resistance, which, along with UBE2T and FANCM, plays key roles in Fanconi anaemia and HR. Inhibition of ATR using berzosertib potently sensitises resistant OS cells to cisplatin and may have therapeutic benefit. DNA damage repair is crucial for cisplatin function, and through inhibition of this crucial process, increased cisplatin efficacy can be achieved at lower doses. Overall, our approach has revealed candidate mechanisms and biomarkers of cisplatin resistance, providing a framework for improving chemotherapy responses in OS.

Linda Hayes, Department of Psychiatry

Designing a model of care in cervical cancer prevention with and for women with Severe Mental Illness (SMI)



Linda Hayes is a second-year PhD student within the Department of Psychiatry and the recipient of a Safer Care Victoria Translational Research Fellowship. My research focuses on improving cervical cancer prevention for people living with severe mental illness. Working alongside consumers, clinicians and key stakeholders, I am investigating how effective screening and vaccination programs can be adapted to address persistent health inequities. At its heart, my research tackles a key implementation challenge: how can the benefits of National screening and vaccination programs be extended to this population group to improve access, uptake and health outcomes?

Abstract

People with severe mental illnesses (SMI), including schizophrenia, schizoaffective disorder, bipolar disorder, severe depression, and other psychotic illness, experience substantially poorer cancer outcomes compared to the general population. For example, women with psychotic disorders have a higher incidence of cervical cancer and are more likely to present with advanced disease. Yet people with SMI experience multiple barriers to cervical cancer prevention. This means that despite the effectiveness of cervical cancer prevention programs, people with SMI are less likely to participate and opportunities for prevention and early detection may be being missed.

Aim: To improve participation in cervical cancer prevention with and for people with SMI.

Methods: This program of research includes: (1) a systematic review comparing uptake of cervical cancer prevention among people with and without mental illness; (2) a linked-data study to generate robust estimates of participation in cervical cancer prevention within a large Australian health service; (3) qualitative interviews examining implementation determinants at individual, service, and system levels; and (4) development of recommendations through a Delphi consensus process.

Results: Existing evidence demonstrates consistently lower participation in cervical cancer prevention among people with SMI. However, important knowledge gaps remain regarding follow-up care after screening, uptake of HPV vaccination, and the impact of recent screening innovations. The planned studies will improve understanding of barriers and facilitators influencing participation and identify strategies to improve access and equity.

Conclusion: As Australia progresses towards cervical cancer elimination, population-level gains may mask persistent inequities. Cervical cancer prevention programs are effective, but equitable access remains challenging. Without targeted action to support people with SMI, disparities in cervical cancer outcomes may persist or worsen.

PLEASE VOTE!

People’s Choice Award Voting Form

THANK YOU

Conclusion of the PhD Student Showcase Symposium.

Winners to be announced at the end of the event !