ASMR
AUSTRALIAN SOCIETY FOR MEDICAL RESEARCH

ASMR NSW Annual Scientific Meeting

THURSDAY, 10 SEPTEMBER 2026

CHILDREN'S CANCER INSTITUTE
HEALTH TRANSLATION HUB

Get in touch
+61 (02) 9230 0333 nsw@asmr.org.au www.asmr.org.au/nsw/

ACKNOWLEDGEMENT OF COUNTRY

We acknowledge the traditional owners of the land that we meet on today and pay our respects to the Gadigal people of the Eora Nation. We also acknowledge that our research institutes are located on the lands of many Traditional Custodians in Victoria and around Australia. We recognise their ongoing connection to the land and contributions to research, university and wide Australian society. We pay our respects to Indigenous Elders, past, present and emerging and will continue to acknowledge their living culture and their continuing roles in the life of this region.

The ASMR's vision is for a healthy and equitable Australia. There is no doubt that a significant gap remains between the health outcomes of First Nations people and other Australians, and the ASMR will continue to work towards closing this gap.

The ASMR believes that our diversity is our strength: health and medical research belongs to everyone, and everyone belongs in health and medical research.

ASMR
AUSTRALIAN SOCIETY FOR MEDICAL RESEARCH

ABOUT THE AUSTRALIAN SOCIETY FOR MEDICAL RESEARCH

The Australian Society for Medical Research (ASMR) is a non-politically aligned, non-profit organisation established in 1961. ASMR is the peak organisation representing health and medical researchers in Australia through public, political, and scientific advocacy. ASMR's members include individuals from diverse career stages and research areas, as well as affiliate and associate members drawn from specialist medical societies, medical colleges and institutes, and consumer groups.

The ASMR is dedicated to achieving a secure and sustainable health and medical research workforce to facilitate increased productivity in Australian health and medical research.

ASMR's activities focus on:

- fostering excellence in health and medical research
- expanding the interface between basic science and clinical research
- promoting community understanding and support
- holding annual scientific meetings, including our National Scientific Conference and state Scientific Meetings
- creating opportunities for Australian health and medical researchers through the ASMR Research Awards and student travel grants
- keeping members up to date about medical research activites around Australia through our eBulletin
- offering Professional and Career Development Programs
- helping to foster new relation between current members

Become an ASMR Member today!

Scan for further information regarding ASMR membership!

OUTLINE OF ASMR NSW ANNUAL SCIENTIFIC MEETING PROGRAM

8:00 - 8:45 am`	REGISTRATION
9:00 - 9:15 am	Acknowledgement of Country Conference overview
9:15 - 10:00 am	Plenary Presenter Professor Jürgen Götz
10:00 - 11:00 am	Oral Session 1
11:00 - 12:00 pm	Morning Tea & Poster Session
12:00 - 1:00 pm	Oral Session 2
1:00 - 2:15 pm	Lunch & Networking
2:15 - 3:00 pm	Plenary Presenter Professor Victoria Cogger
3:00 - 4:00 pm	Oral Session 3
4:00 – 4:20 pm	Afternoon Tea
4:20 - 4:55 pm	Alan Skyring Memorial Award Session
4:55 – 5:10 pm	Awards
5:10 – 5:15 pm	Closing Remarks & Meeting Close

A MESSAGE FROM THE ORGANISING COMMITTEE

Dear Delegates,

On behalf of the ASMR NSW Committee, we look forward to welcoming you to the 2026 ASMR NSW Annual Scientific Meeting.

It is with great pleasure that I welcome you to the 34th ASMR NSW Annual Scientific Meeting. The Scientific Meeting highlights the achievements of NSW medical researchers across all areas of health and medical research. It encourages scientists to look beyond their own fields, engage with the wider NSW research community, and explore interdisciplinary approaches to medical research.

We have an outstanding line up including internationally renowned plenary speakers, a broad range of talks from research assistants, Honours and PhD students, early career researchers and mid-career researchers as well as poster presentations.

I would like to thank our generous sponsors for their support in what has been a challenging year for all businesses, universities and research institutes and I encourage you to visit the trade displays throughout the day.

We would also like to let you know that registration are open for the upcoming 65th ASMR National Scientific Conference to be held in Melbourne in November.

If you are not a member of ASMR, please consider joining. Help support our activities in promoting Australian health and medical research, and our roles in public, political and scientific advocacy. Visit the ASMR website at www.asmr.org.au, follow us on Twitter (@theASMR1), attend the Twitter conference using #ASMRNSW2026 and like us on Facebook (theASMR), to receive regular updates of ASMR activities and important issues affecting health and medical researchers.

Your feedback is very important to us. Please let us know what you think of this year's meeting and make suggestions for next year by completing our feedback questionnaire.

We wish all delegates an enjoyable and rewarding conference.

Organising Committee,
ASMR NSW Scientific Meeting

ASMR
AUSTRALIAN SOCIETY FOR MEDICAL RESEARCH

ASMR NSW COMMITTEE 2026

Dr. Angelica Merlot

Dr. Jason Girkin

Dr. Michelle Stubbs

Dr. Camille Esneau

Esmaeil Roohparvar Basmenj

Issac Findley

Neha Neha

Dr. Ashleigh Paparella

Dr. Dan Enosi Tuipulotu

Dr. Ramon Martinez Marmol

Dr. Ellen Donohoe

Dr. Aaminah Khan

Aqsa Mazhar

Dr. Amiee Dowdell

Dr. Gustavo Espinoza Vergara

Dr. Ernest Moles Meyer

Nathan Bryant

2026 ASMR Partners

THE UNIVERSITY OF NEWCASTLE AUSTRALIA

UNSW SYDNEY BABS

Exclusive sponsor of the ASMR Medallist in NSW

the GrantEd Group

VENUE SPONSOR OF ASMR NSW SCIENTIFIC MEETING 2026

Children's Cancer Institute

SILVER SPONSOR OF ASMR NSW MEDICAL RESEARCH WEEK® 2026

UNSW SYDNEY

School of Biomedical Sciences
Faculty of Medicine & Health

BRONZE SPONSOR/S OF ASMR NSW RESEARCH WEEK® 2026

Victor Chang Cardiac Research Institute

NSW ANNUAL SCIENTIFIC MEETING EXHIBITOR SPONSORS

the GrantEd Group

CHILDREN'S MEDICAL RESEARCH INSTITUTE

Jeans for Genes®

NEW ENGLAND Biolabs®

SARSTEDT

Pathtech
First Choice In Science

UNITED BIO RESEARCH

ASMR
AUSTRALIAN SOCIETY FOR MEDICAL RESEARCH

UNSW SYDNEY
BABS

Team BABS vision

We excite, educate, inspire and advance knowledge in molecular biosciences to address grand challenges and create opportunities.

More than a School....

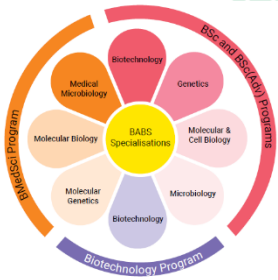
At the School of Biotechnology and Biomolecular Sciences, we are a community united by passion, collaboration, and excellence. We value diversity and celebrate the uniqueness of each member. Students, faculty, and staff work together to foster curiosity, innovation, and belonging.

We believe an institution's strength lies in its people.

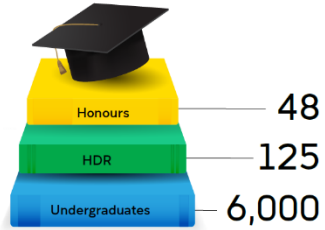
Our focus is on the well-being and development of everyone, offering academic opportunities, mentorship, support, and lasting connections. Our inclusive environment encourages learning, growth, and personal development.

We are more than just a school — we nurture leadership, creativity, and groundbreaking science. Our goal is to develop not only skilled scientists but individuals who will make meaningful contributions
to society and drive innovation for the betterment of the world.

What we Teach



Students



Vision for research

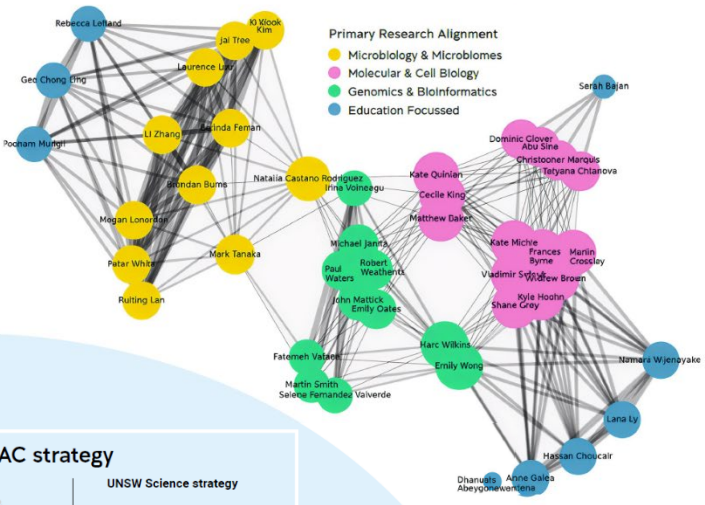
To foster research excellence in BABS through ensuring stable funding (before), excellent research infrastructure and institutional support (during), high impact publications (after) and providing support and strategic advice to the BABS leadership team and BABS research community

– Director of Research A/Professor Cecile King

What does success look like for research

- Keep supporting the BABS research community and BABS leadership through a range of initiatives
- Explore additional funding sources to deal with low Cat1 funding rates
- Continue to build relationships with other UNSW Schools to enable collaboration and ideas sharing.

Team BABS research & Similarity Network (weighted features)



BABS place in the UNSW and SCIFAC strategy

UNSW strategy

6 Pillar 6: Accelerate the transition to a sustainable society and planet

7 Pillar 7: Advance economic and social prosperity

8 Pillar 8: Enable healthy lives

9 Pillar 9: Strengthen societal resilience, security and cohesion

UNSW Science strategy

Addressing grand challenges through our science attractors

- Building a Liveable and Healthy Future
- Healthy Brains, Healthy Minds

BABS.unsw.edu.au

Victor Chang
Cardiac Research Institute

SCAN TO LEARN MORE

The home of heart research fighting heart disease.

Every day, researchers at the Victor Chang Cardiac Research Institute are making discoveries that improve the prevention, diagnosis and treatment of cardiovascular disease.

Through world-leading research, innovation and collaboration, we are working towards a future where fewer lives are lost to heart disease and more families have time together.

Discover the research, breakthroughs and people helping shape the future of heart health.

www.victorchang.edu.au

VictorChangInst

VictorChangInst

CHILDREN'S MEDICAL RESEARCH INSTITUTE

Jeans for Genes®

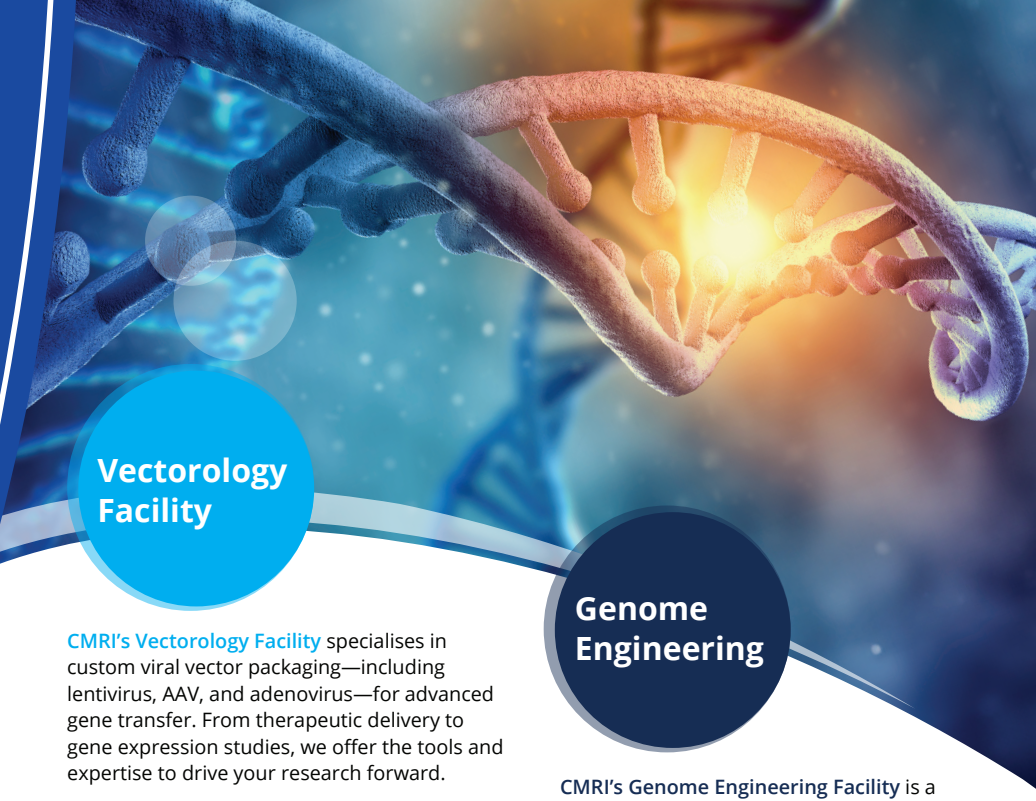
RESEARCH FACILITIES

Children's Medical Research Institute (CMRI) is world-renowned for our work on cancer, gene therapy, telomeres, and stem cells. We operate multiple research facilities to help advance our cutting-edge discovery and translational research but also to benefit our research collaborators and the wider scientific community.

Your research Accelerated

Josh Studdert
Research Facilities Manager
Ph: 02 8865 2965
E: jstuddert@cmri.org.au

cmrifacilities.com



Vectorology Facility

CMRI's Vectorology Facility specialises in custom viral vector packaging—including lentivirus, AAV, and adenovirus—for advanced gene transfer. From therapeutic delivery to gene expression studies, we offer the tools and expertise to drive your research forward.

Our Services Include:

- AAV Production – Medium to large scale
- Lentivirus Production – Crude and purified formats
- Viral Titre Analysis – Using advanced ddPCR technology
- Plasmid Services – Cloning, transformations, and purification
- Expert Consultation – Tailored guidance for your project

Genome Engineering

CMRI's Genome Engineering Facility is a one-stop shop for custom genome editing in immortalised cells, primary human/murine cells, and iPSCs—supporting fundamental and preclinical research.

Our Services Include:

- Knock-out, knock-in & prime editing (bulk or clonal)
- CRISPRa/i functional screens
- Genome-scale gRNA libraries

Pathtech
First Choice In Science

AUSTRALIA'S *FIRST CHOICE* IN LIFE SCIENCE FOR OVER 40 YEARS

- LIQUID HANDLING
- PIPETTE TIPS
- NATA ACCREDITED LAB
- SAMPLE STORAGE
- BENCHTOP EQUIPMENT
- PLASTIC CONSUMABLES

OUR TRUSTED PARTNERS

SARTORIUS

NEPTUNE SCIENTIFIC

Pathtech CalLab
First Choice in Calibration

Benchmark Scientific

LVL technologies

DiData

WATSON BIO LAB
MADE IN JAPAN SINCE 1988

Genesee Scientific
A Life Science Company

Australian Biostain
a CSA Pathology brand

RATEK

phasetwo

PATHTECH.COM.AU | 1800 069 161

Your Partner in Medicine & Science Worldwide

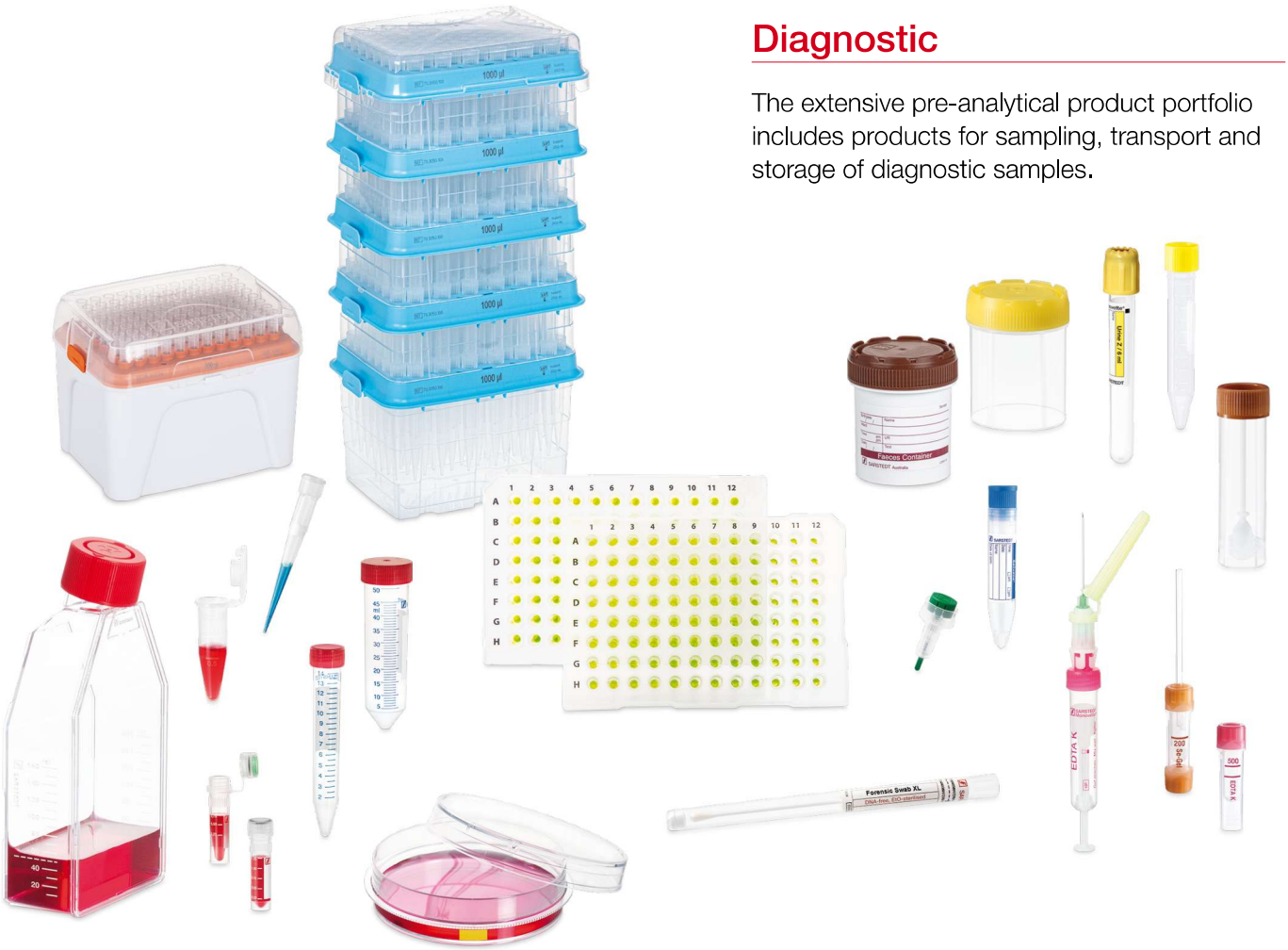
WE KNOW LABORATORY NEEDS.

Laboratory

We offer tailor-made product solutions for the most varied areas of use, including molecular biology, biochemistry and cell biology.

Diagnostic

The extensive pre-analytical product portfolio includes products for sampling, transport and storage of diagnostic samples.



customerservice.au@sarstedt.com • www.sarstedt.com

SARSTEDT

SARSTEDT Australia Pty. Ltd. · 16 Park Way, Mawson Lakes · South Australia, 5095 · Tel: +61 8 8349 6555 · Fax: +61 8 8349 6882

ASMR
AUSTRALIAN SOCIETY FOR MEDICAL RESEARCH

ASMR 65TH NATIONAL SCIENTIFIC CONFERENCE

Melbourne | 16-18 Nov 2026

Abstracts sought from all areas of health and medical research and all career stages for this truly multidisciplinary conference.

Including, but not limited to:

- Foundational & Translational Research
- Innovation, Technology & Discovery
- Health Systems, Mental Health & Society
- Clinical & Applied Research
- Research Culture, Training & Careers
- Industry, Commercialisation & Partnerships
- Global & Planetary Health
- Science Communication & Advocacy

2026 ORATORS

Prof Madhu Bhaskaran
RMIT University
Functional Materials & Microsystems

Prof Yugeesh Lankadeva
The Florey
Stroke & Critical Care

Dr Fernando Gordillo Altamirano
Alfred Health | Monash University
Antimicrobial Resistance & Bacteriophages

Registration includes FREE access to high-quality professional development workshops:

- Science Communication
- Grant Writing and Funding
- Career Pathways in Research
- AI in Research

PLUS enjoy a night of science-inspired fun, networking and great company!

Over $8,000 in prizes available for ASMR members

SUBMIT ABSTRACTS BY 21 SEP

Registration

ASMR Member Students: $225
Non-Member Students: $340
ASMR ECR/MCR Members: $310
Non-Member ECR/MCRs: $510
single & 2-day registrations also available

Register

With thanks to our Victorian Event Sponsors

THE UNIVERSITY OF MELBOURNE

murdoch children's research institute

Trade & Digital Supporters

agrf Stago NEW ENGLAND Biolabs

nsc@asmr.org.au call or text ASMR on 0499 764 165 www.asmr.org.au/nsc/

PLENARY SPEAKER

PROFESSOR JÜRGEN GÖTZ

Director of Clem Jones Centre
for Ageing Dementia Research
(CJCCADR)

Queensland Brain Institute
The University of Queensland

Contact details:
j.goetz@uq.edu.au

Professor Jürgen Götz (PhD, Dr. habil, FAA, FAHMS, GAICD) is Foundation Chair of Dementia Research, Director of the Clem Jones Centre for Ageing Dementia Research (CJCADR) at the Queensland Brain Institute (University of Queensland), Director of an NHMRC CRE (Centre of Research Excellence), NHMRC Leadership Fellow and Ultrasound Team Leader. In 2023 he became the Lesleigh Green - Bill and Nancy Green Endowed Chair in Dementia Research.

Jürgen Götz performed undergraduate studies at the Biocenter of the University of Basel, before joining the laboratory of Nobel Laureate Georges Köhler to obtain his PhD degree in immunology. Subsequently, he took up postdoctoral positions at UCSF (San Francisco) and Sandoz Ltd (now Novartis, Basel), and worked as Research Group Leader at the University of Zurich (Switzerland). Before taking up his current position, Jürgen Götz was a Professor and Chair of Molecular Biology at the University of Sydney.

A major focus of the Götz laboratory is to understand how tau and amyloid separately and synergistically contribute to Alzheimer's disease. Prof Götz has published more than 240 papers in leading journals including Cell, Science and Neuron, and has authored many authoritative reviews in the Nature Reviews journal family. The laboratory further explores therapeutic ultrasound as a treatment modality for Alzheimer's disease and other brain diseases, both by transiently opening the blood-brain barrier and as neuromodulatory tool. His team has built a clinical trial-ready therapeutic ultrasound device, completed a first-in-human safety trial in Alzheimer's disease patients and initiated a Phase 2 trial with a second-generation device. Moreover, his work resulted in the start-up Ceretas that has been spun out of UQ, with Ceretas being listed on the ASX in July 2026.

PLENARY SPEAKER

PROFESSOR VICTORIA COGGER

Executive Director of the Sydney Biomedical Accelerator

The University of Sydney

Contact details:
victoria.cogger@sydney.edu.au

An international research leader in ageing liver and targeted drug delivery with a strong track record in research commercialisation, Professor Cogger was appointed founding Executive Director of the Sydney Biomedical Accelerator in March 2026. This visionary partnership between the University of Sydney, NSW Government and Sydney Local Health District has attracted $780M investment to establish a globally significant hub for biomedical research, innovation and translation in Sydney.

Professor Cogger undertook both her undergraduate and PhD studies with the University of Sydney. She was awarded a Healthy Ageing Postdoctoral Fellowship and travelled to the National Institutes of Health, USA to complete her postdoctoral studies. She is a professor in the University's Faculty of Medicine and Health, and continues to lead a research team focused on understanding the biology of the liver relating to ageing and disease, and translating this knowledge to develop targeted nanomedicines for disease prevention and treatment.

In 2023, Professor Cogger co-founded spinout company Endo Axiom to lead the development of nanomedicines for clinical use. A culmination of 20 years of research by Professor Cogger and colleagues has seen its lead product, nanotechnology-based oral insulin for diabetes treatment, progress to phase 1 clinical trials.

In 2026, she was recognised by the National Health and Medical Research Council in their 17th edition of *10 of the Best* for her project 'Turning back the clock on ageing with nanomedicines.'

Prior to her appointment with the Sydney Biomedical Accelerator, Professor Cogger held several leadership positions across the University of Sydney and Sydney Local Health District, including Director of the ANZAC Institute, Co-Director of the District's Centre for Education and Research on Ageing, and Associate Dean (Research Education) in the University's Faculty of Medicine and Health.

ORAL SESSION 1: PRESENTATION SCHEDULE

Session Chair: Dr. Ellen Donohoe

Time	Speaker	Presentation Title	Affiliation
10:00 – 10:10	Isabella Moore	An integrated tumouroid and multi-omics platform for therapeutic discovery and testing in drug-resistant breast cancer	*The University of Newcastle*
10:10 – 10:20	Elsa Kalluzhathil	IRGM, a key player in gastric carcinogenesis	*University of New South Wales*
10:20 – 10:30	Aritra Ghosh	Pathogenic Moraxella species trigger diverging inflammasome pathways	*The Australian National University*
10:30 – 10:40	Ashley Abe	An evolutionary scavenger (receptor) hunt to explain SMuRF-less Coronary Artery Disease	*The University of Sydney*
10:41 – 10:45 Lightning	Poonam Jadhav	Guanylate-binding proteins restrain cellular proliferation during tumorigenesis	*The Australian National University*
10:46 – 10:50 Lightning	Janhavi Mahajan	Supporting longer platelet storage for transfusion: stacking maintains platelet quality over 21 days in the cold	*Australian Red Cross Lifeblood*
10:51 – 10:55 Lightning	Michelle Wang	Physiotherapists experiences with cognitive behavioural therapy for insomnia (CBT-I) training: mixed methods study	*The University of Sydney*
10:56– 11:00 Lightning	Yuan Fang	Development of novel collagen-modified contact lenses for sustained ciprofloxacin delivery in bacterial keratitis	*The University of Sydney*

ORAL SESSION 1: SCIENTIFIC ABSTRACT #1

An integrated tumouroid and multi-omics platform for therapeutic discovery and testing in drug-resistant breast cancer

Isabella G. Moore[1,2], Madeleine Walton-Smith[1,2], Severine Roselli[1,2], Vimala Anthonydhason[1,2], Nikita Panicker[1,2], Lauren F. Watt[1,2], Joshua S. Brzozowski[1,2], Heather C. Murray[1,2], Nicole M. Verrills[1,2]

1. School of Biomedical Sciences and Pharmacy, College of Health, Medicine and Wellbeing, The University of Newcastle, Callaghan, NSW, Australia
2. Precision Medicine and Health Research Program, Hunter Medical Research Institute, New Lambton Heights, NSW, Australia

Keywords: Breast cancer. Tumouroids. Omics. Therapeutic resistance. Targeted therapies.

Introduction: Therapeutic resistance causes treatment failure for many breast cancer patients. The HER2-positive breast cancer subtype has the highest relapse rate as targeted therapy resistance occurs in 30-40% of patients. But it remains unclear why breast cancer patients acquire targeted therapy resistance and how to overcome this.

Objective: To develop a breast cancer therapy discovery pipeline by incorporating omics analysis for target identification with tumouroid drug efficacy testing.

Methods and Results: We utilised a HER2-driven breast cancer mouse model with mammary-specific conditional knockout of the tumour suppressor PP2A-B55a (*Ppp2r2a*) which is associated with drug resistance and poor breast cancer outcomes. Using wild-type, heterozygous and homozygous *Ppp2r2a* knockout breast tumours from this mouse model we established and characterised an in vitro breast tumouroid model. Tumouroids matched the histological structure and HER2, CK8 and KI67 breast cancer marker expression of mouse tumours. Additionally, differences in tumouroid morphology and growth rate were observed between wild-type and *Ppp2r2a* knockout tumouroids. We then developed a tumouroid drug screening platform for breast cancer therapeutic efficacy testing. All tumouroids showed similar dose-dependent responses to the oestrogen receptor modulator tamoxifen, HER2 inhibitor lapatinib, EGFR inhibitor erlotinib and PP2A activator FTY720 validating the applicability of this model. Meanwhile, combination FTY720 and tamoxifen synergistically reduced tumouroid growth, suggesting FTY720 may improve tamoxifen's effectiveness in breast cancer. Next, we performed phosphoproteomics and RNAseq analysis of breast tumours from each mouse model genotype. This revealed PP2A-B55a loss was associated with alterations in cancer-associated signalling pathways including hedgehog, MAPK/ERK, HIPPO, mTOR, oestrogen and MYC signalling, indicating potential targets for treatment.

Conclusions: We established the first mouse *Ppp2r2a* knockout HER2-positive breast tumouroid drug screening model. Additionally, phosphoproteomics and transcriptomics analysis unveiled potential therapeutic targets that we can now test using our tumouroid drug screening platform to find new targeted therapies for resistant breast cancer.

ORAL SESSION 1: SCIENTIFIC ABSTRACT #2

IRGM, a key player in gastric carcinogenesis

Elsa Kalluzhathil[1], Laurence Luu[1], Isidora Simovic[1], Khean-Lee Goh[2], Kwong Ming Fock[3], Shaghayegh Baradaran Ghavami[4], Hamid Asadzadeh Aghdaei[5], Gloria Liliana Porras-Hurtado[6], José Luis Cardona-Deazza[6], Juan José Montoya-Martinez[6], Antonio Javier Cadavid-Velez[6], Héctor William Toro-Hidalgo[6], Alba Ruth Cobo-Alvarado[6], Ofelia del Socorro Hincapié-Rincón[6], Nadeem Kaakoush[7], Natalia Castaño-Rodríguez[1]

1. University of New South Wales, School of Biotechnology and Biological Sciences, Sydney, NSW
2. Department of Medicine, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia.
3. Division of Gastroenterology, Department of Medicine, Changi General Hospital, Singapore City, Singapore.
4. Gastroenterology and Liver Diseases Research Center, Research Institute for Gastroenterology and Liver Diseases, Shahid Beheshti University of Medical Sciences, Tehran, Iran.
5. Basic and Molecular Epidemiology of Gastrointestinal Disorders Research Center, Research Institute for Gastroenterology and Liver Diseases, Shahid Beheshti University of Medical Sciences, Tehran, Iran
6. Clínica Comfamiliar Risaralda, Pereira, Risaralda Colombia
7. University of New South Wales, School of Biomedical Sciences, Sydney, NSW

Keywords: Gastric Cancer, Autophagy, *Helicobacter pylori*

Introduction: Gastric cancer (GC) is the fifth leading cause of cancer mortality worldwide. Chronic *Helicobacter pylori*-induced inflammation is a major driver of GC. Autophagy regulates intracellular pathogen clearance and inflammation, with immunity-related GTPase M (IRGM) serving as a key regulator.

Objective: Given the association of IRGM variants with inflammatory diseases, we investigated the biological mechanisms of IRGM rs4958847 and its potential role in gastric carcinogenesis.

Methods and Results: CRISPR/Cas9-engineered IRGM rs4958847 knock-in gastric epithelial (AGS) cells were infected with a highly virulent *H. pylori* clinical isolate (GC026) to assess autophagic and inflammatory responses. In parallel, the effects of rs4958847 on the gastric mucosal transcriptome and microbiota were evaluated using RNA sequencing (15 GC cases, 31 controls) and 16S rRNA amplicon sequencing (23 GC cases, 195 controls), respectively. Upon H. pylori infection, rs4958847 knock-in AGS cells exhibited impaired autophagic responses and an enhanced TNF-α inflammatory signature. Transcriptomic analysis of rs4958847 carriers revealed enrichment of inflammatory pathways (CCL22, CCR4 etc), upregulation of oncogenic genes (FOS, FOSB), and suppression of mitochondrial function (MT-CO1, MT-CO2 etc). In GC patients, the variant was associated with persistent inflammatory signalling (TLR5, NLRP11 etc) and repression of detoxification via UGT1A8, SULT1A4. Furthermore, rs4958847 carriers displayed increased microbial alpha diversity, altered beta diversity, and enrichment of *Streptococcus* spp., taxa previously implicated in gastric carcinogenesis.

Conclusions: IRGM rs4958847 is associated with disrupted autophagy, heightened inflammatory signalling, and alterations in the gastric microbiota. These findings support rs4958847 as a potential biomarker of *H. pylori*-associated gastric carcinogenesis and suggest that IRGM-mediated host-microbial interactions may contribute to disease progression.

ORAL SESSION 1: SCIENTIFIC ABSTRACT #3

Pathogenic *Moraxella* species trigger diverging inflammasome pathways

Aritra Ghosh[1], Manjul Gautam[1], Si Ming Man[1]

1. Division of Immunology and Infectious Diseases, The John Curtin School of Medical Research, The Australian National University, Canberra, Australia.

Keywords: *Moraxella*; inflammasome; NLRP3; caspase-11; guanylate-binding proteins

Introduction: Inflammasomes detect microbial and cellular danger signals to initiate inflammatory cytokine release and pyroptotic cell death. Although individual bacterial pathogens can engage distinct inflammasomes, whether closely related species within the same bacterial genus trigger conserved or divergent inflammasome pathways remains poorly understood. *Moraxella* comprises Gram-negative species associated with respiratory, otic, ocular and systemic infections in humans and animals, yet how these bacteria are sensed by inflammasomes is largely unknown.

Objective: To systematically define inflammasome responses across pathogenic *Moraxella* species and determine whether closely related bacteria engage conserved or species-selective inflammasome pathways.

Methods and Results: We systematically screened nine pathogenic *Moraxella* species for inflammasome activation and cell death in primary macrophages and uncovered two temporally and mechanistically distinct modes of inflammasome activation. *M. ovis*, *M. bovis* and *M. bovoculi* induced rapid canonical NLRP3 inflammasome activation during acute infection through a mechanism requiring live bacteria and ion flux, but independent of guanylate-binding proteins (GBPs). In contrast, *M. canis*, *M. porci* and *M. cuniculi* evaded acute inflammasome recognition but triggered delayed activation of the non-canonical caspase-11 pathway following prolonged infection. This delayed response required GBP5 for efficient caspase-11 activation, with GBP5 and caspase-11 recruited to bacterial LOS. Despite their distinct upstream recognition mechanisms, both pathways converged on gasdermin D cleavage, pyroptosis and IL-1β and IL-18 release. In a mouse model of acute systemic infection, inflammasome signalling restricted bacterial burden while concurrently promoting systemic inflammation.

Conclusions: Our findings reveal that inflammasome recognition is not conserved across the *Moraxella* genus but is shaped by both bacterial species and infection duration. Closely related *Moraxella* species engage either rapid canonical NLRP3 or delayed GBP5-dependent caspase-11 signalling, with both pathways contributing to inflammatory responses and bacterial restriction *in vivo*. These findings uncover unexpected diversity in how the innate immune system discriminates between related bacterial pathogens and establish *Moraxella* as a model for understanding species-selective inflammasome recognition.

ORAL SESSION 1: SCIENTIFIC ABSTRACT #4

An evolutionary scavenger (receptor) hunt to explain SMuRF-less Coronary Artery Disease

Ashley Abe[1,2,3], Thomas Heuneburg[1,2], Navneet Singh[1,2], Natalie Smith[1,2], Patricia Schwarzkopf[2,3], Tung Nguyen[3], Madhuvanthi Chandrakanthan[3], Barbara Fazekas de St Groth[1,2], Gemma Figtree[3,4], Helen McGuire[1,2]

1. Translational Immunology Lab, Charles Perkins Centre, University of Sydney, Sydney, NSW, Australia
2. Faculty of Medicine and Health, School of Medical Science, University of Sydney, Sydney, NSW, Australia
3. Cardiovascular Discovery Group, Kolling Institute of Medical Research, Royal North Shore Hospital, Sydney, NSW, Australia
4. Department of Cardiology, Royal North Shore Hospital, Northern Sydney Local Health District, Sydney, NSW, Australia

Keywords: Immunology, cardiovascular, inflammation, lipids, metabolism

Introduction: Cardiovascular disease, particularly coronary artery disease (CAD) driven by atherosclerosis, remains a significant health issue despite established strategies for traditional risk factors. This highlights a crucial need to identify mechanisms of pathogenesis and novel therapies. Immune-mediated inflammation and lipid metabolism have been suggested as central drivers of CAD. Central to this model are the multifunctional scavenger receptors CD36 and LOX-1, mediating oxidised lipid uptake and foam cell formation. Interestingly, our closest animal relatives, the chimpanzee (*Pan troglodytes*), rarely develop CAD despite historic blood lipid levels considered pro-atherogenic in humans.

Objective: To identify novel biomarkers for atherosclerosis and CAD by profiling the peripheral immune systems of chimpanzees alongside humans with and without disease.

Methods and Results: We utilised the BioHEART biobank to isolate the peripheral blood mononuclear cells of humans (both with and without CAD), and chimpanzees. These were stained with both a 42-marker metal-conjugated cardiovascular antibody panel for mass cytometry by time of flight, and a multiplex fluorescence panel for imaging flow cytometry.

We identified significantly diminished expression of CD36 and LOX-1 on chimpanzee monocytes, alongside reduced lipid uptake in chimpanzees. Further, the immune profile of healthy human subjects was more similar to that of chimpanzees, compared with diseased patients.

Conclusions: These results support that heighted immune reactivity contributes to increased CAD risk, perhaps due to increased immune dysregulation via lipid internalisation. Our findings can close the significant clinical gap of defining early CAD and stratifying risk, providing improved personalised primary prevention strategies beyond traditional risk factors.

ORAL SESSION 1: SCIENTIFIC ABSTRACT #5

Guanylate-binding proteins restrain cellular proliferation during tumorigenesis

Poonam S. Jadhav[1], Aritra Ghosh[1], Si Ming Man.

1. Division of Immunology and Infectious Diseases, The John Curtin School of Medical Research, The Australian National University, Canberra, 2601, Australia.

Keywords: Guanylate-binding proteins, Colorectal cancer, Cell proliferation, mTOR signalling, Tumour suppression.

Introduction: Guanylate-binding proteins (GBPs) are interferon-inducible GTPases, with seven human and eleven mouse GBPs identified to date. These GBPs mediate host defence against microbial infections by disrupting microbial membranes and releasing concealed ligands that are subsequently sensed by innate immune sensors, driving antimicrobial responses. These antimicrobial responses of GBPs have been studied primarily in immune cells. However, GBPs are expressed across immune and non-immune cell types in humans and mice, but their roles in non-infectious diseases such as cancer remain largely unexplored.

Objective: This study aimed to examine the role of mouse GBPs in the development of intestinal cancer by utilising mice carrying a heterozygous mutation in the adenomatous polyposis coli gene (*Apc*$^{Min/+}$), a well-established model of colorectal cancer.

Methods and Results: To study the role of GBPs in intestinal tumorigenesis, we crossed GBP mice lacking one allele of GBP (*Gbp*$^{+/-}$ mice) with *Apc*$^{Min/+}$ mice, which spontaneously develop polyps throughout the entire intestinal tract. Tumour burden was analysed in 20-week-old littermate *Apc*$^{Min/+}$ mice and *Apc*$^{Min/+}$ mice lacking one of the 11 mouse GBPs. Among the 11 individual mouse GBPs tested, we found that *Apc*$^{Min/+}$ mice lacking GBP8 (*Apc*$^{Min/+}$*Gbp8*$^{-/-}$ mice) developed more tumours, exhibited substantially greater histopathological damage and had markedly more proliferating intestinal epithelial cells compared with *Apc*$^{Min/+}$ littermates. These protective effects of GBP8 were independent of major inflammatory markers, type I interferon and β-catenin signalling. Instead, we discovered that GBP8 and its human orthologue GBP4 directly interact with the mechanistic target of rapamycin (mTOR), a serine/threonine protein kinase that promotes cell growth and proliferation. Mechanistically, the coiled-coil 1 domain of GBP8 interacts with mTOR, and the HEAT repeat, FAT, and kinase domains of mTOR interact with GBP8. Binding of GBP8 to mTOR leads to reduced activation of the serine-threonine kinase AKT, resulting in decreased cell proliferation. We also found that organoids and intestinal fibroblasts derived from *Gbp8*$^{-/-}$ mice undergo uncontrolled proliferation, which could be restricted following treatment with the mTOR inhibitor Torin-1.

Conclusions: This study uncovers a previously unrecognised tumour-suppressive function of GBP8. Moreover, reduced expression of GBPs could serve as a candidate biomarker for colorectal cancer, and the GBP-mTOR axis represents a potential therapeutic target to restrain intestinal tumorigenesis.

ORAL SESSION 1: SCIENTIFIC ABSTRACT #6

Supporting longer platelet storage for transfusion: stacking maintains platelet quality over 21 days in the cold

Janhavi Mahajan[1,2], Pearl Lei[1], Christopher Roan[1], Matthew P Padula[2], Denese C Marks[1,3], Lacey Johnson[1,2,4]

1. Research and Development, Australian Red Cross Lifeblood, Alexandria, NSW, Australia.
2. School of Life Sciences, University of Technology Sydney, Ultimo, NSW, Australia.
3. Sydney Medical School, University of Sydney, Camperdown, NSW, Australia.
4. School of Science, RMIT University, Melbourne, VIC, Australia.

Keywords: transfusion, platelet, cold storage, storage configuration, stacking.

Introduction: Platelet transfusions are critical to prevent and stop bleeding. However, maintaining a constant supply of platelets is challenging, as their shelf life is limited to 7 days. Currently, platelets are stored at room temperature (20-24°C) in a single layer to facilitate gas exchange and support metabolism. Cold storage (2-6°C) is an attractive alternative, as it may allow for an extension of the shelf life up to 21 days. Cold-stored platelets have reduced metabolic activity, suggesting that greater flexibility in storage practices may be possible. Stacking platelet bags may be an effective strategy for maximising refrigerator space.

Objective: To determine whether stacking platelet bags during cold storage affects *in vitro* quality parameters.

Methods and Results: A paired study design, with three platelet bags obtained from the same donor (n = 8 individual donors), was used to assess platelet quality over 21 days of cold storage. Platelet bags were refrigerated in a single layer (control), stacked vertically (bags stored upright), or stacked horizontally (bags stored flat). Data are presented from day 21, reflecting a worst-case scenario (expiry).

Platelet aggregates (clumping), which are a major cause of product discard, were identified in 75% of vertically stacked components at expiry, while no aggregates formed in control or horizontally stacked components. Vertically stacked platelets were also significantly more activated, with greater loss of functional glycoproteins (GPIbα, GPVI) and an increase in activation markers (P-selectin, phosphatidylserine, extracellular vesicles). In contrast, horizontally stacked platelets were comparable to controls, with no significant differences in platelet activation.

Conclusions: Vertically stacked platelets were more activated and prone to aggregate formation when stored for 21 days, suggesting that this configuration alters platelet quality and is unsuitable during cold storage. In contrast, horizontal stacking may represent a practical storage option that preserves platelet quality while improving space efficiency in refrigerators.

ORAL SESSION 1: SCIENTIFIC ABSTRACT #7

Physiotherapists experiences with cognitive behavioural therapy for insomnia (CBT-I) training: mixed methods study

Michelle Wang[1], Jillian Eyles[2], Samantha Bunzli[3,4], David Klyne[5], Belinda Lawford[6], Susan M. McCurry[7], Amanda Dinh[2], Robert Schütze[8,9], Alan Nguyen[1], Daniel I. Rhon[10], Rana S. Hinman[6], Simon S. Smith[11,12], Peter Window[4,5], Stacey Grealis[13], Michelle Hall[1,2]

1. Musculoskeletal Research Hub, Charles Perkins Centre, The University of Sydney, Sydney, NSW 2050, Australia
2. Sydney Musculoskeletal Health, The Kolling Institute, The University of Sydney, Sydney, NSW 2050, Australia
3. Nathan Campus, School of Health Sciences and Social Work, Griffith University, Brisbane, QLD 4029, Australia
4. Physiotherapy Department, Royal Brisbane and Women's Hospital, Herston, QLD 4006, Australia
5. The University of Queensland, Centre for Innovation in Pain and Health Research (CIPHeR), School of Health and Rehabilitation Sciences, Brisbane, QLD 4072, Australia
6. Centre for Health, Exercise & Sports Medicine, The University of Melbourne, Melbourne, VIC 3053, Australia
7. School of Nursing, University of Washington, Seattle, WA 98195, USA
8. Curtin School of Allied Health, Curtin University, Perth, WA 6102, Australia
9. Multidisciplinary Pain Management Centre, Royal Perth Hospital, Perth, WA 6000, Australia
10. Department of Rehabilitation, School of Medicine, Uniformed Services University of Health Sciences, Bethesda, MD 20814, USA
11. ARC Centre of Excellence for Children and Families over the Life Course, Brisbane, QLD 4067, Australia
12. Child Health Research Centre, The University of Queensland, Brisbane, QLD 4101, Australia
13. UCD Centre for Arthritis Research, School of Medicine, Conway Institute of Biomolecular & Biomedical Research, University College Dublin, Belfield, Dublin 4, Ireland

Keywords: Cognitive Behavioural Therapy for Insomnia, Physiotherapists, Conservative therapies, chronic musculoskeletal pain, Implementation

Introduction: Insomnia is among the most common sleep disorders in people with chronic musculoskeletal pain, yet rarely managed in routine care. Cognitive behavioural therapy for insomnia (CBT-I) is a recommended first-line treatment. Despite strong evidence supporting CBT-I, access remains limited due to a shortage of trained providers. Physiotherapists are well positioned to deliver CBT-I and offer scalable opportunities to expand access.

Objective: Explore physiotherapists' experiences with CBT-I training to understand factors influencing implementation and upscaling.

Methods and Results: Using a mixed-methods approach, data were collected through questionnaires and semi-structured interviews from twelve physiotherapists (3-27 years' experience) who completed a CBT-I training program consisting of online workshops and a pilot case. The Educational Course Assessment Toolkit (EDUCATOOL) evaluated reaction, learning, expected outcomes and behaviours in relation to the CBT-I training, and the Determinants of Implementation Behaviour Questionnaire (DIBQ), assessed barriers and enablers to the implementation of CBT-I. Interviews explored physiotherapists' experiences of the training, guided by the theoretical framework of acceptability. Data were analysed thematically, with themes subsequently mapped to the Theoretical Domains Framework. The median EDUCATOOL overall score was 92.38 (IQR = 87.35 - 99.00) out of 100. 87% of DIBQ items were rated favourably to their respective domains (>4 on 7-point Likert scale). Four themes were generated from the interviews. Physiotherapists reported confidence in their capability to deliver CBT-I, reflecting (1) Readiness for implementation in their professional role. (2) Training delivery and design contributed positively to knowledge acquisition. (3) Experiential learning enhanced understanding and confidence. However, physiotherapists noted (4) Barriers affecting the integration of CBT-I in physiotherapy practice, including stakeholder engagement, and feasibility considerations related to billing, and delivery.

Conclusions: Physiotherapists felt confident to deliver CBT-I following a training program. For successful upscaling, future initiatives must address identified systemic and practical barriers, including stakeholder engagement and delivery feasibility.

ORAL SESSION 1: SCIENTIFIC ABSTRACT #8

Development of novel collagen-modified contact lenses for sustained ciprofloxacin delivery in bacterial keratitis

Yuan Fang[1], Clara Tran[2], Marcela Bilek[2], Gerard Sutton[3,4,5], Mark Willcox[6], Jingjing You[1,3,6]

1. Sydney Biomanufacturing Incubator, School of Medical Sciences, University of Sydney, Sydney, New South Wales, Australia
2. School of Physics and School of Biomedical Engineering, The University of Sydney, Sydney, New South Wales, Australia
3. Save Sight Institute, University of Sydney, Sydney, New South Wales, Australia
4. NSW Tissue Bank, Sydney, New South Wales, Australia
5. Vision Eye Institute, Sydney, New South Wales, Australia
6. School of Optometry and Vision Science, University of New South Wales, Sydney, New South Wales, Australia

Keywords: Bacterial keratitis; contact lens; collagen; surface modification; ocular drug delivery

Introduction: Bacterial keratitis is a potentially sight-threatening corneal infection requiring effective antimicrobial treatment. Topical antibiotics, including ciprofloxacin, are commonly used as first-line therapy; however, their therapeutic efficacy is limited by rapid tear turnover and corneal barriers, resulting in low ocular drug retention. Consequently, frequent administration of eye drops is often required, which can impose a substantial treatment burden, reduce patient adherence, and ultimately compromise treatment effectiveness.

Objective: This study aimed to develop a plasma-treated collagen-coated therapeutic lens for sustained ciprofloxacin delivery for the treatment of bacterial keratitis.

Methods and Results: Silicone hydrogel therapeutic contact lenses (Air Optix® Night & Day®) were used in this study. The lenses were loaded with ciprofloxacin (25 μg/lens), treated with an atmospheric-pressure plasma jet [1], and coated with collagen type I. Biocompatibility was evaluated using a Live/Dead viability assay after 24 h culture with human corneal epithelial cells (HCE-T), confirming good cytocompatibility. Ciprofloxacin release was assessed by cumulative release profiling, demonstrating sustained drug release. Therapeutic efficacy was evaluated using an ex vivo porcine corneal infection model with repeated PBS rinsing to simulate tear-fluid turnover. Infected corneas were treated with either the developed lenses or repeated hourly ciprofloxacin eye drops as a comparator, with both groups subjected to repeated PBS rinsing. Bacterial burden was quantified by colony-forming unit (CFU) enumeration, demonstrating antibacterial efficacy of the developed lenses comparable to that of repeated hourly eye-drop treatment.

Conclusions: The developed therapeutic lenses enabled sustained ciprofloxacin delivery while maintaining high corneal epithelial cell compatibility and antibacterial efficacy, highlighting their potential as a drug delivery platform to reduce dosing frequency and improve the management of bacterial keratitis.

ORAL SESSION 2: PRESENTATION SCHEDULE

Session Chair: Dr. Long Nguyen

Time	Speaker	Presentation Title	Affiliation
12:00 – 12:10	Alexis Minchaca	A Multimodal Human 3D Blood–Brain Barrier Organoid Platform for Assessing Barrier Integrity and Nanoparticle Penetration	*Children's Cancer Institute*
12:10 – 12:20	Michael Trpceski	Pinpointing and targeting novel drivers of pancreatic cancer progression and metastasis using TRAP-seq.	*The Garvan Institute of Medical Research*
12:20 – 12:30	Andrew Yam	Microbial Immunotherapy Reprograms the Tumour Microenvironment from Chronic to Acute Inflammation	*University of New South Wales Sydney*
12:30 – 12:40	Ellen Donohoe	Towards predictive occupational respiratory hazard assessment: Uncovering pro- fibrotic lung responses following repeated amorphous silica exposure	*Asbestos and Dust Diseases Research Institute*
12:41 – 12:45 Lightning	Thanh-Thao Nguyen	A Digital Twin Framework for Accessible Health Information Systems in Deaf Healthcare	*TwinU*
12:46 – 12:50 Lightning	Aqsa Mazhar	Uncovering Chemotherapy-Resistant Factors Through Synthetic Lethal Genome-Wide CRISPR KO Screening in High-Risk Rhabdomyosarcoma	*Children's Cancer Institute*
12:51 – 12:55 Lightning	Rokhsareh Ghalandarabadi	Development of a Multiplex qPCR Assay for Detecting HPV16 in Anal Squamous Cell Carcinoma	*University of Technology Sydney*

ORAL SESSION 2: SCIENTIFIC ABSTRACT #1

A Multimodal Human 3D Blood–Brain Barrier Organoid Platform for Assessing Barrier Integrity and Nanoparticle Penetration

Alexis Z. Minchaca[1,2,3], Aria Ahmed-Cox[1,2,3], Estrella Gonzales-Aloy[1,2,3], Nyoman Kurniawan[4,5], Amal J. Sivaram[4,5], Maria Tsoli[1,3], Taskeen Janjua[6], Ganesh R. Kokil[2,7], David S. Ziegler[1,3], Joshua A. McCarroll[1,2,3,9], Kristofer J. Thurecht[4,5], Amirali Popat[6,8], Tushar Kumeria[7], Maria Kavallaris[1,2,3,9].

1. Children's Cancer Institute at Minderoo Children's Comprehensive Cancer Centre, Sydney; NSW, Australia.
2. UNSW Australian Centre for Nanomedicine, Faculty of Engineering, UNSW Sydney; NSW, Australia.
3. School of Clinical Medicine, Faculty of Medicine & Health, UNSW Sydney; NSW, Australia.
4. Centre for Advanced Imaging, ARC Research Hub for Advanced Manufacture of Targeted Radiopharmaceuticals, The University of Queensland; St Lucia, QLD, Australia.
5. Australian Institute for Bioengineering and Nanotechnology, The University of Queensland; Brisbane, QLD, Australia.
6. School of Pharmacy and Pharmaceutical Sciences, The University of Queensland; Brisbane, QLD, Australia.
7. School of Materials Science and Engineering, UNSW Sydney; NSW, Australia.
8. Department of Functional Materials and Catalysis, Faculty of Chemistry, University of Vienna, Vienna, Austria.
9. UNSW RNA Institute, Faculty of Science, UNSW Sydney; NSW, Australia.

Keywords: BBB organoids; nanomedicine; nanoparticle penetration; barrier integrity; brain targeting.

Introduction: Predicting nanomedicine interactions with the blood–brain barrier (BBB) remains a major challenge for central nervous system drug development. While three-dimensional (3D) BBB organoids better recapitulate the cellular architecture of the BBB than conventional two-dimensional models, quantitative approaches for assessing barrier state and nanoparticle behaviour in intact organoids remain limited.

Objective: To establish a fully immortalised human 3D BBB organoid platform with complementary readouts for evaluating barrier integrity and nanoparticle penetration, and to determine whether the model can distinguish formulation-dependent differences in mesoporous silica nanoparticle (MSN) kinetics.

Methods and Results: BBB organoids were generated from immortalised human brain endothelial cells, astrocytes and GFP-labelled pericytes which self-assembled within 48 h under standard culture conditions. Organoids reproduced key BBB features, including peripheral endothelial organisation, expression of tight junction proteins and P-glycoprotein, confirmed by immunofluorescence. Barrier state and nanoparticle behaviour were assessed using three orthogonal readouts: trans-spheroidal electrical resistance (TSER), an adaptation of resistance-based barrier measurement to 3D organoids; contrast-enhanced magnetic resonance imaging (MRI); and live confocal imaging with 3D kinetic analysis. TSER resolved barrier modulation by histamine and different MSN formulations (31-60% reduction). MRI, with clinical gadolinium-based contrast agent, provided an orthogonal measure of barrier integrity and demonstrated that barrier-associated differences could be detected in intact BBB organoids as small as ~300µm in diameter. Live imaging identified distinct formulation-dependent penetration profiles. PEGylated MSNs showed the greatest overall penetration, whereas lactoferrin-functionalised particles exhibited 2-fold higher diffusivity within the organoid core. The relative ordering of nanoparticle penetration was consistent with previously reported in vivo observations.

Conclusions: Together, these complementary measurements provide a practical framework for comparing nanomedicine formulations in a human 3D BBB model which supports the preclinical evaluation and prioritisation of brain-targeted therapeutics, providing a foundation for extending resistance- and imaging-based readouts to other multicellular systems.

ORAL SESSION 2: SCIENTIFIC ABSTRACT #2

Pinpointing and targeting novel drivers of pancreatic cancer progression and metastasis using TRAP-seq.

Michael Trpceski[1,2], Alice Tran[1], Anna Howell[1], Kenny Ip[1,2], Cecilia Chambers[1,2], Jef Servaes[1], Maaike De Moes[1], Stephan Oranth[1], Jessie Zhu[1,2], Shona Ritchie[1,2], Daniel Reed[1,2], Marjan Naeini[1,2], Leonard Goldstein[1,2], Grazi Vieira[1], Robert Weatheritt[1,2], Herbert Herzog[1,2], Kendelle Murphy[1,2], Patrick Caswell[3], Johanna Ivaska[4], Paul Timpson[1,2], David Herrmann[1,2].

1. The Garvan Institute of Medical Research & The Kinghorn Cancer Centre, Sydney, NSW, Australia,
2. St Vincent's Clinical School, Faculty of Medicine, University of New South Wales Sydney, Sydney, NSW, Australia.
3. Manchester Centre for Cell-Matrix Research, The University of Manchester, Manchester, UK.
4. Turku Bioscience Centre, University of Turku and Åbo Akademi University, Turku, Finland.

Keywords: Pancreatic cancer. Metastasis. Translatome. Gene targeting. Mouse models.

Introduction: Pancreatic cancer (PC) has a dismal 5-year survival rate of only 13%, which can be attributed to its rapid metastatic spread and resistance to therapies. Consequently, more effective strategies to efficiently treat PC are required.

Objective: Here, we aimed to use innovative Translating-Ribosome-Affinity-Purification followed by RNA-sequencing (TRAP-seq) to assess the deregulated translatome of metastatic PC and identify potential novel therapeutic targets.

Methods and Results: Genetically engineered mouse models of PC are driven by a KrasG12D mutation and either loss of Trp53 (KPflC; poorly metastatic) or a gain-of-function mutation in Trp53 (Trp53R172H; KPC; highly metastatic). TRAP-seq of primary tumours from these mice identified 200 genes that were deregulated in the KPC TRAP versus KPflC TRAP translational profiles. From these, a candidate gene that was exclusively upregulated in the translating mRNAs of the KPC TRAP tumours, when compared to the KPflC TRAP tumours was selected as a novel target for further investigation. Functional models that mimic aspects of the PC metastatic cascade in vitro were used to investigate candidate gene function. Excitingly, KPC cancer cells with target genetic knockdown exhibited a reduced capacity to migrate and invade as well as grow in suspension, when compared to control. Moreover, target knockdown in vivo exhibited a significant reduction in liver metastasis in an intrasplenic transplant model. This was further corroborated by an almost complete depletion of liver metastases and significant increase in survival in mice bearing target knockdown pancreatic tumours when compared to control, which was further enhanced upon combination of target knockdown with chemotherapy.

Conclusions: Together, these findings suggest that the novel gene identified using TRAP-seq is required for the highly metastatic phenotype typically observed in the KPC model. Future investigations in patient-derived models may provide further insight into potential future co-targeting to improve response to chemotherapy and reduce metastasis in PC.

ORAL SESSION 2: SCIENTIFIC ABSTRACT #3

Microbial Immunotherapy Reprograms the Tumour Microenvironment from Chronic to Acute Inflammation

Andrew O. Yam[1,2,11], Francis Lin[1], Jacqueline Bailey[1,2], Andrew N. McCorkindale[1,2], Arnolda Jakovija[1,2], Catherine Gatt[1], Scott E Youlten[4], Claudio Counoupas[5], Matthias Gunzer[6,7], Tobias Bald[8], Trent M Woodruff[9], James A Triccas[5], Robert Brink[2], Shane Grey[1], Fabio Zanini[10], Leonard D. Goldstein[1,3], Tatyana Chtanova[1,2]

1. School of Biotechnology and Biomolecular Sciences, Faculty of Science, University of New South Wales Sydney, Kensington, NSW 2052, Australia
2. Garvan Institute of Medical Research, Sydney, NSW 2010, Australia
3. St Vincent's Clinical School, Faculty of Medicine, University of New South Wales Sydney, NSW 2052, Australia
4. Kinghorn Centre for Clinical Genomics, Garvan Institute of Medical Research, Sydney, New South Wales, Australia.
5. Discipline of Infectious Diseases and Immunology, School of Medical Sciences, Faculty of Medicine and Health, University of Sydney, Camperdown, New South Wales, Australia.
6. Institute for Experimental Immunology and Imaging, University Hospital, University Duisburg-Essen, Essen, Germany.
7. Leibniz-Institut für Analytische Wissenschaften - ISAS-e.V., Dortmund, Germany.
8. Institute of Experimental Oncology University Hospital Bonn Venusberg-Campus 1, Bonn, Germany.
9. School of Biomedical Sciences, The University of Queensland, Research Road, St Lucia, Brisbane, Queensland, Australia.
10. Prince of Wales Clinical School, Faculty of Medicine and Health, University of New South Wales Sydney, Kensington, NSW 2052, Australia
11. The Kinghorn Cancer Centre, St Vincent's Hospital Sydney, Darlinghurst NSW 2020, Australia

Keywords: Immunotherapy, Cancer, Neutrophils, Tumour microenvironment, Microbial
Career Stage: Early Career Researcher.

Introduction: Neutrophils are recruited to sites of inflammation, including tumours. The tumour microenvironment is characterised by chronic inflammation, akin to a non-healing wound, which promotes immunosuppressive neutrophils and tumour progression.

Objective: We investigated whether intratumoral microbial therapy could reprogram the tumour microenvironment into an acute inflammatory state, thereby promoting anti-tumour neutrophils that control tumour growth and protect against recurrence.

Methods and Results: Using a murine Lewis lung carcinoma model, mice were treated with intratumoral Staphylococcus aureus bioparticles. Tumour neutrophil recruitment, phenotype and function were assessed using flow cytometry, intravital microscopy, photoconversion-based cell tracking, single-cell RNA sequencing, and tumour growth analysis. Neutrophil depletion experiments determined their contribution to tumour control. In mice whose tumours were cured with Staphylococcus aureus bioparticles, we investigated whether they were protected against a tumour challenge to assess their immunity against cancer recurrence. Microbial bioparticle treatment rapidly recruited neutrophils into tumours and converted them from a pro-tumour to an anti-tumour state. Activated neutrophils displayed increased motility and production of inflammatory mediators. Single-cell RNA sequencing demonstrated extensive transcriptional reprogramming. Neutrophils entering a microbial-treated tumour rapidly developed anti-tumour features, including activation, chemokine production and increased mobility. Neutrophil depletion significantly reduced the anti-tumour efficacy of microbial therapy, confirming that neutrophils are key mediators of tumour control. Remarkably, mice that were cured by microbial bioparticles were protected from tumour rechallenge, supporting that microbial bioparticles activated the adaptive arm of immunity.

Conclusions: These findings show that neutrophil activation is dynamic and reversible, using microbial therapy. Treatment controlled tumour growth and protected against tumour recurrence. Understanding the mechanisms controlling these changes may identify new strategies to maintain anti-tumour neutrophil responses and improve cancer immunotherapy.

ORAL SESSION 2: SCIENTIFIC ABSTRACT #4

Towards predictive occupational respiratory hazard assessment: Uncovering pro-fibrotic lung responses following repeated amorphous silica exposure

Ellen Donohoe[1,2], Vivek Dharwal[1,2], Sakthi Priya Selvamani[1,2], Thomas R. O'Neil[3], Richard Zelei[1,2], Andrew N. Harman[2,3], Sharyn Gaskin[4], Deborah Yates[1,5], Anthony Linton[1,2], Elham Hosseini-Beheshti[1,2]

1. Asbestos and Dust Diseases Research Institute, Concord Hospital, Concord, NSW, Australia.
2. School of Medical Sciences, Faculty of Medicine and Health, University of Sydney, NSW, Australia
3. The Westmead Institute for Medical Research, University of Sydney, Westmead NSW, Australia.
4. School of Public Health, Adelaide Exposure Science and Health, Adelaide University, Adelaide, SA, Australia
5. School of Clinical Medicine, University of New South Wales, Sydney, NSW, Australia.

Keywords: Occupational lung disease; Ultrafine particles; Silica; Fibrosis; 3D modelling

Introduction: The silicosis crisis has highlighted limitations of reactive occupational hazard identification, with severe lung disease recognised only after substantial worker exposure and irreversible harm. Following restrictions on crystalline silica-containing engineered stone, amorphous silica-containing alternatives are marketed as safer options despite limited evidence of long-term safety. Although emerging evidence suggests silica pathogenicity cannot be explained by crystallinity alone, regulatory frameworks continue to rely on crystalline silica content as a marker of hazard. Biologically relevant approaches are needed to identify disease-causing potential of emerging materials before irreversible lung disease develops in at-risk workers.

Objectives: To investigate the effects of repeated low-dose amorphous silica exposure across respirable particle sizes, and evaluate fibrogenic potential in an advanced human lung model.

Methods & Results: Human bronchial epithelial cells were exposed to four amorphous silica (AS) particle sizes (11 nm, 500 nm, 1 µm and 3 µm) under acute (3 days) and chronic (17 days) low-dose exposure conditions. Acute exposure induced oxidative stress, endoplasmic reticulum stress and DNA damage across all particle sizes, with ultrafine (11 nm) AS eliciting the strongest response. Repeated low-dose exposure produced persistent cellular stress despite limited cytotoxicity, characterised by altered antioxidant defence, dysregulated DNA repair pathways and increased heat shock protein expression. Ultrafine AS promoted cytoskeletal remodelling and increased expression of pro-fibrotic mediators TGF-β1, MMP2 and β-catenin. Ultrafine AS was subsequently evaluated in a 3D bioprinted lung model. Repeated exposure drove a temporal shift from inflammatory signalling to fibroblast activation and extracellular matrix remodelling, recapitulating hallmarks of fibrotic lung disease.

Conclusions: Repeated exposure to ultrafine amorphous silica induced persistent stress responses and fibrogenic remodelling, challenging assumptions of low risk. This work highlights the value of biologically informed repeated-exposure models for identifying emerging respiratory hazards, supporting evidence-based regulation, and shifting occupational health practice from reactive disease management to proactive worker protection.

ORAL SESSION 2: SCIENTIFIC ABSTRACT #5

A Digital Twin Framework for Accessible Health Information Systems in Deaf Healthcare

Thanh-Thao Nguyen[1]*, Hai H Nguyen[1]*, Hoang A Truong[1], Ha S Pham[1], Veronica HB Nguyen[1], Huy X. Dao[1], Minh N. Tran[1], Nathan LH Yang[2], Huong TT Nguyen[3], Phuc T Nguyen[4], Loc V Nguyen[4], Thuy T Pham[4]

1. Computing & Health Science Research Center, TwinU, Hanoi, Vietnam.
2. Faculty of Medicine and Health, The University of Sydney, Sydney, Australia.
3. Faculty of Pharmaceutical Management and Economics, Hanoi University of Pharmacy, Hanoi, Vietnam.
4. Faculty of Engineering and IT, University of Technology Sydney, Sydney, Australia.

*Contributed equally to this work.

Keywords: Digital twin, healthcare EMR/PHR, medication education, sign language, point-of-care accessibility

Introduction: Deaf individuals face substantial barriers to healthcare communication around medication instructions, emergency procedures, and treatment adherence, as many rely on sign language rather than spoken language. Accessible health information systems can improve preventive health knowledge and treatment adherence. Inclusive approaches range from sign-language training to mobile technology, but in Vietnam, educational materials remain scarce, and automated translation for Vietnamese Sign Language (VSL) is similarly underdeveloped.

Objective: This study aimed to pilot a digital twin framework — a live-updating virtual representation of a patient's medical record — combining AI-driven VSL interpretation with community-based training to improve health information accessibility for the Deaf community in Vietnam, with a planned second phase extending the framework to Auslan in Australia.

Methods and Results: We conducted short training courses at four deaf schools across four provinces in Vietnam, introducing preventive healthcare and emergency-response knowledge in sign language — the first such training these schools had received. Post-training surveys (October 2025) at one school (students aged 11–15, literacy equivalent to grade 2) found 73% (medicine) and 90% (nutrition) of learners rated content as new. We designed a digital twin Electronic Medical Record/Personal Holistic Record (EMR/PHR) system with inclusive sign-language features, deployed by our industry partner TwinU for planned large-scale evaluation. We also piloted an AI-based VSL gloss translation module on 30 prescription images from a Hanoi point-of-care site (May 2026) and public datasets, rated by ten Deaf Vietnamese adults (aged 25–37, seven provinces) via 5-point Likert scale. Evaluators gave a mean usability rating of 3.98 ± 0.93 (median 4.0/5); 78% rated outputs4–5, only 6.3% scored 1–2.

Conclusions: Together, these pilots support the feasibility of a digital twin, sign-language-inclusive EMR/PHR framework: translated prescriptions were rated clear andclinically appropriate by Deaf users, alongside positive training outcomes among Deaf children, supporting improved point-of-care accessibility for the Deaf community in Vietnam.

ORAL SESSION 2: SCIENTIFIC ABSTRACT #6

Uncovering Chemotherapy-Resistant Factors Through Synthetic Lethal Genome-Wide CRISPR KO Screening in High-Risk Rhabdomyosarcoma

Aqsa Mazhar[1,2], Qian Wang[1,2], Satyanarayana Gadde[1], Daenikka Ravindrarajah[1], Belamy B. Cheung [1,2], Glenn M. Marshall[1,3]

1. Children's Cancer Institute, Minderoo Comprehensive Cancer Centre, UNSW Sydney, NSW, Australia.
2. School of Clinical Medicine, UNSW Sydney, Australia.
3. Kids Cancer Centre, Sydney Children's Hospital, Randwick, NSW, Australia.

Keywords: CRISPR KO Screen. Rhabdomyosarcoma. Chemoresistance. Irinotecan. Synthetic lethality

Introduction: Chemoresistance in rhabdomyosarcoma (RMS), the most common paediatric soft-tissue sarcoma, remains a major obstacle to improving survival, particularly in high-risk fusion-positive RMS, which is clinically aggressive and has a strong propensity for metastasis; survival following relapse is below 24%. Although irinotecan remains one of the most effective chemotherapeutic agents for RMS, the emergence of resistance highlights the urgent need to develop rational combination therapies to target the genetic robustness and functional redundancy of RMS cells.

Objective: To identify therapeutic vulnerabilities capable of overcoming irinotecan resistance, we performed a genome-wide clustered regularly interspaced short palindromic repeat (CRISPR) knockout screen in fusion-positive RMS cells which will lead to better prognosis and survival in high-risk RMS.

Methods and Results: The screen identified 47 candidate prognostic genes whose deletion potentially sensitizes RMS cells to irinotecan treatment. Pathway enrichment analysis revealed that the DNA damage repair pathway is the most significantly enriched pathway among candidate resistance determinants, suggesting that its disruption could induce synthetic lethality with irinotecan. Among the top hits, we validated FGFR4 and PARP1 as key modulators of resistance, and siRNA-mediated silencing of these genes sensitizes RMS cells to irinotecan. While FGFR4 was significantly enriched as a gene dependency in fusion positive RMS. In addition, pharmacological inhibition of these genes enhanced irinotecan sensitivity, with a favourable therapeutic window, supporting their potential for clinical translation. In RMS xenograft model, FGFR4 inhibition combined with irinotecan delayed tumour growth and prolonged survival.

Conclusions: Collectively, this study provides a new framework for converting genome-scale functional screening discoveries into rational combination therapies and offers a promising strategy to improve outcomes for children with high-risk, relapsed or treatment-resistant RMS.

ORAL SESSION 2: SCIENTIFIC ABSTRACT #7

Development of a Multiplex qPCR Assay for Detecting HPV16 in Anal Squamous Cell Carcinoma

Rokshsareh Ghalandarabadi[1,2], Dayna Sais[3], Thomas Hansen[4], Richard Hillman[4], Megan Barnet[4], and Nham Tran[2]

1. School of Life Sciences, University of Technology Sydney, Sydney, New South Wales, Australia
2. Non-Coding RNA Biology Lab, Discipline of Biomedical Engineering, University of Technology Sydney, Sydney, New South Wales, Australia
3. Sais Lab, Discipline of Biomedical Engineering, University of Technology Sydney, Sydney, New South Wales, Australia
4. St Vincents Hospital, Sydney, New South Wales, Australia

Keywords: HPV16. Anal squamous cell carcinoma. E6/E7 mRNA. URR DNA. Quantitative PCR.

Introduction: Anal cancer is a rare malignancy, but its incidence is increasing worldwide, especially in Australia. Anal squamous cell carcinoma (ASCC) represents 80% of anal cancer cases and is predominantly associated with persistent high-risk human papillomavirus (HPV) infection, particularly HPV16. The HPV16 oncogenes E6 and E7 contribute to carcinogenesis by disrupting tumour suppressor pathways, leading to uncontrolled cell growth. Current HPV diagnostic methods detect either viral presence or viral activity, but not both simultaneously.

Objective: This study aims to develop a multiplex qPCR assay targeting the HPV16 upstream regulatory region (URR) DNA as a marker of viral presence and E6/E7 mRNA as a marker of transcriptional activity.

Methods and Results: Using formalin-fixed paraffin-embedded (FFPE) tissue and anal swab samples, total nucleic acid (TNA) extraction was optimised on the KingFisher Duo for downstream RT-qPCR analysis. The average TNA (DNA/RNA) yield was 163.8 ng/131.1 ng for FFPE and 205 ng/164.3 ng for swab (ThinPrep) samples, respectively. Multiplex RT-qPCR assays targeting E6, E7, and URR were confirmed using HPV16-positive cell lines (SCC090 and SCC154), HPV16-negative cell line (SCC22B), and synthetic genomic blocks. The assay showed high sensitivity, detecting HPV16 URR from 50,000–1 copies and E6/E7 from 50,000–5 copies. We then successfully combined all three viral targets and two housekeeping genes (β-actin and GAPDH) to develop a five-plex RT-qPCR assay. The assay was tested on 30 ThinPrep ASCC samples to assess clinical feasibility for HPV16 detection.

Conclusions: This assay could support clinical decision-making by providing an improved assessment of HPV status for ASCC patient stratification and treatment.

ORAL SESSION 3: PRESENTATION SCHEDULE

Session Chair: Dr. Ramon Martinez Marmol

Time	Speaker	Presentation Title	Affiliation
3:00 – 3:10	Ngoc Ha Tran	Sensitive and Specific Multiplex qPCR Assays for HPV16 Detection, Oncogenic Activity, and Prognostic SNP Genotyping in Head and Neck Cancer	*University of Technology Sydney*
3:10 – 3:20	Indranil Mukherjee	PP2A Activation Restores Tamoxifen Sensitivity and Overcomes Endocrine Resistance through Ceramide-Mediated Autophagy in ER-Positive Breast Cancer	*The University of Newcastle*
3:20 – 3:30	Rotina Kapini	Synergistic Combinations of Australian Native Products for Skin Inflammation and Wound Healing	*Western Sydney University*
3:30 – 3:40	Magdalena Lerch	Insights into neurological immune-related adverse events associated with immune checkpoint inhibitor cancer therapy	*The University of Sydney*
3:41 – 3:45 Lightning	Wenyang Shi	EN1 silencing reverses the TGF-β-driven fibrotic transcriptome in renal fibroblasts: a candidate anti-fibrotic target for chronic kidney disease	*Kolling Institute*
3:46 – 3:50 Lightning	Ishani Roy	(E,E)-bisantrene suppresses MYC expression in Acute Myeloid Leukemia	*The University of Newcastle*
3:51 – 3:55 Lightning	Rizwan Khan	Harnessing Immunopeptidoimcs to Eliminate EBV Driven Cancers	*University of New South Wales*
3:56– 4:00 Lightning	Heather Murray	Moving Beyond Genomics for Precision Medicine in Acute Myeloid Leukaemia	*The University of Newcastle*

ORAL SESSION 3: SCIENTIFIC ABSTRACT #1

Sensitive and Specific Multiplex qPCR Assays for HPV16 Detection, Oncogenic Activity, and Prognostic SNP Genotyping in Head and Neck Cancer

Ngoc Ha Tran[1,2], Dayna Sais[3], Nicole Lima[4], Alison Todd[4], Michael Elliott[5,6], and Nham Tran[2]

1. School of Life Sciences. University of Technology Sydney, Sydney, Australia
2. Non-Coding RNA Biology Lab, Discipline of Biomedical Engineering, University of Technology Sydney, Sydney, Australia
3. Sais Lab, Discipline of Biomedical Engineering, University of Technology Sydney, Sydney, NSW, Australia
4. SpeeDx Pty Ltd, Eveleigh, Sydney, Australia
5. Chris O'Brien Lifehouse, Sydney, Australia
6. Sydney Medical School, University of Sydney, Sydney, Australia

Keywords: diagnostic assay, multiplex qPCR, DNAzyme, HPV16, single nucleotide polymorphism.

Introduction: HPV16 is a major risk factor for oropharyngeal cancers, which are a subset of head and neck cancers. However, HPV16 presence alone does not guarantee oncogenic activity or require immediate intervention; instead, HPV16 transcriptional status is increasingly used to monitor cancer development. HPV16 single-nucleotide polymorphisms (SNPs) have also recently been linked to poorer prognosis. This project aimed to develop two multiplex diagnostic assays using catalytic DNAzymes to detect HPV16 viability and prognostic viral SNPs.

Objective: To develop two sensitive and specific multiplex DNAzyme-based qPCR assays that distinguish transcriptionally active HPV16 from viral DNA, and to genotype four clinically relevant HPV16 SNPs.

Methods and Results: The transcriptional-activity assay used PlexZyme technology to detect HPV16 E6, E7, and a non-transcribed URR subregion. For each target, two partial catalytic DNA molecules, termed partzymes, bind adjacently on the target to form an active DNAzyme, cleaving a reporter probe to release the fluorescent signal. As recognition occurs at the partzyme level, targets can be multiplexed using different reporter colours. A second assay used PlexPrime technology to genotype four HPV16 SNPs, applying the same principle with allele-specific primers whose terminal base matches the mutant or wild-type sequence. Targets were first tested in singleplex in gBlock templates at 1000 and 50 copies. All three PlexZyme singleplex reactions (E6, E7, URR) can detect their gBlock targets, and the multiplexed assay was sensitive to 5 copies. Furthermore, the assay was specific to HPV16-positive SCC090/SCC154 cells, undetectable in HPV16-negative SCC22B and HPV18-positive HeLa cells. For the SNPs, the PlexPrime assay was able to detect the mutant allele in singleplex at 50 copies and can discriminate mutant from wild-type templates.

Conclusions: The multiplex PlexZyme assay enabled sensitive and specific detection of HPV16 E6, E7, and URR, while the PlexPrime SNP assays achieved allelic discrimination in singleplex. Together, they offer a robust platform for assessing HPV16 presence, oncogenic activity, and prognostic genotype in one workflow and are potentially adaptable across sample types, supporting a practical HPV16 diagnostic tool.

ORAL SESSION 3: SCIENTIFIC ABSTRACT #2

PP2A Activation Restores Tamoxifen Sensitivity and Overcomes Endocrine Resistance through Ceramide-Mediated Autophagy in ER-Positive Breast Cancer

Indranil Mukherjee[1,2], Madeleine Walton-Smith[1,2], Joshua Brzozowski[1,2], Kasey Miller[1,2], Abdul Mannan[1,2], Nikita Panicker[1,2], Severine Roselli[1,2], Heather Murray[1,2], Nicole Verrills[1,2]

1. School of Biomedical Sciences and Pharmacy, The University of Newcastle, Callaghan, NSW, Australia.
2. Hunter Medical Research Institute, New Lambton Heights, NSW, Australia

Keywords: ER+ Breast Cancer, MCF7 cell line, Tamoxifen, FTY720, PP2A activator, Lysosome biogenesis, Ceramide accumulation, ASAH1

Introduction: Estrogen receptor positive (ER+) breast cancer represents ~70% of all breast cancer cases. Prognosis for ER+ patients treated with endocrine therapy is favourable, however therapy resistance develops in 30-40% of patients. Protein phosphatase 2A (PP2A), a heterotrimeric serine/threonine phosphatase, is often inhibited in breast tumours, and downregulation of the PP2A-B55α subunit is associated with poor tamoxifen response.

Objective: We hypothesised that pharmacological PP2A activators could potentiate endocrine therapy in ER+ breast cancer.

Methods and Results: To test this, we have treated ER+ breast cancer cells (MCF7) with tamoxifen (a selective ER modulator) alone or combined with PP2A activators (FTY720, AAL(S) or DBK-1160). Annexin-V assays showed combination treatment significantly enhanced apoptosis compared to either drug alone. The combination was also effective in MCF7 cells resistant to single-agent tamoxifen due to long-term drug-selection or B55α knockdown. In contrast, PP2A activators did not enhance the efficacy of Fulvestrant, a selective ER-degrader, suggesting the potentiation of tamoxifen is likely ER-independent.
To elucidate the underlying mechanism, we performed proteomic analyses on MCF7 cells treated with tamoxifen, FTY720, and their combination. K-means clustering analysis revealed inhibition of lysosome biogenesis proteins in combination-treated cells. One protein in this cluster was the ceramide-converting enzyme acid ceramidase (ASAH1). Reduced ASAH1 would be expected to increase ceramide levels, which is associated with induction of autophagy and apoptosis. Consistent with this, immunoblotting demonstrated an increased LC3 II/LC3 I ratio with combination treatment, indicating enhanced autophagic flux. This, together with the reduced lysosome biogenesis, suggests apoptosis could be mediated by accumulation of autophagosomes.

Conclusions: Collectively, our findings demonstrate that PP2A activating agents can potentiate the efficacy of tamoxifen in therapy-resistant ER+ breast cancer and this may be mediated by ceramide accumulation and induction of autophagy and apoptosis.

ORAL SESSION 3: SCIENTIFIC ABSTRACT #3

Synergistic Combinations of Australian Native Products for Skin Inflammation and Wound Healing

Rotina Kapini[1], Dennis Chang[1], Gerald Münch[2], Xian Zhou[1]

1. NICM Health Research Institute, Western Sydney University, Westmead, NSW 2145, Australia
2. School of Medicine, Western Sydney University, Campbelltown, NSW 2560, Australia

Keywords: Australian native plants, inflammation, oxidative stress, synergy, combination therapy.

Introduction: Inflammation and oxidative stress are key mechanisms in underlying skin conditions like psoriasis and eczema. Plants, including Australian native plants, contain prominent phytochemicals with potent anti-inflammatory and antioxidant properties. Therefore, plants play a central role in the therapeutic potential of natural products, and a novel strategy to target complex skin disease mechanisms.

Objective: This study aimed to investigate individual and combined effects of natural products of nine herb species for skin protection and healing, focusing on their anti-inflammatory and antioxidant properties.

Methods and Results: The antioxidant potential of the individual and combined products were investigated on reactive oxygen species (ROS) assay followed by luciferase assay in MCF-7 AREc32 cells for nuclear factor erythroid 2-related factor 2 (Nrf2) activation. The anti-inflammatory activities were investigated on lipopolysaccharide (LPS)-induced RAW 264.7 murine macrophages for the inhibition of nitric oxide (NO), tumour necrosis factor (TNF)-α, and interleukin (IL)-6. Synergistic interaction was determined by the combination index model (CI < 1). Combinations(s) showing synergistic and optimal activity were further investigated on LPS-induced human dermal fibroblasts (HDF) cells for IL-6 inhibition and wound healing activity. Three of the tested products demonstrated prominent antioxidant and anti-inflammatory activities including bitter orange, mountain pepper berry and native river mint. Their three-way combination (1:1:1, *w*/*w*) showed prominent synergism (CI < 1) in reducing NO (IC_{50}: 5.82 ± 2.10 mg/mL) and IL-6 (IC_{50}: 7.55 ± 1.58 mg/mL) add IC_{50} values if there is space), along with enhanced Nrf2 activation (4.96 ± 1.35-fold). In LPS-inflamed HDF cells, the combination maintained synergistic inhibition of IL-6 levels (IC_{50}: 5.18 ± 0.74 mg/mL) and promoted wound healing response (81.22 ± 7.05%).

Conclusions: These findings highlight the therapeutic potential of Australian natural products for skin protection and repair attributed to antioxidant and anti-inflammatory activities. However, further mechanistic investigation is needed.

ORAL SESSION 3: SCIENTIFIC ABSTRACT #4

Insights into neurological immune-related adverse events associated with immune checkpoint inhibitor cancer therapy

Magdalena Lerch[1], Suzanne Culican[2], David Brown[2], Ming-Wei Lin[2], David McDonald[2], Markus Reindl[3], Sarosh Irani[4], Matthew Cook[5], Martin Stockler[6], Sudarshini Ramanathan[1,7] on behalf of the AUTOCHECK Collaboration

1. Translational Neuroimmunology Group, ANZAC Research Institute and Kids Neuroscience Centre, Sydney Medical School, Faculty of Medicine and Health, University of Sydney, Sydney, NSW, Australia.
2. NSW Health Pathology-ICPMR, Centre for Immunology and Allergy Research, Westmead Institute for Medical Research, University of Sydney, Sydney, NSW, Australia.
3. Clinical Department of Neurology, Medical University of Innsbruck, Innsbruck, Austria.
4. Mayo Autoimmune Neurology Group, Department of Neurosciences, Mayo Clinic, Jacksonville, Florida, The United States of America.
5. Division of Immunology and Infectious Diseases, John Curtin School of Medical Research, Australian National University and Canberra Clinical Genomics, Canberra, ACT, Australia. Cambridge Institute of Therapeutic Immunology and Infectious Disease, Department of Medicine, University of Cambridge, Cambridge, United Kingdom.
6. Department of Medical Oncology, Concord Cancer Centre, Concord Hospital; Department of Medical Oncology, Chris O'Brien Lifehouse, Sydney, NSW, Australia.
7. Department of Neurology, Concord Hospital, Sydney, NSW, Australia.

Keywords: Checkpoint inhibitor. neurological immune-related adverse events. autoantibodies. neurological autoimmunity. cancer therapy.

Introduction: The development of immune checkpoint inhibitors (ICI) has revolutionised cancer therapy. Patients with malignancies deemed previously incurable can now have excellent responses. However, ICIs may lead to the development of immune-related adverse events (irAEs), resulting in severe disability, interruption of cancer therapy, and even death. Neurological irAEs (nirAEs) occur in 1-10% of patients and can be fatal. These complications can be associated with antibodies (Abs) binding to neuronal or glial antigens.

Objective: We aimed to study the temporal evolution of Abs targeting specific neuronal or glial antigens in the serum of cancer patients during the course of their ICI treatment.
Methods and Results: The presence of immunoglobulin G (IgG) Abs targeting specific neuronal antigens in the serum of 169 cancer patients prior to and after ICI treatment was analysed using live cell-based assays, immunoblot and immunostaining. We found IgG Abs targeting neuronal proteins in 21/169 (12%) of our patient cohort post ICI treatment compared to 1/59 (1.7%) of controls (p=0.019). Out of the Ab positive patient samples, 48% were attributed to Abs recognising cell surface proteins and 52% had Abs binding to intracellular targets. Importantly, 58% of patients that were Ab positive after ICI therapy already had Abs targeting the same protein prior to ICI initiation. 23% of patients developed de novo Abs after their ICI treatment and 15% presented with Abs only before ICI therapy.

Conclusions: The high proportion of patients with specific neuronal IgG prior to and after ICI administration raises implications for undertaking screening of patients before receiving ICIs to develop a more personalised risk profile. Correlation with clinical manifestations, evaluation of IgM positivity, and defining functional effects of IgGs are underway. Understanding the underlying immunobiology will contribute to predicting complications from ICIs, enabling personalised treatment to improve patient survival and quality of life.

ORAL SESSION 3: SCIENTIFIC ABSTRACT #5

EN1 silencing reverses the TGF-β-driven fibrotic transcriptome in renal fibroblasts: a candidate anti-fibrotic target for chronic kidney disease

Wenyang Shi[1,2], Jessica Cao[1,2], Xin-Ming Chen[1,2], Carol A. Pollock[1,2,3]

1. Renal Research Group, Kolling Institute, St Leonards, New South Wales, Australia
2. Sydney Medical School, Faculty of Medicine and Health, The University of Sydney, Sydney, New South Wales, Australia
3. Department of Renal Medicine, Royal North Shore Hospital, Northern Sydney Local Health District, St Leonards, New South Wales, Australia

Keywords: Kidney fibrosis; Engrailed-1 (EN1); TGF-β; RNA sequencing; Myofibroblast

Introduction: Chronic kidney disease affects ~10% of adults worldwide, its progression is driven by tubulointerstitial fibrosis, in which fibrotic cytokine converts resident fibroblasts into matrix-secreting myofibroblasts. No current therapy targets this progress. Engrailed-1 (EN1) is a developmental homeodomain transcription factor re-activated downstream of SMAD3 in skin fibrosis, but its role in the kidney is still unknown.

Objective: To determine transcriptome-wide whether silencing EN1 reverses the TGF-β-induced fibrotic gene programme in renal fibroblasts, and whether that rescue is EN1-specific.

Methods and Results: NRK-49F rat renal fibroblasts were treated for 48 h with TGF-β1 (4 ng/mL) alone or with EN1-targeting siRNA (siEN1), non-targeting siRNA (siNS), or unloaded mesenchymal stem cell exosomes (n = 3/group), then profiled by bulk RNA sequencing. TGF-β1 altered 6,273 genes versus control. EN1 silencing altered 4,594 genes on the TGF-β background, whereas siNS and exosome arms altered none, confirming sequence specificity. Across the 6,273 TGF-β-responsive genes, induction and silencing fold-changes were strongly anti-correlated, with 794 genes directionally reversed versus 9 reinforced. Myofibroblast and matrix genes fell significantly after silencing, including gene such as *Acta2*, *Tagln*, *Col11a1*, *Serpine1*, *Ccn2* and *Col3a1*. Hallmark pathway such as myofibroblast activation and myogenesis were induced by TGF-β1 and inverted by EN1 silencing, and every matrisome compartment enriched by TGF-β1 was significantly de-enriched. Reversed genes were dominated by cell-cycle and nuclear-division terms, indicating release from TGF-β-imposed cytostasis.

Conclusions: EN1 acts as a broad transcriptional switch for the TGF-β fibrotic programme in renal fibroblasts: silencing it inverts, rather than merely blunts, the activated state genome-wide. EN1 is therefore a tractable anti-fibrotic target, exosome-delivered siEN1 is now being tested in murine diabetic kidney model and fibrosis model.

ORAL SESSION 3: SCIENTIFIC ABSTRACT #6

(E,E)-bisantrene suppresses MYC expression in Acute Myeloid Leukemia

Ishani Roy[1,2], Kasey Miller[1,2], Joshua Brzozowski[1,2], Dylan Kiltschewskij[1,2], Nikita Panicker[1], Ameha Woldu[1], Benjamin. J. Buckley[3], Marinella Messina[3], Chelsea Penney[3], Peter Cuthbertson[3], Sumit Sahni[3], Daniel Tillett[3], Michael J. Kelso[3], Jonathan Sillar[2,4,5], Murray Cairns[1,2], Anoop Enjeti[2,4,5], Heather C. Murray[1,2], Nicole M. Verrills[1,2]

1. School of Biomedical Sciences and Pharmacy. College of Health, Medicine and Wellbeing. University of Newcastle. Newcastle. New South Wales. Australia.
2. Precision Medicine Program. Hunter Medical Research Institute. New Lambton. New South Wales. Australia.
3. Racura Oncology Limited. Sydney. New South Wales. Australia.
4. School of Medicine and Public Health. College of Health, Medicine and Wellbeing. University of Newcastle. Newcastle. New South Wales. Australia.
5. College of Health, Medicine and Wellbeing. University of Newcastle. Newcastle. New South Wales. Australia.

Keywords: Acute myeloid leukemia. AML. Bisantrene. MYC. E2F transcription.

Introduction: Acute myeloid leukemia (AML) is genetically diverse with a high unmet clinical need for improved treatment options. Dysregulation of the transcription factor MYC has been found to play a central role in AML progression and therapeutic resistance. (E,E)-bisantrene was recently discovered to inhibit MYC transcription and downstream activity via G4 DNA stabilization.

Objective: This study aimed to evaluate (E,E)-bisantrene in preclinical models of AML and assess the role of MYC silencing in the mechanism of action.

Methods and Results: The in vitro and in vivo activity of (E,E)-bisantrene was determined in a variety of AML models (cell lines, xenograft mouse models, and ex vivo human AML mononuclear cells). Transcriptomic, proteomic and phosphoproteomic analyses were performed after treatment with (E,E)-bisantrene. Analyses of -omics data to identify enriched pathways and upstream regulators was performed. (E,E)-bisantrene demonstrated potent anti-proliferative activity across a panel of nine AML cell lines, inducing apoptosis and reducing S phase proportions. (E,E)-bisantrene was found to significantly prolong survival in cell- and patient-derived xenograft models of AML. Mechanistically, RNA-seq and proteomic analysis of MOLM13 and MV4-11 cells treated with (E,E)-bisantrene showed significant reductions in MYC and E2F transcription. Protein levels of MYC were found to be reduced in a dose- and time-dependent manner. Enrichment of TP53 and inflammation-associated signatures were observed.

Conclusions: (E,E)-bisantrene shows high activity in preclinical AML models, primarily associated with a reduction in MYC levels, together with other cell cycle regulators CDK1/2/4/5 and the E2F family. This study supports the ongoing clinical evaluation of (E,E)-bisantrene in AML and suggesting that MYC silencing is a key driver of clinical efficacy in patients.

ORAL SESSION 3: SCIENTIFIC ABSTRACT #7

Harnessing Immunopeptidoimcs to Eliminate EBV Driven Cancers

Rizwan Khan[1], Chansavath Phetsouphanh[1], Kirti Pandey[2], Anthony W. Purcell[2]

1. Kirby Institute, University of New South Wales, Kensington, NSW, Australia.
2. Biomedicine Discovery Institute, Monash University, Clayton, VIC, Australia.

Keywords: Virally Driven Cancer, EBV, Immunopeptidomics, Orbitap Astral Zoom, TimsTOFF, Orbitrap Exploris 480

Introduction: Epstein Barr virus (EBV) is a ubiquitous oncogenic virus accounting to approximately 200,000 new cancer cases annually and associated with multiple malignancies, including nasopharyngeal and gastric carcinomas and several lymphomas. EBV derived antigens provide attractive targets for T-cell based immunotherapies. However, identifying viral peptides that are naturally processed and presented by tumour cells remains challenging. Mass spectrometry based immunopeptidomics enables direct interrogation of the HLA bound peptide repertoire and provides an opportunity to discover previously unrecognised viral antigens.

Objective: To comprehensively map the EBV derived HLA class I immunopeptidome and identify previously unreported viral peptides that are reproducibly detected across independent analytical platforms.

Methods and Results: HLA-I complexes were immunoaffinity purified from naturally infection EBV positive RAJI cells and HLA bound peptides were analysed using Orbitrap Astral, Orbitrap Exploris 480 and timsTOF mass spectrometry platforms. FAIMS compensation voltages were additionally optimised for Astral based immunopeptidomics. Across five analytical runs, identified peptides displayed the characteristic HLA-I length distribution, with 9-mers predominating. Despite differences in instrumentation and sample input, 1,458 HLA-I peptides were detected across all five runs, demonstrating a reproducible core immunopeptidome. EBV specific mapping identified peptides derived from both latent and lytic viral proteins. Importantly, 12 EBV derived peptides were reproducibly detected across all five runs, with additional viral peptides identified across subsets of analyses. Several reproducible EBV peptides were previously unreported and demonstrated favourable predicted HLA-binding characteristics. Strong binding candidates included peptides derived from G75, BGLF5, BYRF1, BBRF1 and LMP2, with G75 derived peptides additionally exhibiting positive predicted immunogenicity scores.

Conclusions: Cross-platform immunopeptidomics helped in robust identification of naturally presented EBV antigens and expands the known EBV HLA-I immunopeptidome. The reproducible, previously unreported viral peptides identified here represent high confidence candidates for functional T-cell validation and future development of EBV directed immunotherapies.

ORAL SESSION 3: SCIENTIFIC ABSTRACT #8

Moving Beyond Genomics for Precision Medicine in Acute Myeloid Leukaemia

Heather C. Murray[1], Jon Sillar[2], Joshua Brzozowski[1], Kasey Miller[1], Anoop Enjeti[2], Nicole M. Verrills[1]

1. School of Biomedical Sciences and Pharmacy; College of Health, Medicine and Wellbeing, University of Newcastle, and Precision Medicine Research Program, Hunter Medical Research Institute, NSW, 2308, Australia.
2. John Hunter Hospital, Newcastle, 2298, NSW, Australia.

Keywords: Leukaemia, Precision Medicine, Proteomics, *Ex vivo* drug sensitivity, Targeted therapies

Introduction: Acute myeloid leukaemia (AML) is a heterogeneous blood cancer, with a 5-year survival rate of only 27%. Despite the emergence of new targeted therapies, survival rates remain poor in part due to the inability to predict which therapy combination is optimal for each patient. Genomic approaches alone have shown limited predictive power.
Objective: Here, we evaluated the use of multi-omic profiling to predict drug sensitivity in individual patients.

Methods and Results: *Ex vivo* drug sensitivity profiling was performed on 50 AML patient samples. Genomic profiling was conducted through routine clinical tests. Proteomic and phosphoproteomic profiling was performed using the EasyPhos method and high-resolution mass spectrometry.

Ex vivo drug sensitivity profiles were heterogeneous, however profiles correlated within drug class. High correlation within response to chemotherapy agents (daunorubicin, idarubicin, cytarabine), microtubule poisons (vincristine, vinblastine), and BCL2 family targeted therapies (venetoclax, navitoclax, S63845) was observed. Interestingly, correlations across drug classes were also identified, with response to BCL2 family targeted therapies highly correlated with kinase inhibitor sorafenib. Analysis of proteomic profiles identified that enrichment of cell cycle signatures was associated with sensitivity to sorafenib, while venetoclax sensitivity associated with enrichment of RNA metabolism pathways. The combination of sorafenib and venetoclax effected a synergistic reduction in cell survival, in venetoclax-resistant cell lines and patient samples *ex vivo*, suggesting that this approach can identify rational drug combinations.

Combining *ex vivo* drug sensitivity, genomic screening, and proteomics (using a biomarker signature previously developed in our laboratory), we next performed retrospective testing of our profiling platform to predict clinical response to venetoclax treatment, achieving an area under the receiver operator curve (AUROC) of 0.88 (n=10). This improved on the performance of mutation-based prediction of response only (AUROC 0.55).

Conclusions: Individualised multi-omic profiling is a powerful approach for functional characterisation and rational therapy selection in AML.

ALAN SKYRRING MEMORIAL AWARD SESSION

In 1960, after spending 2 years in the United States, Dr Alan Skyring and Dr Barry Firkin arrived back in Australia to take up the positions of Director of Gastroenterology and Director of the Clinical Research Unit, respectively, at the Royal Prince Alfred Hospital in Camperdown. At the time, both felt existing societies in Australia were inadequate for the growing needs of health and medical researchers. Thus, influenced by their time in America, they followed the example of the American Federation for Clinical Research (which showcased multidisciplinary research and fostered young investigators) and formed the Australian Society for Medical Research (ASMR) in 1961. Barry Firkin became the first President of the ASMR, while Alan Skyring became the first Secretary/Treasurer. In its first year, Dr Skyring was tasked with raising money, organising the first Annual General Meeting and publishing the Journal titled "Medical Research". The following year, in 1962, Skyring succeeded Firkin as the second President of the ASMR.

Dr Alan Skyring
Co-founder of the ASMR

The Alan Skyring Memorial Award was proposed by the members of the ASMR New South Wales Committee in December 2018 after Dr Skyring passed away at the age of 92. The award is in recognition of Dr Skyring's significant contributions to health and medicine and the ASMR. The Award will be presented annually to a mid-career investigator for the most outstanding presentation at the ASMR NSW Annual Scientific Meeting.

Session Chair: Dr. Dan Enosi Tuipulotu

Time	Speaker	Presentation Title	Affiliation
4:15 – 4:25pm	Alessandro Castorina	Microglia-specific loss of MERTK expression in the normal-appearing white matter of primary progressive multiple sclerosis	*University of Technology Sydney*
4:30 – 4:40pm	Yen Chin Koay	Restoring the cardiac NAD^+–HMGCS2 ketogenesis axis attenuates cardiac lipotoxicity in cardiometabolic heart failure	*The University of Sydney*
4:45 – 4:55pm	Jason Girkin	First in vivo evidence that N-myristoyltransferase inhibition reduces rhinovirus infection and airway inflammation	*The University of Newcastle*

ALAN SKYRRING MEMORIAL AWARD SESSION: ABSTRACT #1

Microglia-specific loss of MERTK expression in the normal-appearing white matter of primary progressive multiple sclerosis

Judith Brinkman[1], Margo I. Jansen[1], **Alessandro Castorina**[1]

1. Laboratory of Cellular and Molecular Neuroscience, School of Life Sciences, Faculty of Science, University of Technology Sydney, Sydney, NSW, Australia

Keywords: Multiple sclerosis. Primary progressive MS. MERTK. Microglia. Astrocytes. Normal-appearing white matter. Myelin clearance.

Introduction: Multiple sclerosis (MS) is characterised by demyelination, neuroinflammation and progressive neurodegeneration. Although white matter surrounding established lesions appears histologically normal, accumulating evidence indicates that normal-appearing white matter (NAWM) undergoes molecular alterations that may precede overt lesion formation. Identifying these changes may reveal mechanisms driving progression and novel therapeutic targets, particularly in primary progressive MS (PPMS), for which effective treatments remain limited.

Objective: This study aimed to identify molecular alterations in the NAWM of progressive MS and determine whether these changes are associated with specific glial cell populations.

Methods and Results: Differentially expressed genes were identified by integrating three independent MS transcriptomic datasets, revealing 242 overlapping genes. Four genes associated with the TAM receptor pathway, MERTK, AXL, TYRO3 and TYROBP, were selected for validation by RT-qPCR in post-mortem NAWM from non-MS controls (n=5), PPMS (n=4) and secondary progressive MS (SPMS; n=6). MERTK protein expression was further examined by co-immunofluorescence in astrocytes and microglia (n=3/group). AXL expression was increased in both PPMS and SPMS, MERTK in SPMS, and TYROBP and TYRO3 in PPMS. Cell-specific analysis revealed increased proportions of MERTK-positive astrocytes in both progressive MS subtypes, whereas MERTK-positive microglia were significantly reduced specifically in PPMS. MERTK fluorescence intensity was unchanged, indicating that the difference reflected the proportion of MERTK-expressing microglia rather than reduced expression per positive cell. Inflammatory stimulation of primary murine glia did not induce detectable MERTK expression.

Conclusions: Progressive MS is associated with molecular abnormalities in white matter beyond established lesions. The selective reduction of MERTK-positive microglia in PPMS identifies a cell-specific alteration that may impair microglial clearance of myelin debris and remyelination. These findings support further investigation of microglial MERTK signalling as a potential therapeutic target in PPMS.

ALAN SKYRRING MEMORIAL AWARD SESSION: ABSTRACT #2

Restoring the cardiac NAD⁺–HMGCS2 ketogenesis axis attenuates cardiac lipotoxicity in cardiometabolic heart failure

Yen Chin Koay[1,2], Bailey McIntosh[2,3], John F. O'Sullivan[1,2,4]

1. School of Medical Sciences, Faculty of Medicine and Health, The University of Sydney, Sydney, New South Wales, Australia.
2. Charles Perkins Centre, The University of Sydney, Sydney, New South Wales, Australia.
3. School of Life and Environmental Sciences, Faculty of Science, The University of Sydney, Sydney, New South Wales, Australia.
4. Department of Cardiology, Royal Prince Alfred Hospital, Camperdown, New South Wales, Australia.

Keywords: cardiac metabolism; cardiometabolic HFpEF; ketogenesis; NAD+ metabolism; lipotoxicity.

Introduction: Heart failure with preserved ejection fraction (HFpEF) is now the most common form of heart failure. Its predominant subtype, cardiometabolic HFpEF (CM-HFpEF), is driven by the convergence of obesity, insulin resistance and hypertension, yet no targeted therapy exists. Healthy hearts flexibly switch between fuel sources, but this metabolic flexibility is lost in CM-HFpEF. Suppressed glucose oxidation and excessive fatty acid supply promote lipid accumulation and lipotoxicity. The canonical ketogenic enzyme 3-hydroxy-3-methylglutaryl-coenzyme A synthase 2 (HMGCS2) can divert this surplus acetyl-CoA away from this lipotoxic fate, but its role in the heart is unknown.

Objective: To determine how intrinsic cardiac ketogenesis via HMGCS2 regulates substrate metabolism and lipotoxicity in HFpEF, and whether restoring myocardial NAD⁺ reactivates HMGCS2 to improve cardiac function.

Methods and Results: Using human myocardium with transcardiac ketone sampling, a high-fat diet plus L-NAME murine CM-HFpEF model, ex vivo human heart slices and Langendorff 13C-isotope tracing, cardiomyocyte-specific HMGCS2-knockout mice and multi-omics, we demonstrated that the human heart has intrinsic ketogenic capacity through HMGCS2. CM-HFpEF myocardium showed suppressed glycolysis, PDK4-driven metabolic inflexibility, lipid accumulation and increased acetoacetate uptake. In CM-HFpEF, reduced NAMPT protein abundance and myocardial NAD⁺ depletion impaired SIRT3-mediated deacetylation, leaving HMGCS2 hyperacetylated and less active despite increased abundance. Supplemented NR was directly taken up by the heart and restored myocardial NAD⁺. This promoted HMGCS2 deacetylation and reactivation, increased fatty acid and ketone oxidation, while reducing cardiac lipid accumulation and lipotoxicity. NR also improved cardiac relaxation, blood pressure and exercise capacity. These benefits were lost in cardiomyocyte-specific HMGCS2 knockout mice, suggesting that NR acts through HMGCS2 within cardiomyocytes.

Conclusions: Intrinsic cardiac ketogenesis through HMGCS2 provides substrate flexibility that is disabled by NAD+ depletion in CM-HFpEF. Restoring the cardiac NAD⁺–HMGCS2 axis rescues diastolic function by reactivating HMGCS2-dependent lipid metabolism, identifying HMGCS2 as a therapeutic target in cardiometabolic HFpEF.

ALAN SKYRRING MEMORIAL AWARD SESSION: ABSTRACT #3

First in vivo evidence that N-myristoyltransferase inhibition reduces rhinovirus infection and airway inflammation

Jason Girkin[1,2], Nathan E. Bryant1,2, Su-Ling Loo[1,2], Camille Esneau[1,2], Cassandra Jarvis[1,2], Nathan Bartlett[1,2]

1. Viral Immunology and Respiratory Disease Group, School of Biomedical Sciences and Pharmacy, College of Health Medicine and Wellbeing, University of Newcastle, Newcastle, NSW, Australia.
2. Infection Research Program, Hunter Medical Research Institute, New Lambton Heights, NSW, Australia.

Keywords: Rhinovirus; N-myristoyltransferase; antiviral; host-directed therapy; inflammation.

Introduction: Rhinovirus (RV) causes a spectrum of diseases from the common cold to bronchiolitis and pneumonia in high-risk populations, and RV is the most frequent cause of exacerbations of chronic respiratory diseases. Due to the genetic diversity of RV (>170 types), we focused on host factors required for RV replication as therapeutic targets. N-myristoyltransferase (NMT) is an essential host enzyme required for maturation of the RV capsid precursor (VP0) and functional capsid assembly. Although NMT inhibition has previously demonstrated antiviral activity in vitro, its efficacy against rhinovirus infection and virus-induced airway inflammation has not previously been evaluated in vivo.

Objective: Test the hypothesis that the NMT inhibitor IMP1320 suppresses rhinovirus replication and virus-induced airway inflammation in vivo.

Methods and Results: Mice were infected intranasally with RV-A1 and treated twice daily with the NMT inhibitor IMP1320 (10 or 25 mg/kg) or vehicle control by intraperitoneal injection. Viral load was quantified in respiratory tissues using $TCID_{50}$ and qPCR. Airway inflammation was assessed in bronchoalveolar lavage (BAL) by differential cell count and cytokine analysis (LEGENDplex bead array). 10 mg/kg IMP1320 treatment significantly reduced infectious viral load in the upper respiratory tract at 24 hours post-infection (hpi). This was accompanied by a reduction in BAL neutrophils and lymphocytes at 48 hpi, together with decreased concentrations of the inflammatory mediators MCP-1 and CXCL10. Trends towards reduced lung viral load were also observed. Collectively, these findings demonstrate that NMT inhibition reduces RV infection and attenuates virus-induced airway inflammation.

Conclusions: This study provides the first in vivo evidence that NMT inhibition suppresses rhinovirus infection and associated inflammation. By targeting a host factor essential for viral replication, NMT inhibitors represent a promising host-directed antiviral strategy with potential to reduce both viral burden and inflammatory disease exacerbations. These findings support further development of NMT inhibitors for respiratory antiviral therapies.

POSTER SESSION:

Poster #	Poster Title	Author
1	Co-designing physical activity resources for paediatric cancer patients and survivors	Katia Bjorkmann
2	Diabetic Glomerulopathy: A Proteomic Approach to Molecular Pathway Abnormalities in an Animal Model of Type 1 Diabetes	Navneen Kumar Parthiban
3	Investigating the Movement of HIV-1 Subtypes Across South-East Asia	Moutia-Mouna Chaouk
4	AI in Healthcare for Resource Limited Settings: An Exploration and Ethical Evaluation	Rune Chi Zhao
5	Sign-Language-Adapted Attention Training with EEG-Based Outcome Assessment for Deaf Children: A Pilot Closed-Loop Study	Minh Tran
6	The Role of Extracellular Matrix Proteins in Non-Small Cell Lung Cancer Progression	James Seccombe
7	Understanding the evolution of cross-reactive antibodies driving hepatitis C virus – associated cryoglobulinaemic vasculitis (HCV-MC)	Sophie Nides
8	Sleep and Human Movement: Mapping Their Potential Relationship in Osteoarthritis and Low Back Pain	Zhenbo Ma
9	Ilex paraguariensis-derived compound combinations exert synergistic anti-neuroinflammatory properties in LPS-stimulated mono- and tri-culture models	Isabella Pregardier Klann
10	Harnessing T Cell Immunity Through mRNA Vaccines	Sahara Waide
11	Investigating Epstein-Barr Virus-Associated Transcriptomic Signatures Across Gastric Carcinogenesis and Diverse Populations	Hossein Barzegar
12	Detection of High-Risk Human Papillomavirus in Primary Tumours and Matched Lymph Nodes of Oesophageal Adenocarcinoma	Hossein Barzegar
13	Whole transcriptome sequencing of adipose tissue eosinophils from obese mice identifies new potential anti-obesity therapeutic targets	Tsz Ho Li
14	Identifying retrotransposons that regulate human T-cell immune responses	Billi Bragonzi
15	Evaluating the Quality and User Experience of Mobile Health Applications for Medication Adherence: Bridging Validated App Assessment with Real-World User Perspectives	Mohamed Afifi
16	Oncomir Mir-21 As A Master Regulator Of Non-coding Rnas In Oral Squamous Cell Carcinoma	Sarah Stapleton
17	A Model for Real-Time Detection of HCV Transmission in NSW Prisons	Bethany Horsburgh
18	Deciphering Macrophage-Epithelial-Fibroblast Crosstalk in Silicosis: A Multicellular Preclinical Modelling Approach	Sakthi Priya Selvamani
19	Understanding the isomiR landscape of the parasitic flatworm Fasciola hepatica: Master regulators of developmental transitions and host immune evasion	Dayna Sais
20	Urine-derived kidney tubuloids as a patient-specific model of chronic kidney disease	Long Nguyen
21	Development of 24-Hour Movement Guidelines for Lower-Extremity Osteoarthritis: Protocol for an OARSI Initiative	Boliang Wang
22	Mediation analysis of psychological and behavioural factors after adding aerobic physical activity to resistance exercise in hip osteoarthritis	Boliang Wang

POSTER SESSION:

Poster #	Poster Title	Author
23	Identification of brain long non-coding RNAs involved in memory	Saba Altaf
24	To Enrich or Not to Enrich? Direct Sequencing of CTC-Derived Nucleic Acids from Blood in Childhood Cancer	Hafsah Atif
25	The Use of Liquid Biopsy for High-Risk Stratification of HPV16-Positive Oropharyngeal Cancer Patients.	Nicole Burrows
26	The endogenous retrovirus MMERVK9C maintains immune self-tolerance	Iris Chang
27	Modulation of YAP1 signalling by Compound A in Renal Tubular Injury and Diabetic Kidney Disease	Daisy Chen
28	Measuring circulating cell-free mitochondrial DNA and cell-free DNA may detect biomarkers in gestational diabetes mellitus	Ya-Wen Chiang
29	Impact of tumour model complexity on ovarian cancer growth kinetics and drug response across 2D, 3D spheroid, and tumour-on-chip models	Layal Dani
30	Size and concentration of extracellular vesicles in transfusable blood components are influenced by manufacture and storage conditions	Bailey Deagon
31	Surveillance and Characterisation of Arboviruses in the Hunter Region: Establishing Molecular Surveillance Tools for Detection in Mosquitoes	Carissa Enriquez
32	Citrus aurantium L. (Bitter Orange) Extracts and its Bioactive Compounds Reduces Inflammatory Responses in Macrophage and Human Dermal Fibroblast Models	Belinda Huor
33	N-myristoyltransferase inhibition as a host-directed antiviral strategy against rhinovirus infection in a human airway epithelial model	Cassandra Jarvis
34	Viral and oxidative injury induce epithelial IL-25: a potential driver of lung fibroblast activation	Jingyue Kang
35	Mapping circulating norovirus genotypes in clinical acute gastroenteritis cases 2025	Dillon Lai
36	The RNA Helicase Rig-I in Biomolecular Condensates	Yola Li
37	Small Interfering RNA for the treatment of rhinovirus infections	Ethan McDaid
38	Enhancing the efficacy of and overcoming resistance to Kras Inhibitors in Pancreatic Cancer	Niamh Mills
39	Investigating loss of the tumour suppressor gene PTEN as a potential genetic driver of malignant transformation in endometriosis	Anaida Nirula
40	The Function of Retrotransposon Lx9c11 in the Immune System	Finn O'Loan
41	The Vaginal Microbiome, Cervical Length Shortening and Preterm birth risk: A Systematic Review	Jessica Sydney Ramos
42	PocketPCR: Portable PCR with Integrated Fluorescence Detection for viral Screening	Nima Reji

POSTER SESSION:

Poster #	Poster Title	Author
43	Medication-related problems and pharmacist recommendations for cardiometabolic medicines in Arabic-speaking patients: A retrospective analysis of home medicines reviews in Australia	Rawan Sawalha
44	Single-cell and spatial transcriptomic profiling identifies Cancer-associated fibroblast heterogeneity and *MYCN*-associated stromal remodelling in neuroblastoma	Mekonnen Sisay Shiferaw
45	Clinical outcomes of children and adolescents with type 1 diabetes mellitus in Ethiopia: evidence on glycaemic achievement and diabetic ketoacidosis	Chalie Tiruneh
46	Therapeutic Potential of Adipose-Derived Mesenchymal Stem Cells in Cisplatin-Induced Acute Kidney Injury	Thi Kim Chi Tran
47	Co-targeting thrombospondin-1 (THBS-1) to improve KRAS inhibitor efficacy in pancreatic cancer	Victoria Tyma
48	Type Title Changes in sleep and clinical outcomes in hip osteoarthritis: A longitudinal secondary analysis of a randomised controlled trial	Hanzhi Zhang
49	Novel β-lactam Combination Therapy with Iminosugar AMP-DNJ to Treat Methicillin Resistant *Staphylococcus aureus*	Wentao Xu
50	cGAS Engages Nutrient-Sensing Enzymes To Promote Tumorigenesis	Shreya Mahajan
51	Combined DNA-PK and BCL-2 Inhibition Overcomes Resistance to venetoclax in AML	Madison Chambers

POSTER SESSION: SCIENTIFIC ABSTRACT #1

Co-designing physical activity resources for paediatric cancer patients and survivors

Katia Bjorkmann[1,2], Christina Signorelli[1,2], Callum Joyner[1], Eleanor Cook[2], Adam Nelson[2], Georgia Henshaw[2], Karen Johnston[2], Collene Wright[3], & Lauren Ha[1,2]

1. Behavioural Sciences Unit, Discipline of Paediatrics & Child Health, School of Clinical Medicine, UNSW Sydney, Australia
2. Minderoo Children's Comprehensive Cancer Centre, Sydney Children's Hospital, Randwick, Australia
3. Little Big Steps Organisation

Keywords: paediatric oncology; physical activity; co-design; COM-B model; behaviour change

Introduction: Physical activity engagement supports physical and psychosocial outcomes in children with cancer. However, treatment-related side effects and a lack of cancer-specific guidance contribute to poor participation, with many children not returning to pre-diagnosis activity levels post-treatment. There remains a need for accessible, age-appropriate resources tailored to the physical activity needs of children undergoing cancer treatment.

Objective: We aimed to identify children and young peoples' information needs, barriers, motivators, and preferences regarding physical activity during treatment to inform resource development.

Methods and Results: We used a mixed-methods co-design approach, combining i) cross-sectional surveys in children/young people with cancer (aged 8-25) and parents, assessing physical activity behaviours, barriers, motivators, information needs, and preferences, and ii) online workshops with physiotherapists focused on resource design across age groups (0–5, 6–12 &13–17 years). We conducted inductive-deductive content analysis, and mapped findings to the Capability, Opportunity, Motivation–Behaviour (COM-B) model to identify behavioural determinants that underscore the resource design.

To date, 13 children/young people (mean age=15, SD=4.8) and 15 parents have completed surveys, and four paediatric physiotherapists have participated in six workshops. Participants described capability barriers including limited knowledge of oncology-specific physical activity recommendations, and safety considerations which appeared to reduce their confidence to be active. Treatment-related side effects created significant opportunity barriers, with 91% of children/young people reporting fatigue. Motivational drivers included social connection, confidence, returning to sport or pre-diagnosis fitness, and, for younger children, opportunities for play and developmental progression. Across all domains, participants highlighted the need for practical guidance that supports participation while accommodating fluctuating health and treatment-related limitations.

Conclusions: These findings support the co-design of tailored, age-appropriate resources that translate medical recommendations into practical physical activity guidance. The next phase of research will involve workshops with children and parents to further incorporate lived experience into the resource design.

POSTER SESSION: SCIENTIFIC ABSTRACT #2

Diabetic Glomerulopathy: A Proteomic Approach to Molecular Pathway Abnormalities in an Animal Model of Type 1 Diabetes

Muralikrishna Gangadharan Komala[1,2,3*], Naveen Kumar Parthiban[1,2*], Long The Nguyen[1,2], Amgad Zaky[1,2], Umut Rende[2,4], Carol A Pollock[1,2], Sonia Saad[1,2]

1. Department of Medicine, University of Sydney, Sydney, NSW, Australia
2. Renal Research Laboratory, Kolling Institute, Royal North Shore Hospital, St Leonards, NSW, Australia
3. Nepean Blue Mountains Local Health District, Penrith, NSW, Australia
4. ARC Centre of Excellence for Nanoscale BioPhotonics, Graduate School of Biomedical Engineering, Faculty of Engineering, University of New South Wales, Sydney, NSW, Australia

*MuraliKrishna Gangadharan Komala and Naveen Kumar Parthiban are Joint first authors.

Keywords: diabetic nephropathy; glomerular proteomics; laser-capture microdissection; mitochondrial bioenergetics; extracellular matrix remodelling

Introduction: Diabetic kidney disease (DKD) is the most common cause of kidney failure worldwide, affecting 20–40% of people with diabetes. While tubulointerstitial fibrosis is recognised as a driver of progression, the glomerular pathways activated by direct glucotoxicity remain poorly defined, and whole-kidney profiling dilutes glomerular signals with abundant tubular protein. Effective therapies remain particularly limited in type 1 diabetes (T1D).

Objective: To define a glomerulus-resolved proteomic signature of diabetic nephropathy in a mouse model of T1D. We hypothesised that diabetic glomeruli would demonstrate regulation of peptides and pathways associated with mitochondrial dysfunction and extracellular matrix (ECM) deposition.

Methods and Results: Diabetes was induced in male eNOS-knockout mice using low-dose streptozotocin. Kidneys were harvested after 24 weeks of diabetes and formalin-fixed and paraffin-embedded. Glomeruli from four diabetic and four control mice were isolated by laser-capture microdissection and profiled using data-independent acquisition mass spectrometry. Differential abundance was assessed under Benjamini–Hochberg FDR control ($q < 0.05$), followed by GO/KEGG enrichment and high-confidence STRING network analysis (combined score ≥ 0.7). Diabetic mice showed elevated blood glucose ($P<0.01$), mesangial expansion and nodular and global glomerulosclerosis (glomerulosclerosis index, $P<0.0001$). Of 3,318 quantified protein groups, 257 were significantly regulated (219 upregulated, 38 downregulated), of which 64 showed $|\log_2 FC| \geq 1$. Enrichment was dominated by mitochondrial oxidative phosphorylation and electron transport (NDUF/COX/UQCR/ATP synthase subunits), with additional renal transport and ECM/basement membrane terms, including decorin and IGFBP7. STRING analysis yielded a connected network of 123 nodes and 687 edges centred on mitochondrial bioenergetic, ECM and transport proteins, alongside RNA-processing hubs (Hnrnpa3) and loss of the complement regulator CD55/DAF.

Conclusions: Compartment-resolved glomerular proteomics identifies coordinated mitochondrial bioenergetic remodelling and extracellular matrix reorganisation as central features of diabetic glomerulopathy in T1D. This metabolic–remodelling axis, with downstream complement and post-transcriptional regulation, offers candidate nodes for mechanistic interrogation and therapeutic development in DKD.

POSTER SESSION: SCIENTIFIC ABSTRACT #3

Investigating the Movement of HIV-1 Subtypes Across South-East Asia

Chaouk, MM[1,2], Featherstone, L A*[1], van Bockel, D*[1], Horsburgh, B A*[1]

1. The Kirby Institute, Faculty of Health and Medicine, UNSW, Sydney, NSW, Australia. 2. School of Biomedical Science, Faculty of Health and Medicine, UNSW, Sydney, NSW, Australia

Keywords: HIV-1, CRF01_AE, South-East Asia, Phylogenetic Analysis, Phylogeographic Modelling

Introduction: Over the past decade, the Australian HIV-1 epidemic has shifted towards an increasing prevalence of non-subtype B infections, particularly CRF01_AE and subtype C. These predominantly circulate in the region of South-East Asia and India, respectively. Limited publicly available sequences from countries in the region, outside of major transmission hubs (e.g.- Thailand), hindered molecular surveillance and characterisation of transmission dynamics.

Objective: To address this, we sequenced and analysed HIV-1 samples from five geographic location across the region, to understand how regional HIV transmission may be influencing the Australian HIV-epidemic.

Methods and Results: RNA was extracted from plasma samples collected through ENCORE1 and SECOND-LINE studies, reverse transcribed, and amplified using a tiling PCR approach. Oxford Nanopore sequencing generated sequences which were mapped, aligned, and submitted onto Stanford HIV Drug Resistance Database for subtype assignment and drug resistance analysis. Generated sequences were combined with publicly available GenBank sequences for phylogenetic analysis using IQ-TREE 3. Amplification and sequencing were attempted on 334 plasma samples, of which 85% generated >95% coverage over RT. CRF01_AE predominated in Thailand, Singapore, and Malaysia, while subtype C was confined to India and Hong Kong. Preliminary phylogenetic analysis suggested intermixing between Thai, Singaporean, and Malaysian sequences, whereas Indian and Hong Kong sequences formed a distinct clade for subtype C.

Conclusions: These findings provide new molecular epidemiological data from underrepresented geographical regions and suggest distinct transmission patters between the CRF01_AE in South-East Asia and subtype C in India. Ongoing drug resistance and phylogeographic analyses incorporating Australian sequences will further resolve regional transmission pathways and introductions into Australia.

POSTER SESSION: SCIENTIFIC ABSTRACT #4

AI in Healthcare for Resource Limited Settings: An Exploration and Ethical Evaluation

Rune Chi Zhao[1], Xiuyuan Yuan[1]

1. Australian National University, Canberra, ACT, Australia

Keywords: Artificial Intelligence; Healthcare; Resource-Limited Settings; Ethics; Health Equity

Introduction: Artificial intelligence (AI) has significant promise to improve healthcare in resource-limited settings (RLS). By assisting with diagnostic decisions, treatment planning, disease prediction and resource allocation, AI may help bridge the gap in healthcare quality between resource-limited and resource-rich settings. However, shortages of healthcare professionals, fragile infrastructure, and limited regulatory oversight can also leave RLS particularly vulnerable to AI's potential ethical issues. Despite rising expectations, the ethical considerations of AI for healthcare in these settings remain comparatively underexplored.

Objective: To examine how AI is currently being applied in resource-limited healthcare and critically evaluate the ethical challenges that arise when these technologies are deployed in settings with limited human, financial and infrastructural resources.

Methods and Results: We conducted a structured narrative review of 42 sources identified primarily through PubMed and Google Scholar, prioritising literature from 2010 onwards and retaining relevant foundational studies. Studies examining healthcare AI applications, ethical implications, or barriers to adoption in RLS were included, while high-resource studies without transferable relevance were excluded. Evidence was synthesised across application and ethical themes. Four major application areas were identified: decision support, predictive analytics, telemedicine and digital health, and resource management. Across these, AI showed potential to alleviate workforce shortages, improve access to care, and optimise scarce resources. However, our analysis identified five major ethical concerns: bias and fairness, non-maleficence, privacy and security, autonomy, and transparency, with risks amplified by limited local data, fragile infrastructure and weaker regulatory oversight. Further dilemmas arise around human oversight and whether investment in AI represents the best allocation of resources.

Conclusions: AI could help reduce healthcare disparities, but deployment alone does not guarantee equitable outcomes. Instead, responsible implementations require context-specific regulation, culturally sensitive design, meaningful local participation, capacity building, and ultimately, careful consideration of whether these applications truly benefit those it seeks to serve.

POSTER SESSION: SCIENTIFIC ABSTRACT #5

Sign-Language-Adapted Attention Training with EEG-Based Outcome Assessment for Deaf Children: A Pilot Closed-Loop Study

Minh N. Tran[1], Hai H. Nguyen[1], Ha S. Pham[1], Veronica B.H. Nguyen[1], Huy X. Dao[1], Thuy T. Pham[2]

1. Computing & Health Science Research Center, TwinU, Hanoi, Vietnam.
2. Faculty of Engineering and IT, University of Technology Sydney, Sydney, Australia.

Keywords: Attention Training, EEG Biomarkers, Deaf Children, Sign Language, Closed-Loop Intervention, N-Back Working Memory.

Introduction: Deaf children's communication barriers make attentional engagement difficult for teachers to gauge and can mask early signs of psychological difficulty needing referral to a mental health consultant. Attention is a promising target because it is both behaviourally observable and disrupted early in emerging distress; low-cost electroencephalography (EEG) offers an objective way to index it alongside structured training.

Objective: We developed SATT, a sign-language-adapted version of the Attention Training Technique (ATT; Wells, 1990) for deaf children, replacing ATT's auditory attention exercises with visual, sign-based analogues delivered by native Deaf signers, embedded in a closed-loop framework: daily training practiced at home via the TwinU mobile health app, which includes a built-in sign library, and attentional change indexed through EEG-based n-back assessment.

Methods and Results: Prior EEG work in 53 healthy adults showed n-back reaction time, but not accuracy, increased under high load, supporting EEG as a feasible attentional index. Building on this, we now propose a 4-week feasibility pilot of SATT in 20 deaf children aged [6–18 years] in Vietnam. Children will practice SATT for ~12 minutes daily, logged via TwinU's tappable sign library. EEG will be recorded during n-back assessment pre- and post-pilot, alongside the Test of Attentional Performance and teacher-report measures. Feasibility of SATT will be assessed via acceptability criteria, including session adherence (≥70% of prescribed daily sessions completed), attrition, and caregiver-reported satisfaction. Ethics approval will be sought from the Australia Vietnam Strategic Technologies Research Center, Hanoi, Vietnam, prior to recruitment, with consent and study materials delivered in sign language.

Discussions: SATT resembles focused-attention meditation and mindfulness-based cognitive therapy. By indexing attention objectively, this framework may help teachers flag learners who warrant closer monitoring or referral to a mental health consultant. Pending feasibility, we plan to extend this pilot into a 12-week trial incorporating real-time EEG neurofeedback, toward accessible attention monitoring in Deaf education.

POSTER SESSION: SCIENTIFIC ABSTRACT #6

The Role of Extracellular Matrix Proteins in Non-Small Cell Lung Cancer Progression

James Seccombe[1], Su Waddy Aung[1], Joveline Sheto[1], Gang Liu[1]

1. School of Life Science, Faculty of Science, University of Technology Sydney, Ultimo, NSW, Australia

Keywords: Extracellular Matrix. Multimerin-1. Lung Cancer. Bioinformatics.

Introduction: Lung cancer is an exceedingly high impact disease, being the leading cause of cancer-related death worldwide. Non-Small Cell Lung Cancer (NSCLC), which accounts for approximately 85% of all lung cancer cases, is characterised by extensive remodelling of the Extracellular Matrix (ECM). Increasing evidence shows that ECM proteins regulate tumour growth, metastasis, immune responses, and resistance to therapy in cancers. However, the mechanism by which ECM molecules contribute to NSCLC progression remains unclear.

Objective: This study was conducted to identify which ECM molecules contribute to tumour progression in NSCLC.

Methods and Results: The expression of approximately 470 ECM genes was assessed in NSCLC compared to healthy controls from 3 gene array datasets, using bioinformatic analysis. These datasets are GSE33532 (n=80 lung cancer, n=20 healthy), GSE19804 (n=60 lung cancer, n=60 healthy), and GSE68465 (n=422 lung cancer, n=20 healthy). The top 20 most-changed ECM genes were identified from the datasets, and a selected, under researched ECM candidate was validated for gene expression by qPCR, and protein expression by immunoblot, in A549 lung cancer cells compared with BEAS-2B healthy lung epithelial cells, after 48-hour culture.

Bioinformatics analysis showed a top group of 20 ECM genes with a fold change ≥ 2 or ≤ -2 in multiple of the datasets above. Multimerin-1 (*MMRN1*) was significantly decreased in tumour samples from NSCLC patients compared to healthy controls in GSE33532 and GSE19804 ($p<0.0001$). *MMRN1* gene and protein expression were significantly decreased in the lung cancer cells (A549 cell line) compared to the normal epithelial cell line (BEAS-2B).

Conclusions: There are 20 ECM proteins consistently and highly dysregulated in NSCLC, and MMRN1 is a key ECM molecule which contributes to lung cancer progression.

POSTER SESSION: SCIENTIFIC ABSTRACT #7

Understanding the evolution of cross-reactive antibodies driving hepatitis C virus – associated cryoglobulinaemic vasculitis (HCV-MC)

Sophie G. Nides[1], Holly Hughes[1], Clara Young[2], Alexander P. Underwood[1,3,4], Rowena Bull[1,3,4], Deborah Burnett[1]

1. School of Biomedical Sciences, Faculty of Medicine and Health, UNSW Sydney, Sydney, NSW, Australia
2. Garvan Institute of Medical Research, Darlinghurst, NSW, Australia
3. Kirby Institute, Faculty of Medicine and Health, UNSW Sydney, Sydney, NSW, Australia
4. Copenhagen Hepatitis C Program (CO-HEP), Department of Infectious Diseases, Copenhagen University Hospital, Hvidovre and Department of Immunology and Microbiology, Faculty of Health and Medical Sciences, University of Copenhagen, Copenhagen, Denmark.

Introduction: HCV-associated cryoglobulinaemic vasculitis (HCV-MC) is an autoimmune disease affecting 10–15% of chronically infected HCV patients; up to 18% relapse after HCV clearance. Relapse is thought to reflect virus-induced molecular mimicry, potentially via the HCV E2 glycoprotein, though direct evidence is lacking. As E2 is the leading HCV vaccine candidate, clarifying its role in HCV-MC is important for anticipating vaccine-induced autoimmunity risks.

Methods: RF antibody sequences were sourced from two HCV-MC patient cohorts (Rockefeller University Hospital/New York-Presbyterian Hospital and St Vincent's Hospital SEARCH-C study) and from publicly available sequences representing other diseases. Germline-reverted RFs were cloned, co-transfected with CD79 into HEK293F cells, and assessed by flow cytometry for binding to Klickmer® reagents loaded with IgG or recombinant E2 (rE2) protein from an HCV-MC patient.

Results: Twenty RF sequences were identified, including eight from six HCV-MC patients and twelve from other disease contexts. All 20 used the IGHV1-69 germline gene, consistent with previously reported germline restriction proposed to underlie E2 cross-reactivity. Although these RFs were validated to bind IgG in their mature form when germline-reverted RFs were successfully expressed in HEK293F cells, only a minority bound IgG. We also discovered evidence of an RF that bound rE2 in its germline-reverted state.

Conclusions: For the first time, we have found evidence that a germline-reverted RF antibody can show cross-reactivity with HCV E2, providing preliminary evidence for molecular mimicry in HCV-MC. However, the majority of patient-derived RFs did not bind rE2, suggesting IGHV1-69 configuration alone is insufficient for E2 cross-reactivity. Larger repertoires and autologous E2 proteins are needed to clarify the contribution of antibody maturation and viral variation to pathogenesis, and to inform the safety of E2-based HCV vaccines.

POSTER SESSION: SCIENTIFIC ABSTRACT #8

Sleep and Human Movement: Mapping Their Potential Relationship in Osteoarthritis and Low Back Pain

Zhenbo Ma[1], Akhila Polisetti[1,2], Hanzhi Zhang[1], Laura E Diamond[3,4], David Klyne[5], Paulo Ferreira [1], Yareni Guerrero Ayala[6], Scott C. Starkey[7], Boliang Wang[1], Michelle Hall[1]

1. Musculoskeletal Research Hub, Charles Perkins Centre, The University of Sydney, Sydney, New South Wales, Australia
2. Boston University, Boston, Massachusetts, United States of America
3. Australian Centre for Precision Health and Technology (PRECISE), Griffith University, Gold Coast, Australia.
4. School of Allied Health, Sport and Social Work, Griffith University, Gold Coast, Australia.
5. Centre for Innovation in Pain and Health Research (CIPHeR), School of Health and Rehabilitation Sciences, The University of Queensland, Brisbane, QLD, Australia
6. Sydney Orthopaedic Research Institute, Landmark Orthopaedics, St Leonards, Sydney, Australia
7. Centre for Health, Exercise and Sports Medicine, Department of Physiotherapy, The University of Melbourne, Melbourne, Australia

Keywords: sleep, biomechanics, osteoarthritis, low back pain, musculoskeletal pain

Introduction: Sleep disturbance and altered movement (including gait, joint motion, coordination, and range of motion) are independently associated with pain and physical dysfunction in osteoarthritis (OA) and low back pain (LBP). However, the relationship between sleep and movement remains poorly understood. Understanding this relationship may help identify mechanisms linking sleep disturbance with pain and dysfunction, and opportunities to integrate sleep into musculoskeletal assessment and management.

Objective: This scoping review aimed to map the existing evidence examining associations between sleep disturbance and measures of movement and neuromuscular function.

Methods and Results: We searched PubMed and Scopus for studies measuring sleep and movement and movement or neuromuscular function in people with OA or LBP. Of 1,768 records identified, eight studies in OA (n=3) or LBP (n=5) were eligible. Six examined associations between sleep and movement or neuromuscular function. In LBP, greater sleep disturbance was generally associated with poorer balance and lumbar movement control. In knee OA, coexisting obstructive sleep apnoea was associated with lower peak knee extensor torque, whereas eccentric torque and submaximal torque steadiness did not differ. During rehabilitation for LBP, increased multifidus muscle volume was associated with reduced insomnia severity, whereas change in lumbar extensor strength was not. A pilot randomised controlled trial, targeting a reduction in knee loading by modifying sleeping position, found greater improvement in function, but not pain, compared to controls. A case-control study found greater sustained paraspinal muscle activity during sleep in people with LBP and paraspinal muscle spasm than in controls.

Conclusions: Preliminary evidence suggests a relationship between sleep disturbance and movement and neuromuscular function in OA and LBP. Overall, evidence was limited, heterogeneous, and predominantly cross-sectional, precluding conclusions about causality or directionality. Longitudinal and experimental studies integrating objective sleep and movement assessments are needed to clarify the direction and causality of this relationship.

POSTER SESSION: SCIENTIFIC ABSTRACT #9

***Ilex paraguariensis*-derived compound combinations exert synergistic anti-neuroinflammatory properties in LPS-stimulated mono- and tri-culture models**

Isabella Pregardier Klann[1], Dennis Chang[1], Gerald Muench[1,2], Xian Zhou[1]

1. NICM Health Research Institute, Western Sydney University, Westmead, NSW 2145, Australia.
2. School of Medicine, Western Sydney University, Campbelltown, NSW, 2560, Australia.

Keywords: Neuroinflammation, Synergy, *Ilex paraguariensis*, NF-κB, Tri-culture model

Introduction: Neuroinflammation is a key pathological feature of neurodegenerative disorders and contributes to neuronal dysfunction. *Ilex paraguariensis* (IP), a native South American plant traditionally consumed in Brazil as an herbal tea, has long been associated with health-promoting properties. However, the contribution of its individual compounds remains unexplored regarding central nervous system modulation.

Objective: This study aimed to investigate the effects of IP compounds in combination to achieve synergistic anti-neuroinflammatory and neuroprotective outcomes in lipopolysaccharide (LPS)-stimulated microglial cells.

Methods and Results: Network pharmacology and molecular docking analysis were performed to predict target compounds from IP and genes associated with neuroinflammation. Biological screening for 6 compounds (A – beta carotene, B – catechin, C – ellagic acid, D – eriodictyol, E – kaempferol, F – quercetin) was performed on LPS-induced microglia cells using Griess and Alamar Blue assays. Synergistic relation and dose ratio prediction were established by using CompuSyn and MATLAB software. Tumor Necrosis Factor (TNF)-α and interleukin (IL)-6 were assessed by ELISA. The nuclear factor-kappa B (NF-κB) translocation was examined by immunofluorescence, and proteins within the TLR4/TAK1/IκBα/NF-κB/COX-2/NLRP3 pathway were identified by Western blotting. A tri-culture model was applied with 3 different cell types (microglial N11, endothelial Bend.3 and neuronblastoma N2A) to mimic the neuronal environment. Barrier integrity was assessed using transendothelial electrical resistance and Evans blue permeability.

The DF (1:1) and DEF (30.8:32.1:37.1) combinations were predicted as optimal and synergistic by the software and selected for further investigation. DEF at 12.5 μg/mL produced the strongest effect in decreasing NO, TNF-α, and IL-6 in both models. Results were associated with attenuated NF-κB translocation, restored endothelial barrier, and thus presented a great potential for neuroprotection. This is in line with the illustration from *in sillico* analysis.

Conclusions: The combination of eriodictyol, kaempferol and quercetin exerted anti-neuroinflammatory properties and preserved neurovascular integrity following LPS stimulation in mono- and tri-culture models.

POSTER SESSION: SCIENTIFIC ABSTRACT #10

Harnessing T Cell Immunity Through mRNA Vaccines

Sahara Waide[1], Feyza Colakoglu Veli[1], Grace Peters[1], James Di Lisio[1], Alice Wong[1], Daniel Fernandez Ruiz[1]

1. University of New South Wales, Sydney, Australia

Keywords: mRNA vaccine, T cell immunology

Introduction: mRNA vaccines have transformed infectious disease prevention through their rapid development and versatility. However, their capacity to induce durable CD8+ T cell immunity against intracellular pathogens or cancer remains poorly understood. Defining these responses is essential for the advancement of next-generation mRNA vaccines.

Objective: To quantify and characterise CD8+ T cell memory responses, including circulating and tissue-resident memory subsets, elicited by commercially licensed mRNA-LNP vaccines in naïve C57BL/6 recipient mice.

Methods and Results: Naïve T cell receptor transgenic T cells specific for the model antigen chicken ovalbumin (OVA), termed OT-I cells, were isolated from the spleen of OT-I transgenic BL/6 mice and transferred intravenously into recipient naïve C57BL/6 mice. These mice were subsequently vaccinated 24 hours later with either Moderna 'Spikevax', Pfizer-BioNTech 'Comirnaty' or Patisiran 'Onpattro' lipid nanoparticle's containing mRNA encoding for the OT-I cell target, OVA. Harvesting of livers and spleens occurred at early effector, short-term and long-term memory timepoints. Relative frequencies and phenotypes of effector and memory OT-I cells were examined using the Cytek Aurora flow cytometer. All three of these mRNA-LNP vaccines induced robust effector T cell responses, but poor long-term memory. A prime-boost regime was investigated, with up to three booster doses given, but no improvement on memory responses was found. We also reveal, for the first time, that each vaccine differed in their capacity to induce circulating and resident memory T cell subpopulations.

Conclusions: While current mRNA-LNP platforms generate strong CD8 T cell effector responses, optimisation is required to establish durable circulatory and tissue-resident memory. These findings provide insight into platform optimisation for next-generation mRNA vaccines that induce long-lived protective CD8 T cell immunity against infectious diseases and cancer.

POSTER SESSION: SCIENTIFIC ABSTRACT #11

Investigating Epstein-Barr Virus-Associated Transcriptomic Signatures Across Gastric Carcinogenesis and Diverse Populations

Hossein Barzegar[1,2], Laurence Don Wai Luu[1], Mohammad Rabiei[2,3], Shanmugarajah Rajendra[2,3], Isidora Simovic[1], Khean-Lee Goh[4], Kwong Ming Fock[5,6,7], Shaghayegh Baradaran Ghavami[8], Hamid Asadzadeh Aghdaei[9,10], Gloria Liliana Porras-Hurtado[11], José Luis Cardona-Deazza[11], Juan José Montoya-Martinez[11], Antonio Javier Cadavid-Velez[11], Héctor William Toro-Hidalgo[11], Alba Ruth Cobo-Alvarado[11], Ofelia del Socorro Hincapié-Rincón[11], Natalia Castaño Rodríguez[1]

1. School of Biotechnology and Biomolecular Science, University of New South Wales, Sydney, Australia.
2. Gastro-Intestinal Viral Oncology Group, Ingham Institute for Applied Medical Research, Liverpool, Sydney, NSW 2170, Australia.
3. South Western Sydney Clinical School, University of New South Wales, Kensington, Sydney, NSW, Australia.
4. Department of Medical Microbiology, University of Malaya, Kuala Lumpur, Malaysia.
5. Department of Gastroenterology and Hepatology, Changi General Hospital, Singapore Health Services, Singapore City, Singapore.
6. Yong Loo Lin School of Medicine, National University of Singapore, Singapore City, Singapore.
7. Duke-NUS Medical School, Singapore City, Singapore.
8. Gastroenterology and Liver Diseases Research Centre, Research Institute for Gastroenterology and Liver Diseases, Shahid Beheshti University of Medical Sciences, Tehran, Iran.
9. Basic and Molecular Epidemiology of Gastrointestinal Disorders Research Centre, Research Institute for Gastroenterology and Liver Diseases, Shahid Beheshti University of Medical Sciences, Tehran, Iran.
10. Gastroenterology Department, Lismore Base Hospital, North New South Wales Local Health District, Lismore, NSW, Australia.
11. Clínica Comfamiliar Risaralda, Pereira, Risaralda, Colombia.

Keywords: Epstein-Barr virus; Gastric cancer; Transcriptomics; RNA-sequencing; Biomarkers

Introduction: Epstein-Barr virus (EBV) is detected in approximately 8% of gastric cancers and defines a distinct molecular subtype with unique molecular and pathological features. However, the role of EBV in premalignant gastric lesions and its associated host transcriptomic alterations across disease progression remain poorly understood.

Objective: To characterise EBV infection and associated host transcriptomic changes across the gastric disease spectrum, compare molecular signatures across diverse populations, and identify candidate EBV-associated biomarkers.

Methods and Results: We used a global gastric cancer consortium dataset comprising 92 samples from high-risk populations in Colombia, Iran, Singapore, and Malaysia, providing representation across diverse geographic regions with elevated gastric cancer burden.

Sample collection, RNA sequencing, and raw data preprocessing have been completed. In addition, public RNA-sequencing data from 159 gastric cancer patients were obtained from the GEO database. EBV detection and viral load quantification are being performed using digital PCR (dPCR). Differential expression analysis and cross-population transcriptomic comparisons are underway to identify EBV-associated molecular signatures. Particular attention is being given to pathways implicated in EBV-associated gastric carcinogenesis, including PI3K-AKT, MAPK, Wnt/β-catenin, Notch, TGF-β, and immune-related signalling pathways. Biomarkers identified through transcriptomic analyses will be prioritised for validation using quantitative PCR (qPCR). The final findings will be presented at the conference.

Conclusions: The identification of infection-associated transcriptomic signatures may provide insights into disease mechanisms and support the development of clinically relevant biomarkers for gastric cancer risk stratification and early detection.

POSTER SESSION: SCIENTIFIC ABSTRACT #12

Detection of High-Risk Human Papillomavirus in Primary Tumours and Matched Lymph Nodes of Oesophageal Adenocarcinoma

Hossein Barzegar[1,2], Natalia Castaño Rodríguez[1], Shanmugarajah Rajendra[2,3], Mohammad Rabiei[2,3,4]

1. School of Biotechnology and Biomolecular Science, University of New South Wales, Sydney, Australia.
2. Gastro-Intestinal Viral Oncology Group, Ingham Institute for Applied Medical Research, Liverpool, Sydney, NSW 2170, Australia.
3. South Western Sydney Clinical School, University of New South Wales, Kensington, Sydney, NSW, Australia.
4. School of Health, Medical and Applied Sciences, Central Queensland University, Sydney, NSW 2000, Australia.

Keywords: Oesophageal adenocarcinoma; Human papillomavirus; Lymph nodes; PCR; Digital PCR

Introduction: The role of high-risk human papillomavirus (hr-HPV) in oesophageal adenocarcinoma (OAC) remains uncertain. While HPV-positive lymph node metastases have been reported in cervical cancer, their presence and significance in OAC have not been investigated.

Objective: To evaluate hr-HPV in primary OAC tumours and matched lymph nodes, examine associations with clinicopathological characteristics and survival outcomes, and assess HPV detection using conventional PCR and digital PCR (dPCR).

Methods and Results: Formalin-fixed paraffin-embedded (FFPE) tumour and lymph node specimens, together with clinicopathological data, were collected from 35 patients who underwent surgical resection for OAC. A total of 279 tumour and 243 lymph node tissue sections were analysed for hr-HPV using conventional PCR. Associations between HPV status, clinicopathological characteristics, and survival outcomes were assessed. Hr-HPV was detected in primary tumours from 9/35 patients (25.7%), while 5/35 patients (14.3%) were positive in both primary tumours and matched lymph nodes. Patients with hr-HPV-positive primary tumours had significantly higher BMI than those with hr-HPV-negative tumours. No associations were observed between HPV status and survival outcomes. Viral load quantification and HPV genotyping using dPCR are ongoing.

Conclusions: This study represents the first assessment of hr-HPV in paired primary tumours and matched lymph nodes from OAC patients. Detection of hr-HPV in matched lymph nodes supports further investigation into the potential role of HPV in OAC progression. Ongoing dPCR analyses will provide additional insights into viral load, HPV genotype distribution, and HPV detection performance.

POSTER SESSION: SCIENTIFIC ABSTRACT #13

Whole transcriptome sequencing of adipose tissue eosinophils from obese mice identifies new potential anti-obesity therapeutic targets

Tsz Ho Li[1], Tia Valentini[1], Annalise Spek[1], Merlin Crossley[1], Kate Quinlan[1]

1. School of Biotechnology and Biomolecular Sciences, University of New South Wales, Sydney, New South Wales, 2052, Australia

Keywords: Immunometabolism, adipose tissue, eosinophils, obesity, RNA-sequencing**.**

Introduction: Obesity is a global health crisis affecting more than 650 million adults worldwide. Obesity is caused by energy imbalance, where there is greater energy intake than expenditure. The excess dietary energy is stored mainly as fat in adipose tissue (AT), and excessive fat storage results in obesity.

Adipose tissue-resident eosinophils (ATE) play an important role in regulating energy metabolism in adipose tissue. Eosinophil-deficient ΔdblGATA mice have increased adiposity compared to wild-type C57BL/6 mice when fed a high-fat diet (HFD), whereas hypereosinophilic IL5tg mice have smaller AT and better glucose tolerance. Moreover, ATE have been reported to drive AT beiging, a process that increases energy expenditure through thermogenesis, through secreted proteins.

Objective: To characterize obesity-associated changes in the ATE transcriptome and identify candidate ATE-secreted proteins with the potential to promote AT beiging and therefore that may represent anti-obesity therapeutic targets.

Methods and Results: We harvested the inguinal white adipose tissue (iWAT) and epididymal white adipose tissue (eWAT) from male C57BL/6JAusb mice fed either a chow or HFD for 12 weeks. Fluorescence-activated cell sorting was used to isolate ATE from both depots for bulk RNA-sequencing. In iWAT, 659 genes were upregulated and 551 genes were downregulated, while in eWAT, 436 genes were upregulated and 796 genes were downregulated in ATE from HFD-fed mice compared with chow-fed controls (adjusted p-value < 0.1). Computational screening of differentially expressed genes for secreted proteins and ligands using UniProt annotation and curated ligand-receptor database identified 255 candidate ATE-secreted proteins.

Conclusions: Using a murine model of diet-induced obesity, we show that the ATE transcriptome is extensively altered in obesity and identify 255 candidate ATE-secreted proteins. These findings provide a foundation for further investigation into the beiging-inducing potential of ATE-secreted proteins and their potential as novel therapeutic targets for obesity.

POSTER SESSION: SCIENTIFIC ABSTRACT #14

Identifying retrotransposons that regulate human T-cell immune responses

Billi Bragonzi[1], Samantha Owens[1], Renan Almeida[1], Cecile King[1]

1. School of Biotechnology & Biomolecular Sciences, UNSW, Sydney, NSW, Australia

Keywords: Immunology, Transposons, Cancer, Immune Checkpoints

Introduction: Retrotransposons are related to ancient viral sequences integrated into the genome and have recently emerged as important regulators of gene expression. Their species-specific evolution parallels that of immune genes, suggesting co-evolution with the immune system. LINE1 (Long Interspersed Nuclear Element-1) is the only autonomous and most active retrotransposon family in humans. Our group recently showed that a LINE1 element is essential for the biogenesis of a long non-coding RNA that modulates inflammation in mice, raising the possibility that LINE1 elements also regulate signalling pathways and immune responses in human immune cells.

Objective: To identify LINE1's retrotransposons that regulate T-cell activation and immune signalling in humans

Methods and Results: The retroCRISPR library targeting ~ 25,000 LINE1's was designed with cutting sites at regulatory region. Transduced Jurkat T-cells were stimulated with anti-CD3/28 to activate T-cell receptor signalling. Assessment of programmed cell death protein-1 (PD-1) and the high affinity chain of interleukin-2 receptor (CD25) by flow-cytometry enabled the identification of differential activation signatures associated with LINE1 perturbation. Preliminary screenings identified LINE1's whose disruption altered the proportion of activated T-cells, suggesting a functional role in immune regulation. In unstimulated transduced cells, spontaneous activation of PD-1 and CD25 was observed, indicating that a subset of LINEs may act as negative regulators of basal immune activation. PD-1 expression was also reduced in a pool of stimulated transduced cells, suggesting that additional LINE1's may function as positive regulators of activation-induced PD-1 expression.

Conclusions: These findings support the hypothesis that specific LINE1's contribute to the regulation of human T-cell activation and demonstrate the utility of retroCRISPR for the interrogation of LINE1's in immunity. The observed regulation of immune checkpoint PD-1 by LINE1's raises the possibility that retrotransposons influence immune responses within the tumour microenvironment and may represent a novel class of targets for future immunomodulatory drug discovery.

POSTER SESSION: SCIENTIFIC ABSTRACT #15

Evaluating the Quality and User Experience of Mobile Health Applications for Medication Adherence: Bridging Validated App Assessment with Real-World User Perspectives

Mohamed Afifi[1,2,3], Neil Spratt[1,2,3], Christopher Levi[1,2,3], Beata Bajorek[1,2,3]

1. School of Biomedical Sciences and Pharmacy, College of Health, Medicine and Wellbeing, University of Newcastle, Newcastle, NSW, Australia
2. Hunter Medical Research Institute, Newcastle, NSW, Australia
3. Hunter New England Local Health District, Newcastle, NSW, Australia

Keywords: medication adherence; mHealth; mobile applications; MARS; patient experience

Introduction: People living with chronic diseases play a central role in day-to-day self-management and develop considerable expertise through lived experience. Appropriate skills, resources and support may strengthen patients' capacity to manage medicines and improve health outcomes. Mobile health (mHealth) apps offer a potentially accessible means of supporting medication adherence; however, reminder or information-delivery functions alone may not provide meaningful self-management support. Effective apps should also be engaging, evidence-based and responsive to patients' real-world needs.

Objective: To identify the highest-quality freely available English-language medication adherence mHealth apps and examine the relationship between app quality, assessed using the Mobile Application Rating Scale (MARS), and end-user feedback.

Methods and Results: A desktop search of the iOS App Store and Android Play Store identified freely available English-language mHealth apps designed to support medication adherence. Eligible apps were evaluated using MARS across engagement, functionality, aesthetics and information quality. App-store ratings and reviews were examined to capture real-world user experiences, with reviews analysed thematically and compared with MARS findings. Most apps achieved good-to-excellent overall MARS scores (≥4.0). Jini Health (4.80), Medisafe Pill Reminder (4.70) and MyTherapy Medication Reminder (4.60) ranked highest. Functionality was consistently the strongest domain, while engagement and perceived impact were more variable. Users valued simplicity, reliable reminders, medication tracking and comprehensive drug information; dissatisfaction commonly related to technical problems and paywalls.

Conclusions: Freely available medication adherence apps can demonstrate high technical quality, yet validated quality scores do not always align with patients' real-world experiences. Combining MARS assessment with end-user perspectives provides a more patient-centred evaluation of app quality. Future apps should move beyond reminders to deliver engaging, accessible and evidence-based support that strengthens patients' knowledge, confidence and capacity to self-manage their medicines.

POSTER SESSION: SCIENTIFIC ABSTRACT #16

OncomiR miR-21 as a master regulator of non-coding RNAs in oral squamous cell carcinoma

Sarah M Stapleton[1], Dayna Sais[2], Deborah J Marsh[3], Nham Tran[1]

1. ncRNA Biology Lab, School of Biomedical Engineering, University of Technology Sydney, Sydney, NSW, Australia.
2. Sais Lab, Discipline of Biomedical Engineering, University of Technology Sydney, Sydney, NSW, Australia.
3. Translational Oncology Group, School of Life Sciences, University of Technology Sydney. Sydney, NSW, Australia

Keywords: MicroRNA. LncRNA. OSCC. Oral Cancer. Regulation.

Oral cancer is a highly aggressive malignancy with complex molecular underpinnings. Non-coding RNAs (ncRNAs), particularly microRNAs (miRNAs), play a crucial role in the regulation of gene expression and cellular processes associated with cancer progression. We believe that miRNAs not only regulate protein-coding genes but also interact with other miRNAs, and other long non-coding RNAs (lncRNAs) forming intricate regulatory networks. This study investigates the complexity of miRNA-mediated regulation of ncRNAs in oral cancer, with the hypothesis that oncogenic miRNAs (OncomiRs) play a critical role in a cascade of ncRNA regulation leading to increases in the hallmarks of cancer.

To investigate this, CRISPR Cas-9 was utilised to knock out miR-21 in the Oral Squamous Cell Carcinoma cell lines, UMSCC22B and SCC4. RNA-seq was undertaken to elucidate these potential miRNA-ncRNA interactions. Our results showed that miR-21 is a regulator to specific miRNAs such as miR-196a and miR-486 and long ncRNAs, including LINC07007. Interestingly, there is an enrichment of miR-21 sites on some of these ncRNA targets. Mapping of these interactions reveal nuanced ncRNA networks which could play a role in driving oral cancer carcinogenesis.

These findings are one of the few studies which provide in-depth analysis of the non-coding RNA networks in oral cancer. It expands on the idea that miRNAs can target other miRNAs and ncRNAs to orchestrate and/or redirect pathways required for transformation. With a greater understanding of regulatory networks and targets, this data could direct the future selection of biomarker and therapeutic targets for improved Oral cancer management.

POSTER SESSION: SCIENTIFIC ABSTRACT #17

A Model for Real-Time Detection of HCV Transmission in NSW Prisons

Bethany Horsburgh[1], Mykola Pinkevych[1], Alice Michie[2], Charles Foster[2], Lucy Peck[1], Nidhi Rao[1], Anthony Kelleher[1], Lachlan Coin[3], Behzad Hajarizadeh[1], William Rawlinson[2], Andrew Lloyd[1], Deborah Cromer[1] & Rowena Bull[1,4].

1. The Kirby Institute, Faculty of Health and Medicine, UNSW, Sydney, NSW, Australia
2. Serology and Virology Division (SAViD) NSW Health Pathology
3. University of Melbourne, Melbourne, Victoria, Australia
4. School of Biomedical Science, Faculty of Health and Medicine, UNSW, Sydney, NSW, Australia.

Hepatitis C virus (HCV) elimination efforts are challenged by ongoing silent transmission in high-risk settings, such as prisons. Genomic surveillance offers opportunities for early outbreak detection. Identifying recent transmission is difficult as HCV evolves rapidly and infections may remain undiagnosed for prolonged periods. Static genetic distance thresholds, commonly applied in pathogen surveillance, have limited utility for distinguishing recent from historical transmission events. We developed a model for identifying HCV transmission clusters that also accounts for ongoing virus evolution.

We developed a probabilistic model that accounts for the expected accumulation of HCV genetic diversity over time. Using more than 1,000 HCV sequences derived from prospective prison-based cohorts in Australia and previously published data, including epidemiologically confirmed transmission pairs and unrelated infections, we modelled the relationship between sampling interval and viral genetic distance.

The resulting framework generates a non-linear threshold that distinguishes likely recent transmission events (<1–2 years) from historical transmission events (>2 years). This model completely recapitulated the clustering patterns of a training dataset. When using the model on a testing dataset, fewer clusters requiring follow-up were identified compared to analysis using a classical genetic threshold. This model is now being applied to an MRFF -funded genomics-informed surveillance program operating across New South Wales prisons. The program integrates HCV whole-genome sequencing with demographic, behavioural and prison location data to identify emerging transmission clusters and generate actionable outbreak reports for prison health authorities.

A time-aware genomic model can improve identification of recent HCV transmission and provides a foundation for real-time public health surveillance. Integration of this approach into prison health systems represents a novel strategy to improve predictions from whole genome data, strengthen HCV outbreak detection and support progress towards HCV elimination in Australia.

POSTER SESSION: SCIENTIFIC ABSTRACT #18

Deciphering Macrophage-Epithelial-Fibroblast Crosstalk in Silicosis: A Multicellular Preclinical Modelling Approach

Sakthi Priya Selvamani[1,2], Ling Zhuang[1], Ben Johnson[1,2], Ellen Donohoe[1,2], Huaikai Shi[1,2], Richard Zelei[1,2], Vivek Dharwal[1,2], Maaike Kockx[4], Caroline Reddel[4], Deborah Yates[4,5,6], Anthony Linton[1,2,7], Elham Hosseini-Beheshti[1,2]

1. Asbestos and Dust Diseases Research Institute, Concord, NSW, Australia.
2. Sydney Medical School, University of Sydney, NSW, Australia.
3. ANZAC Research Institute, Sydney Local Health District, University of Sydney, Sydney, NSW, Australia.
4. Holdsworth House Medical Practice, Darlinghurst, NSW, Australia.
5. School of Clinical Medicine, University of New South Wales, NSW, Australia.
6. Respiratory & Sleep Medicine, Macquarie University Hospital, Macquarie University, NSW, Australia.
7. Concord Cancer Centre, Concord Repatriation General Hospital, Sydney, NSW, Australia.

Keywords: Silicosis, 3D spheroids, transwell, inflammation, fibrogenesis.

Introduction: Silicosis is an irreversible occupational lung disease that involves complex cellular mechanisms including inflammation, epithelial-mesenchymal transition (EMT), matrix remodelling, and fibrosis. Most of the research has utilised conventional 2D models. While animal models have provided valuable information on silicosis, they do have limitations and are difficult to access. With rapid technological advancements, it is important to explore 3D models to improve the understanding of silicosis.

Objective: To decipher the complex crosstalk among different cell types involved in the pathogenesis of silicosis – macrophages, alveolar and bronchial epithelial cells, and fibroblasts in various preclinical platforms.

Methods and Results: Silica nanoparticles induced cytotoxicity and inflammation in macrophages, as demonstrated by LDH assay and real-time PCR. 2D models of epithelial cells showed altered cell viability and inflammation upon exposure to silica-challenged macrophage secretome. 3D spheroid models of epithelial monocultures revealed differences in cell survival, inflammation, and EMT phenotype in alveolar epithelials as compared to bronchial epithelial cells, determined by viability assays, real-time PCR, and western blotting. Alveolar epithelial and fibroblasts co-cultured in 3D models demonstrated significant effects on cell viability, inflammation, and fibrotic remodeling compared to bronchial epithelial co-cultures by real-time PCR and western blotting. Transwell co-culture model highlights variations in the phenomenon of fibroblast transformation attributed to differences in paracrine signaling factors from alveolar and bronchial epithelial cells.

Conclusions: This study demonstrates that the silica secretome drives a cascade of disease pathogenesis, such as inflammation, a hybrid EMT phenotype, and fibrogenesis, that differ distinctly between alveolar and bronchial epithelial cells. Evidence from this research emphasises that transition to 3D models reveals cellular alterations poorly captured in 2D models. Collectively, our study highlights the importance of incorporating multicellular 3D models to understand better mechanisms from the onset to disease progression in silicosis, thereby serving as a platform for therapeutic screening.

POSTER SESSION: SCIENTIFIC ABSTRACT #19

Understanding the isomiR landscape of the parasitic flatworm *Fasciola hepatica*: Master regulators of developmental transitions and host immune evasion

Dayna Sais[1], Sumaiya Chowdhury[2], Sheila Donnelley[3], Nham Tran[4]

1. Sais Lab, Discipline of Biomedical Engineering, University of Technology Sydney, Sydney, NSW, Australia
2.School of Life Science, University of Technology Sydney, Sydney, NSW, Australia
3. Institute for Health, Discovery and Innovation, School of Biological and Chemical Sciences, University of Galway, Galway, Ireland
4. Non coding RNA Biology Lab, Discipline of Biomedical Engineering, University of Technology Sydney, Sydney, NSW, Australia

Keywords: microRNA, Fasciola hepatica, parasite, immune regulation, RNA

Introduction: Helminths survive within their hosts by evading innate immune detection. While microRNAs (miRNAs) are increasingly recognised as critical regulators of pathogen crosstalk, the role of structural variants (isomiRs) is largely unknown. We propose that *Fasciola hepatica* generates isomiRs during early development to exploit specific mammalian immune pathways to facilitate parasite growth and survival.

Objective: This study aimed to elucidate stage-specific dynamics of *F. hepatica* microRNA variants (isomiRs) and evaluate their functional capacity to engage host cellular machinery and modulate early inflammatory responses.

Methods and Results: *F. hepatica* has three distinct development stages, newly excysted juveniles (NEJ), Juveniles and Adult worms. Our data showed that isomiRs were temporally expressed during parasite maturation, with the highest abundance in the NEJ. Notably, >10% of isomiRs in NEJs are 5' variants which could fine tune target gene regulation. Interestingly, cleavage sites for *F. hepatica* primary miRNAs resemble mammalian motifs, suggesting an evolutionary adaptation to utilise the host RNAi network. We then investigated if cross kingdom regulation was possible. In mice infected with *F. hepatica,* host peritoneal macrophages at 6hr, 18hr and 5days post-infection, these cells showed an enrichment of parasite-derived miRNAs. One of the most abundant was the fhe-miR-125b-5p, 3` trimmed isomiR which could regulate the pro-inflammatory gene (Hif1a, Stat3 and Pin1). IP pulldowns show that this isomiR was highly associated with mammalian Ago2 compared to its canonical version.

Conclusions: Our findings offer insights into host-parasite cross-kingdom communication through the isomiRs network. We believe that isomiRs are an RNA wide adaptation for Fasciola to infect a wide range of hosts by immune evasion and regulation of developmental pathways.

POSTER SESSION: SCIENTIFIC ABSTRACT #20

Urine-derived kidney tubuloids as a patient-specific model of chronic kidney disease

Long The Nguyen[1,2], Sonia Saad[1,2] and Carol Pollock[1,2]

1. Renal Research Group, Kolling Institute, The University of Sydney
2. Kolling Institute, Royal North Shore Hospital

Background: Chronic kidney disease (CKD) remains to present significant challenges to both individuals and healthcare systems worldwide. Urine contains exfoliated living cells of the renal origin that have been shown to retain key pathology characteristics of CKD. Aim: The current study aims to develop an organoid model from patients-derived urinary cells to examine individual pathology and potential drug responsiveness.

Methods: Urinary cells from either healthy volunteer or patients with type 2 diabetes (T2D) with or without CKD were extracted and grown in two- and three-dimensional culture environment and characterised by RT-PCR, immunofluorescence and single-cell RNA sequencing.

Results: which showed increased deposition of collagen type 1 and reduced albumin uptake in the presence of Transforming growth factor beta 1. Single-cell RNA sequencing of the urinary tubuloids revealed 12 different cell clusters. Of these, 5 clusters were annotated as PTCs, 3 clusters were mesenchymal/stromal cells ad 2 were distal tubules/collecting ducts cells. Tubuloids from the T2D patient showed higher levels of adaptive, proliferative and remodelling PTC compared to the healthy control, suggesting adaptative responses. On the other hand, tubuloids derived from patients with CKD showed higher levels of injured and dedifferentiating PTCs, mesenchymal and fibroblasts in comparison to the control, suggesting irreversible tubular damage and fibrosis that resemble tubulointerstitial fibrosis in the actual tissue, which is well known as a hallmark of CKD progression.

Conclusion: Such close resemblance of pathology to each disease stage supports the use of urinary tubuloids as a patient-specific model of CKD.

POSTER SESSION: SCIENTIFIC ABSTRACT #21

Development of 24-Hour Movement Guidelines for Lower-Extremity Osteoarthritis: Protocol for an OARSI Initiative

Boliang Wang[1], Michelle Hall[1], Gillian Hawker[2], Robin Christensen[3,4], Melanie A. Holden[5], Joshua F. Baker[6,7], Dorothea Dumuid[8], Ryan S. Falck[9,10], Mengyun Luo[11,12], Monserrat Conde[13], Jillian Eyles[14], Siyi Zhu[15], Sandra Crone[16], Trish Silvester-Lee[17], Linda C. Li[9,17,18] †, Daniel K. White[19] †

† White and Li contributed equally as joint senior authors.

1. Musculoskeletal Research Hub, Charles Perkins Centre, The University of Sydney, Sydney, NSW, Australia
2. Department of Medicine, University of Toronto, Toronto, Ontario, Canada
3. Section for Biostatistics and Evidence-Based Research, the Parker Institute, Bispebjerg and Frederiksberg Hospital, Frederiksberg, Denmark
4. Research Unit of Rheumatology, Department of Clinical Research, University of Southern Denmark, Odense University Hospital, Odense, Denmark
5. School of Medicine, Keele University, Keele, United Kingdom
6. Division of Rheumatology, University of Pennsylvania, Philadelphia
7. Corporal Michael J. Crescenz VA Medical Center, Philadelphia, USA
8. School of Allied Health and Human Performance, College of Health, Adelaide University, Adelaide, Australia
9. Centre for Aging SMART, Vancouver Coastal Health Research Institute
10. School of Biomedical Engineering, Faculty of Applied Science, The University of British Columbia, Vancouver, British Columbia, Canada
11. Prevention Research Collaboration, Sydney School of Public Health, Faculty of Medicine and Health, The University of Sydney, Sydney, NSW, Australia
12. The Charles Perkins Centre, The University of Sydney, Sydney, NSW, Australia
13. Nuffield Department of Primary Care Health Sciences, University of Oxford, Oxford, UK
14. Sydney Musculoskeletal Health, Kolling Institute, Faculty of Medicine and Health, The University of Sydney, Sydney, NSW, 2065, Australia
15. Rehabilitation Medicine Center and Rehabilitation Medicine Research Institute, Key Laboratory of Rehabilitation Medicine in Sichuan Province, West China Hospital, Sichuan University, Chengdu, 610041, China
16. Patient Partner, Melbourne, Australia
17. Arthritis Research Canada, Vancouver, Canada
18. Department of Physical Therapy, Faculty of Medicine, University of British Columbia, Vancouver, Canada
19. Department of Physical Therapy, University of Delaware, Newark, USA.

Keywords: osteoarthritis, 24 hr movement behaviour, physical activity, sedentary behaviour, sleep

Introduction: Osteoarthritis is a leading cause of pain and disability. Physical activity (PA) is widely recommended for osteoarthritis management. Despite evidence linking sedentary behaviour and poor sleep with greater pain and physical dysfunction, these behaviours are often overlooked. Recent public health approaches increasingly recognise PA, sedentary behaviour, and sleep as interrelated behaviours across the 24-hour day, supporting integrated movement recommendations. However, no 24-hour movement guidelines currently exist for osteoarthritis.

Objective: To outline the development process for an Osteoarthritis Research Society International (OARSI) initiative to develop evidence-based 24-hour movement guidelines for lower-extremity osteoarthritis, considering PA, sedentary behaviour, sleep, and their combinations.

Methods and Results: A multidisciplinary international steering committee (n = 16) and independent consensus panel (n ≈ 20–30), including clinicians, researchers, methodologists, policymakers, and patient partners, will guide development. The guideline initiative was endorsed by OARSI in June 2026. Four systematic reviews will examine associations between (1) PA, (2) sedentary behaviour, (3) sleep, and (4) combinations of these behaviours with pain and physical function as critical outcomes, and health-related quality of life, fatigue, patient global assessment, and adverse events as important outcomes. Eligible studies will include cross-sectional, longitudinal, and interventional studies examining associations between movement behaviours and outcomes among adults with, or at high risk of, lower-extremity OA. Where appropriate, random-effects meta-analyses and dose-response analyses will be conducted. Findings will inform Grading of Recommendations Assessment, Development and Evaluation (GRADE) evidence profiles and recommendations developed through structured consensus using Evidence-to-Decision frameworks. Recommendations will be classified by strength and direction, with no recommendation made where evidence is insufficient or inconsistent.

Conclusions: This project will deliver the first evidence-informed 24-hour movement guidelines for adults with lower-extremity osteoarthritis. By considering PA, sedentary behaviour, and sleep, these guidelines aim to support clinicians, patients, and policymakers in optimising movement behaviours to improve osteoarthritis-related outcomes.

POSTER SESSION: SCIENTIFIC ABSTRACT #22

Mediation analysis of psychological and behavioural factors after adding aerobic physical activity to resistance exercise in hip osteoarthritis

Boliang Wang[1], Michelle Wang[1], Fiona Dobson[2], Aidan G. Cashin[3,4], David Klyne[5], Paulo Ferreira[1], Kim Allison[2], Gabrielle Knox[2], Rana Hinman[2], Kim L. Bennell[2], Libby Spiers[2], Michelle Hall[1,6]

[1]*Musculoskeletal Research Hub, Charles Perkins Centre, The University of Sydney, Sydney, New South Wales, Australia*
[2]*Centre for Health, Exercise and Sports Medicine, Department of Physiotherapy, The University of Melbourne, Melbourne, Victoria, Australia*
[3]*School of Population Health, Faculty of Medicine and Health, University of New South Wales, Sydney, Australia*
[4]*Centre for Pain IMPACT, Neuroscience Research Australia, Sydney, Australia*
[5] *Centre for Innovation in Pain and Health Research (CIPHeR), School of Health and Rehabilitation Sciences, The University of Queensland, Brisbane, Queesland, Australia*
[6]*Sydney Musculoskeletal Health, Kolling Institute, Faculty of Medicine and Health, The University of Sydney, Sydney, New South Wales, Australia*

Keywords: hip osteoarthritis, aerobic exercise, strengthening exercise, self-efficacy, mediation

Introduction: Exercise is a core treatment for hip osteoarthritis, yet the mechanisms through which different exercise approaches influence pain and physical function remain unclear. In the PHOENIX trial, adding aerobic physical activity to resistance exercise did not provide additional improvements in pain or physical function compared with resistance exercise alone. Examining psychological and behavioural mediators may help explain this finding.

Objective: To examine whether 3-month changes in psychological and behavioural factors mediate the effects of adding aerobic physical activity to resistance exercise, compared with resistance exercise alone, on 9-month pain and physical function.

Methods and Results: This secondary mediation analysis included 196 adults with hip osteoarthritis from the PHOENIX trial. We examined whether 3-month changes in eight factors (depressive symptoms, anxiety, stress, self-efficacy for walking duration, self-efficacy for walking contexts, fear of movement, sleep quality, and fatigue) mediated 9-month changes in pain and physical function. Indirect, direct, and total effects were estimated using regression-based counterfactual mediation models with bootstrapped confidence intervals (CIs), adjusted for age, sex, body mass index, and baseline mediator and outcome values. Missing data were handled using multiple imputation. The combined group showed greater improvement in self-efficacy for walking duration than resistance exercise alone. Greater improvement in self-efficacy for walking duration was associated with greater improvements in pain (path b −0.016, 95% CI −0.029 to −0.003) and physical function (path b −0.09, 95% CI −0.15 to −0.03). Corresponding indirect effects were −0.13 for pain (95% CI −0.31 to −0.03) and −0.83 for physical function (95% CI −1.92 to −0.04). No other factors showed evidence of indirect effects.

Conclusions: Improved self-efficacy for walking duration may be one pathway through which adding aerobic physical activity to resistance exercise influences pain and physical function. However, this pathway alone is unlikely to produce meaningful additional benefits over resistance exercise alone.

POSTER SESSION: SCIENTIFIC ABSTRACT #23

Identification of brain long non-coding RNAs involved in memory

Saba Altaf[1], Seonghee Jung[2], Mitchell Cummins[1], Rowan Heggen[1], Lachlan Ferguson[3], Anne Poljak[4], Kelly Clemens[3], Fabien Delerue[5], Lars Ittner[2], John Mattick[1,2]

1. School of Biotechnology and Biomolecular Sciences, Faculty of Science, UNSW, Sydney, Australia
2. Dementia Research Centre and Macquarie Medical School, Faculty of Medicine, Macquarie University, Sydney, Australia
3. School of Psychology, Faculty of Science, UNSW, Sydney, Australia
4. Bioanalytical Mass Spectrometry Facility, Mark Wainwright Analytical Centre, UNSW, Sydney, Australia
5. Department of Genetics, The University of Texas MD Anderson Cancer Center, Houston, USA

The mammalian brain expresses thousands of long noncoding RNAs (lncRNAs) with highly specific expression patterns, yet most remain functionally uncharacterised. Using RNAscope multiplex fluorescent in situ hybridisation combined with immunohistochemistry, we mapped the expression of hippocampal- and cerebellum-enriched lncRNAs in mouse brain, revealing distinct regional and subcellular distributions. Several lncRNAs were detected in neuronal projections, suggesting roles in synaptic regulation, while two lncRNAs, *Dory* and *Rodin,* were highly enriched in hippocampal subregions critical for learning and memory, with cell-type-specific expression in excitatory, inhibitory, and immature neurons.

Loss of *Dory* function in mice impaired spatial memory in females but not males, accompanied by perturbation of hormonal pathways, pointing to a role in sex-differentiated neural circuitry. This finding was corroborated by targeted antisense oligonucleotide knockdown of *Dory* in rat dorsal hippocampus, providing independent in vivo causal evidence for its role in hippocampus-dependent spatial memory. Loss of *Rodin* in mice prevented hippocampal memory consolidation. Together, these findings establish brain-expressed lncRNAs as key regulators of neural circuit function, with direct relevance to psychiatric and neurodevelopmental disorders.

POSTER SESSION: SCIENTIFIC ABSTRACT #24

To Enrich or Not to Enrich? Direct Sequencing of CTC-Derived Nucleic Acids from Blood in Childhood Cancer

Hafsah Atif[1,2], Yves Du Toit[2], Mojgan Toumari[2], Aurelie Moya[2], Kristina Warton[3], Omid R. Faridani[2,3], Rob Salomon[2]

1. School of Biomedical Sciences, University of New South Wales, Sydney, Australia
2. Children's Cancer Institute at Minderoo Children's Comprehensive Cancer Centre, Sydney, Australia
3. School of Clinical Medicine, University of New South Wales, Sydney, Australia

Keywords: Liquid Biopsy, Circulating Tumour Cells, Precision Oncology, Clinical translation, Molecular Diagnostics

Background: Liquid biopsy presents a minimally invasive approach to tumour detection and disease monitoring. Circulating tumour cells (CTC), can provide clinically informative material for characterising large structural variants and fusion genes, but their rarity in blood has limited their widespread use. Most current CTC-based approaches therefore rely on specialised enrichment or isolation workflows that can be technically complex, costly, difficult to standardise across laboratories, and risk loss of an already rare cell population. To overcome these limitations, direct analysis of CTC-associated nucleic acids from blood without enriching CTCs has the potential to retain clinically relevant molecular information while simplifying sample processing and improving translational feasibility.

Objective: To evaluate the feasibility of a CTC-informed liquid biopsy approach based on direct molecular analysis without prior CTC enrichment or isolation.

Methods and results: Nucleic acids were extracted from peripheral blood samples and analysed using low-pass whole genome sequencing (lpWGS). Sequencing libraries were generated from multiple patient samples, enabling genomic profiling without dedicated CTC enrichment. lpWGS identified tumour-associated genomic signals, supporting the feasibility of recovering biologically meaningful molecular information directly from unenriched blood. Building upon these results, targeted hybridisation capture and RNA-based workflows have been performed. The capability to increase detection sensitivity and characterise the molecular landscape accessible through this approach will be evaluated.

Conclusion: These findings demonstrate the potential of direct molecular analysis of CTCs as a simplified liquid biopsy strategy. Ongoing work will continue establishing the capability of different molecular techniques to increase the sensitivity and specificity of CTC detection. By reducing reliance on specialised CTC isolation technologies, this approach may facilitate broader clinical implementation of CTC-informed liquid biopsy workflows and support the development of more sensitive, accessible, scalable, and comprehensive blood-based molecular diagnostics.

POSTER SESSION: SCIENTIFIC ABSTRACT #25

ABSTRACT

The Use of Liquid Biopsy for High-Risk Stratification of HPV16-Positive Oropharyngeal Cancer Patients.

Nicole Burrows[1,2,3], Dayna Sais[1,2,3], Nham Tran[1,2,3]

1. School of Life Sciences. University of Technology Sydney. Sydney. NSW. Australia
2. Sais Lab, Discipline of Biomedical Engineering. University of Technology Sydney. Sydney. NSW. Australia.
3. Non-Coding RNA Biology Lab. Discipline of Biomedical Engineering. University of Technology Sydney. Sydney. NSW. Australia.

Keywords: Head and neck cancer; Human papillomavirus; Oropharynx cancer; P16 gene; P16-positive

Introduction:
Oropharyngeal squamous cell carcinoma (OPSCC) is cancer of the head and neck region and globally rising due to the virus, Human Papilloma Virus 16 (HPV16). HPV-16 contributes to HNSCC development through its E6 and E7 oncoproteins, which degrade the tumour suppressor protein p53 (TP53) and retinoblastoma (Rb). These OPSCC patients are typically HPV16-positive in approximately 90% of cases and generally have a better prognosis than HPV-negative patients. However, approximately 20% of patients experience disease recurrence, potentially associated with persistence or reactivation of the viral oncogenes. Currently, the standard clinical approach for determining HPV-associated OPSCC is immunohistochemical detection of the surrogate marker p16 in FFPE tumour tissue. While this is well established in routine pathology, it requires an invasive tissue biopsy and primarily indicates HPV association rather than ongoing viral oncogene activity. There is therefore a need to develop a non-invasive test that can detect not only the presence of HPV16, but also the activity or reactivation of the viral oncogenes, particularly in patients at risk of recurrence.

Objective: To develop a salivary HPV16 diagnostics to determine HPV16 presence and transcriptional activity using RT-qPCR..

Methods and Results: A multiplex RT-qPCR was utilised for the detection of the oncogenes E6 and E7, and URR. Optimisation of the assay initially involved testing each target in singleplex reactions using synthetic gBlock controls and a panel of OPSCC cell lines, including the HPV16-positive cell lines SCC154 and SCC90 and the HPV16-negative cell line 22B. Following singleplex optimisation, the targets were combined into a multiplex configuration containing all HPV16 targets together with a positive reference gene. The multiplex assay demonstrated a broad analytical detection range, reliably detecting HPV16 targets from 50,000 copies down to 5 copies per reaction. We next investigated the clinical applicability of the assay using saliva samples collected from patients with OPSCC. Total nucleic acid (TNA) was extracted from 116 self-collected saliva specimens. TNA was isolated from 200 µL of saliva using an automated KingFisher Duo system, with an average nucleic acid yield of approximately 50 ng/µL. Application of the multiplex assay to these clinical samples demonstrated successful detection of HPV16, while the reference gene was detected in all samples, confirming successful nucleic acid extraction and sample integrity.

Conclusions: Our findings indicate that saliva is a suitable source material for the detection of HPV16 using a four-plex RT-qPCR assay.

POSTER SESSION: SCIENTIFIC ABSTRACT #26

The endogenous retrovirus MMERVK9C maintains immune self-tolerance

Iris Chang[1], Howard Wang[1], Cecile King[1]

1. School of Biotechnology & Biomolecular Sciences, University of New South Wales, Sydney, NSW, Australia

Keywords: Immunology, Autoimmune disease, Lupus, Transposons, Endogenous retroviruses

Introduction: Endogenous retroviruses are remnants of past retroviral infections which have integrated into the genome, and can influence host gene expression and cellular function. These genetic elements have substantial regulatory potential within the immune system, as retroviral insertions are enriched near immune response genes. The endogenous retrovirus MMERVK9C is of particular interest due to its location within an interferon-stimulated gene cluster previously implicated in autoimmune disease.

Objective: This study aims to identify the potential genetic and immune regulatory functions of MMERVK9C.

Methods and Results: A MMERVK9C-/- mouse line was generated by CRISPR-Cas9 mediated deletion of the entire MMERVK9C retroviral sequence on a C57BL/6JAusB mouse background. Following this, genetic and phenotypic analyses were performed by RT-qPCR, flow cytometry, immunofluorescence microscopy, and ELISA. MMERVK9C-/- mice spontaneously developed a lupus-like phenotype from 20 weeks of age characterised by splenomegaly, expansion of neutrophils, T cell activation with reduced regulatory T cell populations, as well as increased germinal centre and extrafollicular B cells and elevated circulating anti-nucleic acid autoantibodies.

Conclusions: Although endogenous retroviruses are typically associated with the promotion of autoimmune disease, these findings identify MMERVK9C-/- as a novel mouse model of spontaneous lupus and suggest that MMERVK9C helps maintain immune self-tolerance, thereby restraining autoimmune disease. This study provides unique evidence that endogenous retroviruses can provide regulatory function in the immune system.

POSTER SESSION: SCIENTIFIC ABSTRACT #27

Modulation of YAP1 signalling by Compound A in Renal Tubular Injury and Diabetic Kidney Disease

Qr Chen, Qh Cao, Xm Chen, C Pollock

University of Sydney, Kolling Institute, Royal North Shore Hospital, New South Wales, Sydney, Australia.

Background: Diabetic kidney disease (DKD) is characterised by progressive tubular injury and fibrosis. Yes-associated protein (YAP1), a key effector of the Hippo signalling pathway, regulates cellular proliferation, survival, and tissue homeostasis. Dysregulated YAP1 signalling has increasingly been implicated in renal injury and fibrosis. However, its contribution to renal tubular injury in DKD remains incompletely understood. Compound A is an investigational pharmacological agent with established redox-modulating properties, but its effect on YAP1 signalling in kidney injury remains unclear. We therefore investigated whether Compound A modulates YAP1 activation and YAP1-associated profibrotic signalling.

Aim: This study investigates the renoprotective effects of Compound A and determines whether these are associated with modulation of YAP1 signalling in renal tubular injury in vitro and in vivo.

Method: The effects of Compound A on renal injury and YAP1 signalling were investigated in endothelial nitric oxide synthase-deficient (eNOS-/-) mice with streptozotocin (STZ) induced type 1 DKD and in rat renal proximal tubular epithelial (NRK52E) cells. Renal YAP1 expression and localisation were assessed by immunohistochemistry. In vitro, lysophosphatidic acid (LPA) was used to induce YAP1 activation, and time-course experiments were performed to characterise the kinetics of YAP1 signalling. YAP1 activation and nuclear localisation were assessed by immunofluorescence microscopy and Western blotting. YAP1 downstream targets (CTGF/CCN2, CYR61/CCN1, ANKRD1 were further examined by qPCR and Western blotting. Pharmacological YAP1 inhibition (Verteporfin) and YAP1-specific siRNA were used to further intvestigate the functional contribution of YAP1 signalling to renal injury and fibrosis.

Results: YAP1 was aberrantly activated and upregulated in renal proximal tubular epithelial cells under experimental injury conditions in vitro and *in vivo*. Time-course analysis revealed dynamic LPA-induced YAP1 activation in NRK-52E cells, accompanied by enhanced nuclear localisation. Compound A attenuated LPA-induced YAP1 activation and nuclear translocation *in vitro*. In diabetic kidneys, tubular YAP1 expression was upregulated, whereas Compound A treatment attenuated this response.

Conclusion: These findings identify dysregulated YAP1 signalling as a potential contributor to renal tubular injury and fibrosis and demonstrate that compound A attenuates YAP1 activation under experimental kidney injury condition. Modulation of YAP1 signalling may therefore contribute to the renoprotective effects of Compound A, highlighting YAP1 as a potential therapeutic target in DKD.

POSTER SESSION: SCIENTIFIC ABSTRACT #28

Measuring circulating cell-free mitochondrial DNA and cell-free DNA may detect biomarkers in gestational diabetes mellitus

Ya-Wen Chiang[1,2], Therese Becker[2,3], Tanzila khan[2,3], Jon Hyett [1,4,5], Yafeng Ma [1,2]

1. South Western Sydney Obstetric Research Unit, Ingham Institute for Applied Medical Research, Liverpool, NSW, Australia
2. South Western Clinical School, UNSW, Liverpool, NSW, Australia
3. Centre for Circulating Tumor Cell Diagnostics and Research, Ingham Institute for Applied Medical Research, Liverpool, NSW, Australia
4. School of Medicine, Western Sydney University, Sydney, NSW, Australia
5. Fetal-Maternal Unit, Liverpool Hospital, Sydney, NSW, Australia

Background: Gestational diabetes mellitus (GDM) affects 18% of pregnancies in Australia and may have pathological long-term effects on mothers and infants. Prevention of the condition requires better diagnostic markers. Emerging evidence suggests mitochondrial dysfunction may be associated with GDM. Mitochondrial DNA (mtDNA) functions as an inflammatory signalling molecule, and circulating cell-free mitochondrial DNA (ccf-mtDNA) in maternal circulation induces intracellular pro-inflammatory signalling and hyperactive innate immune responses during pregnancy, potentially causing complications. ccf-mtDNA copy number (CN) and methylation changes may thus be associated with GDM.

Hypothesis: ccf-mtDNA CN and methylation changes are early diagnostic GDM biomarkers.

Aims: 1) to establish dual droplet digital PCR (ddPCR) assays to quantify ccf-mtDNA-CN and methylation levels; 2) circulating cell- free DNA (ccfDNA) fragmentation; 3) to determine the potential clinical ccf-mtDNA utility as an early GDM predictor.

Methods: First-trimester pregnant patients are recruited through the Leto biobank (Ethics:2023/PID00973, n=95). Clinical patient outcome data are collected from the local health district eMR. ccfDNAs are extracted from plasma samples. Dual ddPCR assays are designed to measure ccfDNA fragmentation by normalisation to 70bp and 360bp amplicons of the LINE-1 reference gene. ccf-mtDNA CN is determined by detection of mitochondrial genes MT-ND1 and MT-CYB, normalised to RPP30 DNA and 18S ribosomal RNA (rRNA) reference genes. cff-mtDNA methylation levels are detected in the mitochondrial D-loop gene.

Results: GDM (n=31) and healthy (n=35) pregnancy patients recruited, complete clinical data extracted, and cffDNA isolated. To clarify the diagnostic utility of these tests, ddPCR data acquisition and analysis are underway.

POSTER SESSION: SCIENTIFIC ABSTRACT #29

Impact of tumour model complexity on ovarian cancer growth kinetics and drug response across 2D, 3D spheroid, and tumour-on-chip models

Layal Dani[1], Amy L. Bottomley[2], Kristie-Ann Dickson[1], Deborah J. Marsh[1], and Amani Alghalayini[1]

1. Translational Oncology Group, School of Life Sciences, Faculty of Science, University of Technology Sydney, Ultimo, NSW, Australia.
2. Microbial Imaging Facility, School of Life Sciences, Faculty of Science, University of Technology Sydney, Ultimo, NSW, Australia.

Keywords: Ovarian Cancer, tumour microenvironment, 3D spheroid models, microfluidic tumour-on-chip, precision medicine.

Introduction: Ovarian cancer is the most lethal gynaecological malignancy worldwide. Current preclinical models have limited ability to recapitulate the tumour microenvironment. Conventional 2D cell culture lacks complex cellular interactions, extracellular matrix components, and physiological gradients present *in vivo*. 3D spheroid and tumour-on-chip culture conditions offer increasingly relevant platforms for modelling tumour behaviour and therapeutic response.

Objective: To determine how increasing model complexity, through 3D architecture, stromal fibroblasts and endothelial cells influences ovarian cancer cell growth dynamics and therapeutic drug response.

Methods and Results: High-Grade Serous Ovarian Cancer cell line Kuramochi, immortalised Normal Ovarian Fibroblasts, NOF151-hTERT, and primary Human Umbilical Vein Endothelial Cells (HUVECs) were cultured under mono-, co-, and tri-culture conditions in 2D monolayers, 3D spheroids and microfluidic tumour-on-chip models. Growth dynamics were monitored using live-cell imaging platforms and tumour growth, spheroid formation, morphology, and viability were quantified using IncuCyte Live-Cell Analysis and Fiji ImageJ software. Preliminary experiments demonstrated distinct growth patterns between models and culture conditions. Drug sensitivities to the chemotherapeutic drug carboplatin, and the repurposed therapeutic drug efavirenz, were tested in all models and compared across all culture conditions. Statistically significant differences in drug sensitivities were identified.

Conclusions: Preliminary findings suggest that incorporation of stromal fibroblasts and endothelial cells altered ovarian cancer growth dynamics and therapeutic response. Distinct differences, such as increases in proliferation rate and decreases in drug sensitivity, were observed between different model and culture conditions, highlighting the importance of model selection for therapeutic evaluation. Increasingly complex models that better mimic *in vivo* conditions have the potential to provide enhanced physiological relevance for investigating ovarian cancer growth, multicellular interactions, and therapeutic responses, by better capturing the key features of the tumour microenvironment. Ongoing work will further characterise growth kinetics, drug sensitivity, and microenvironmental influences to support the development of more predictive *in vitro* ovarian cancer models.

POSTER SESSION: SCIENTIFIC ABSTRACT #30

Size and concentration of extracellular vesicles in transfusable blood components are influenced by manufacture and storage conditions

Bailey Deagon[1,2], Ben Winskel-Wood[1], Joanne Tan[1,3], Matthew Padula[2], Denese Marks[1,3], Lacey Johnson[1,2,4]

1. Research and Development, Australian Red Cross Lifeblood, Alexandria, NSW, Australia
2. School of Life Sciences, University of Technology Sydney, Ultimo, NSW, Australia
3. Sydney Medical School, University of Sydney, Camperdown, NSW, Australia
4. School of Science, RMIT University, Melbourne, VIC, Australia

Keywords: Transfusion, extracellular vesicles, haemorrhage, haemostasis, storage

Introduction: Transfusable blood components are critical for managing haemorrhage and deficiencies in essential blood factors. Extracellular vesicles (EVs) are also present in blood components; although the impact of collection, manufacturing and storage conditions on their biogenesis in blood components remains unclear.

Objective: To quantify EV size and concentration in transfusable blood components.

Methods and Results: Whole blood (WB; n=6) was collected into CPD anticoagulant and filtered to remove leukocytes but retain platelets. Red cell concentrates (RCCs; n=8) were prepared in SAG-M. WB and RCCs were stored at 2-6°C and tested over six weeks. Platelets were collected via apheresis in 60% PAS-E/40% plasma and stored at 20-24°C with agitation (RT-PLTs; n=8), at 2-6°C (Cold-PLTs; n=8) or frozen with 5-6% DMSO (Cryo-PLTs; n=8). RT-PLTs and Cold-PLTs were tested over three weeks, and Cryo-PLTs were tested after thawing.

Crude, EV-containing supernatants were collected using centrifugation at 800*x*g for 20 minutes and analysed using Nanoparticle tracking analysis (NS300, Malvern Panalytical). Baseline (day 1) EV concentrations were highest in WB (3.32±2.68x10^{11}/mL), followed by platelets (1.01±5.81x10^{11}/mL) and RCCs (0.06±0.08x10^{11}/mL). The largest increase in EV concentration was observed in RCC following standard 42-day shelf-life (0.47±0.34x10^{11}/mL; p=0.031). PLT-EVs increased significantly after storage for 21 days (RT-PLTs: 3.43±1.59x10^{11}/mL; p=0.002; Cold-PLTs: 3.44±2.51x10^{11}/mL; p=0.040), but were highest in Cryo-PLTs (5.61±1.54x10^{11}/mL; p<0.001).

The mean particle size of EVs increased during storage in WB (day 1: 113.7nm; day 42: 157.9nm; p=0.048), RCCs (day 1: 139.1nm; day 42: 182nm; p=0.003) and Cold-PLTs (day 1: 106.9nm; day 21: 149.3nm; p=0.002). Larger EVs were observed in cold-platelets (152±19nm) compared to RT-PLTs (112±9 nm; p<0.0001) from day 7 onwards.

Conclusions: These findings demonstrate that EV concentration and size are influenced by storage conditions, likely reflecting differences in cellular origin. Phenotypic characterisation is underway using markers to define cell of origin, externalised-phosphatidylserine and tetraspanin expression.

POSTER SESSION: SCIENTIFIC ABSTRACT #31

Surveillance and Characterisation of Arboviruses in the Hunter Region: Establishing Molecular Surveillance Tools for Detection in Mosquitoes

Carissa Enriquez[1,2], Camille Esneau[1,2], Verlaine Timms[3], Brett Neilan[3], Nathan Bartlett[1,2]

1. School of Biomedical Sciences and Pharmacy, University of Newcastle, Callaghan, NSW, Australia
2. Hunter Medical Research Institute, New Lambton Heights, NSW, Australia
3. School of Science, University of Newcastle, Callaghan, NSW, Australia

Keywords: Arbovirus; Surveillance; Mosquito; Newcastle.

Introduction: According to the NSW Arbovirus Surveillance and Mosquito Monitoring Program data from the last four monitoring seasons, Newcastle has reported high to very high weekly average numbers of two species of mosquitoes: *Aedes vigilax* and *Culex annulirostris*. These are important vectors of Ross River virus (RRV), Barmah Forest virus (BFV), Murray Valley encephalitis virus (MVEV), and Japanese encephalitis virus (JEV). As such, these viruses are monitored in the program's limited panel of targets. However, the full diversity of circulating arboviruses, including emerging variants or novel viruses in Newcastle are not characterised. Given Newcastle's ecological suitability for mosquito proliferation and proximity to an international transport hub, enhanced molecular surveillance is needed to better understand local arbovirus circulation.

Objective: To expand on virus-specific qPCR-based surveillance by incorporating genus-level arbovirus detection, identify circulating arboviruses using field-collected mosquitoes, and characterise detected viruses using sequence analysis, molecular cloning techniques and cell-based assays.

Methods and Results: Using Encephalitis Virus Surveillance (EVS) traps, mosquitoes were collected across locations throughout the Newcastle area from 2022 to 2024, and in 2026. Species identification was performed, resulting in the identification of 21 species. Currently, a total of 362 samples were generated from pools of mosquitoes of varying sizes (1-50). These samples consist of 19 NSW Health-provided positive controls (RRV, BFV, MVEV and JEV) and 343 total RNA from field-collected mosquitoes. Currently, 249 samples have been screened for BFV and RRV using virus-specific qPCR. Excluding positive controls, no arboviruses have been detected to date in field-collected samples. Wider screening and genus-level detection, pan-flavivirus and pan-alphavirus primers are currently being optimized. This, coupled with high-throughput automated extraction and expanded field sampling will support broader study of arbovirus diversity in Newcastle.

Conclusion: These early findings lay the groundwork for expanded arbovirus surveillance to better understand arboviruses circulating in Newcastle.

POSTER SESSION: SCIENTIFIC ABSTRACT #32

Citrus aurantium L. (Bitter Orange) Extracts and its Bioactive Compounds Reduces Inflammatory Responses in Macrophage and Human Dermal Fibroblast Models

Belinda Huor[1,2], Dennis Chang[2], Xian Zhou[2],

1. School of Science, Western Sydney University, Campbelltown, NSW 2560, Australia
2. NICM Health Research Institute, Western Sydney University, Westmead, NSW 2145, Australia

Keywords: Citrus Aurantium L., skin inflammation, anti-inflammatory, aringin, neohesperidin

Introduction: Citrus Aurantium L. (bitter orange, BO) has been traditionally used in herbal medicine to relieve indigestion and bloating across several Asian countries. BO is rich in bioactive compounds, including flavonoids, alkaloids, essential oils and vitamin C, which can be attributed to its anti-inflammatory properties. However, studies evaluating the anti-inflammatory potential of BO and its compounds on the skin are limited.

Objective: This study investigated the anti-inflammatory potential of hydroethanolic extracts of different BO parts and its bioactive compounds including naringin and neohesperidin.

Methods and Results: BO was harvested and authenticated by the Royal Botanic Gardens Sydney, and various BO extracts on leaf, pulp and peel were prepared. Naringin and neohesperidin, from the extracts were quantified using high-performance liquid chromatography (HPLC)-photodiode array (PDA). The anti-inflammatory potential of extracts and compounds were evaluated in LPS-induced RAW 264.7 and human dermal fibroblasts (HDF) cells using Griess assay and enzyme-linked immunosorbent assay (ELISA) for interleukin (IL)-6 expression. Naringin content was highest in the pulp extract (25.26±0.90 mg/g) and neohesperidin was highest in the peel extract (26.50±0.20 mg/g). All extracts and compounds reduced nitric oxide (NO) and IL-6 levels in LPS-induced RAW 264.7 cells and HDF cells with a dose-dependent manner. The peel extract had the highest therapeutic index (TI=5.94) on RAW 264.7 cells compared to other extracts and compounds. The pulp extract was the least cytotoxic on HDF cells (LC50>500 µg/mL). However, leaf and peel extracts were more potent inhibitors of NO in RAW 264.7 cells (IC50=26.39±1.46; 27.00±3.85 µg/mL) and IL-6 in HDF cells than pulp extract and the two compounds, suggesting a more complex interplay between the compounds and other bioactive constituents.

Conclusions: Results suggest that BO, naringin and neohesperidin may play a role in alleviating skin inflammation. Further studies are warranted for mechanistic investigation into synergistic combinations of compounds.

POSTER SESSION: SCIENTIFIC ABSTRACT #33

N-myristoyltransferase inhibition as a host-directed antiviral strategy against rhinovirus infection in a human airway epithelial model

Cassandra Jarvis[1,2], Jason Girkin[1,2], Su-Ling Loo[1,2], Camille Esneau[1,2], Ethan McDaid[1,2], Nathan Bartlett[1,2]

1. Viral Immunology and Respiratory Disease Group, School of Biomedical Sciences and Pharmacy, College of Health Medicine and Wellbeing, University of Newcastle, Newcastle, NSW, Australia.
2. Infection Research Program, Hunter Medical Research Institute, New Lambton Heights, NSW, Australia.

Keywords: Rhinovirus; N-myristoyltransferase; antiviral; host-directed therapy; NMT inhibitor

Introduction: Rhinoviruses (RV) are the predominant cause of the common cold and major trigger of exacerbations of chronic respiratory diseases, including asthma and chronic obstructive pulmonary disease. Despite the substantial clinical and economic burden associated with RV-induced diseases, there are currently no licensed antiviral therapies targeting RV. This has led to research into host cell molecules required for RV replication. N-myristoyltransferase (NMT) is a host enzyme required for myristoylation of the viral capsid precursor protein VP0, an essential step in RV assembly and replication. Consequently, NMT inhibition represents a promising host-directed antiviral strategy with a reduced risk of viral resistance. While antiviral activity of NMT inhibitors has previously been demonstrated in monolayer cell cultures, their potential for topical respiratory delivery remains unexplored. Here, we evaluated the NMT inhibitor IMP-1320 in human airway epithelial models to support development of a topical intranasal antiviral and tested the hypothesis that topical NMT inhibition would suppress RV replication.

Objective: To investigate the antiviral activity of an NMT inhibitor against RV infection in human airway epithelial cells.

Methods and Results: BCi-NS1.1 human airway epithelial cells were infected with RV-A1 at a multiplicity of infection of 0.5 and treated with increasing concentrations of IMP-1320. Cells were harvested at 48- and 72-hours post-infection and viral RNA was quantified using qPCR and infectious virus was quantified by 50% tissue culture infectious dose (TCID50) assay. Log_{10} concentration-response curves were generated to estimate the half-maximal inhibitor concentration (IC50) using GraphPad Prism. Preliminary analysis demonstrated a concentration-dependent reduction in RV viral RNA and infectious virus compared with untreated infected controls.

Conclusions: This study using complementary measures of viral replication and supports further development of NMT inhibitors as potent, host-directed antiviral therapies with low probability of resistance from viral drift. Our findings will inform subsequent investigation in more physiologically relevant airway models and translation to *in vivo* studies.

POSTER SESSION: SCIENTIFIC ABSTRACT #34

Viral and oxidative injury induce epithelial IL-25: a potential driver of lung fibroblast activation

Jingyue Kang[1,2], Nathan Bartlett[1,2], Mingtao Liang[1]

1. School of Biomedical Sciences and Pharmacy, College of Health, Medicine and Wellbeing, University of Newcastle, Callaghan, NSW, 2308, Australia.
2. Infection Research Program, Hunter Medical Research Institute, New Lambton Heights, NSW, 2305, Australia.

Keywords: Interleukin-25. Rhinovirus. Alveolar epithelial cells. Lung fibroblasts. Pulmonary fibrosis.

Introduction: Idiopathic pulmonary fibrosis (IPF) is a progressive lung disease characterised by repetitive alveolar epithelial injury, aberrant tissue repair and excessive extracellular matrix deposition. Injured alveolar epithelial cells can release cytokines that promote epithelial–fibroblast crosstalk and fibroblast activation. Previous studies have identified increased IL-25 expression in IPF and demonstrated that IL-25 can promote lung fibroblast proliferation and pro-fibrotic mediator expression. However, how different forms of epithelial injury regulate IL-25 production and whether epithelial-derived IL-25 contributes to subsequent fibroblast activation remain unclear.

Objective: To determine whether viral infection, oxidative stress and DNA damage induce IL-25 expression in alveolar epithelial cells and whether epithelial-derived IL-25 regulates lung fibroblast activation.

Methods and Results: A549 alveolar epithelial cells were infected with RV-A1 (MOI 0.1; 24, 48 or 72 h) or exposed to H2O2 (0.3 or 1 mM for 1 h; harvested 24 h later). IL-25 mRNA was quantified by qPCR. To assess the contribution of IL-25, IL-25-targeted siRNA (10 μM) was transfected into A549 cells 24 h prior to H2O2 treatment. RV-A1 significantly increased IL-25 expression, with the greatest induction at 72 h. H2O2 also increased IL-25 expression, whereas IL-25 knockdown significantly attenuated the H2O2-induced response. Ongoing studies will extend the injury model to etoposide and assess the functional consequences of epithelial-derived IL-25. Conditioned medium from injured A549 cells will be transferred to MRC-5 lung fibroblasts; IL-25 secretion will be quantified by ELISA, and fibroblast proliferation, pro-fibrotic signalling and extracellular-matrix markers (α-SMA, collagen and fibronectin) will be assessed. IL-25 knockdown will test the specific contribution of epithelial-derived IL-25.

Conclusions: Preliminary findings show that viral and oxidative injury induce epithelial IL-25 expression. Ongoing studies incorporating etoposide-induced epithelial injury and conditioned-medium transfer to MRC-5 fibroblasts will determine whether epithelial-derived IL-25 contributes to fibroblast activation and pro-fibrotic signalling.

POSTER SESSION: SCIENTIFIC ABSTRACT #35

Mapping circulating norovirus genotypes in clinical acute gastroenteritis cases 2025

Dillon M. C. Y. Lai[1], Lewis K. Mercer[1,2,3,4], William D. Rawlinson4,5, Stuart Hamilton[1], Peter A. White.

1. School of Biotechnology and Biomolecular Sciences, Faculty of Science, University of New South Wales, Sydney, Australia
2.. NSW Health Pathology, Department of Microbiology, Prince of Wales Hospital, Sydney, A.ustralia
3. School of Medical Sciences, Faculty of Medicine, University of New South Wales, Sydney, Australia
4. School of Women's and Children's Health, Faculty of Medicine, University of New South Wales, Sydney, Australia
5. Virology Research Laboratory, SEALS Microbiology, Prince of Wales Hospital, Sydney, Australia

Main Text

Norovirus causes 680 million cases of acute gastroenteritis (AGE) and is responsible for approximately 220,000 deaths annually worldwide. In addition, the economic impact is estimated at $2.7 billion in direct healthcare costs and $40 billion in societal costs.

The aim of this study was to determine the genetic diversity of norovirus strains circulating in NSW between July 2025 and December 2025, to determine if new strains have emerged, to discover recombinant strains and older strains that have re-emerged. We also aimed to identify norovirus strains that caused outbreaks in NSW.

Viral RNA was extracted from clinical samples and amplified by RT-PCR. Amplicons were Sanger sequenced and genotyped using phylogenetic analysis.

Including all outbreaks and clinical cases, there were 505 total cases of norovirus sampled. 278 samples were selected and of these, 60% were successfully genotyped and the dominant strain was GII.4 Sydney 2012 [P16], which accounted for 39% of cases. In 2024, GII.17 [P17] was the dominant epidemic strain and accounted for 34% of cases. However, during the second half of 2025, GII.17 [P17] reduced in prevalence by 25%, and was replaced by the re-emergence of GII.2 [P16]. Among the four outbreaks analysed, the strains that caused the outbreaks were GII.4 Sydney 2012 [P16], GII.17 [P17], GII.6 [P7] and GII.3 [P12].

Continued dominance of the GII.4 Sydney 2012 [P16] strain illustrates its highly virulent nature. Childhood strains including GII.6 [P7] and GII.3 [P12] were also more prevalent indicating a shift towards childhood strain dominance in the second half of 2025, indicating a shift in herd immunity.

POSTER SESSION: SCIENTIFIC ABSTRACT #36

The RNA Helicase Rig-I in Biomolecular Condensates

Xinyu (Yola) Li, Samantha J. Owens, and Cecile King

1. The School of Biotechnology and Biomolecular Sciences, Faculty of Science, UNSW Sydney, Sydney, New South Wales, Australia

Retnoic acid-inducible gene-I (RIG-I) is a pa6ern recogni5on receptor (PRR) that can recognise RNA viruses in the cytosol. Following the recogni5on of viral RNA, RIG-I interacts with mitochondrial antiviral-signalling protein (MAVS), located on the surface of mitochondria, to initcate signalling that leads to the production of type 1 interferon and proinflammatory cytokines. Recently, RIG-I foci have been observed in the cytoplasm long after (1-3 days) RNA virus infection, suggesting that RIG-I has functions beyond early virus sensing.

To investigate the role of RIG-I beyond the initial recognition of viral RNA, murine macrophages were stimulated with non-specific ligands for RIG-I that stimulate an immune response (lipopolysaccharide (LPS) or Polyinosinic:polycytdylic acid (poly I:C)). We observed that RIG-I-containing condensates formed in the cytoplasm, which were dynamic and diverse in their components. Approximately half of these RIG-I-containing condensates were Ras-GTPase-activating protein SH3 domain binding protein (G3BP)-containing stress granules. Importantly, we demonstrate that RIG-I has a critical role in the formation of these biomolecular condensates as immune cells deficient in RIG-I failed to form G3BP1+ stress granules. These results suggest that RIG-I contributes to the formation or maintenance of biomolecular condensates in response to inflammation. These findings indicate that during inflammation, RIG-I binds endogenous RNA to initiate the formation of biomolecular condensates.

POSTER SESSION: SCIENTIFIC ABSTRACT #37

Small Interfering RNA for the treatment of rhinovirus infections

Ethan McDaid[1, 2], Nathan Bryant[1, 2], Jason Girkin[1, 2], Kai Jarvis[1, 2], Camille Esneau[1, 2], Su-Ling Loo[1, 2], Nathan Bartlett[1, 2]

1. Viral Immunology and Respiratory Disease Group, School of Biomedical Sciences and Pharmacy, College of Health, Medicine and Wellbeing, University of Newcastle, Newcastle, NSW, Australia
2. Hunter Medical Research Institute, New Lambton Heights, NSW, Australia

Keywords: siRNA**,** RNA, rhinovirus, antiviral, respiratory

Introduction
Rhinovirus (RV) induces a profound socioeconomic burden on society and poses a serious threat to vulnerable populations including children, people with chronic respiratory diseases and the immunocompromised. However, due to the major genetic heterogeneity across ~180 subtypes, vaccination efforts have failed to generate effective cross-serotype immunity, and antiviral therapies have largely been unsuccessful in reducing viral load and symptoms. We tested the hypothesis that RNA interference, harnessing endogenous cellular machinery to deliver small interfering RNAs (siRNAs) targeting conserved regions of the RV genome, would provide broad-spectrum antiviral protection.

Objective
To establish proof-of-concept evidence that prophylactic administration of siRNA can reduce viral load across multiple RV subtypes *in vitro*.

Methods and Results
RV infections were studied in rhabdomyosarcoma (RD) cells engineered to overexpress ICAM-1, generating a highly permissive cell line expressing receptors for multiple RV strains. RD-ICAM1 cells were transfected 24 hours prior to infection with different siRNAs (25nM in 500µl) targeting a conserved region of the 5'-UTR in the RV genome. Cells were infected with RV-A1, RV-A16, RV-A43, or RV-A49 at a multiplicity of infection of 0.5. Cells were harvested 24 hours post-infection and viral load was assessed by qPCR. A statistically significant reduction in viral RNA was achieved across the four subtypes, up to 72% in minor group RV with a reduction in cytopathic effect. siRNA transfection was not cytotoxic.

Conclusions
Prophylactic targeting of conserved 5'UTR regions with siRNA effectively reduced viral RNA *in vitro* across multiple RV subtypes from both the major and minor groups. We also demonstrated tolerance to a single nucleotide mismatch within the seed region of a siRNA sequence, indicating its potential for broader cross-reactivity and its tolerance of viral escape which are primary concerns for previous antivirals. Overall, this work established that siRNAs can target multiple RV subtypes, supporting further development of siRNAs as broad-spectrum therapeutics against RV.

POSTER SESSION: SCIENTIFIC ABSTRACT #38

Enhancing the efficacy of and overcoming resistance to Kras Inhibitors in Pancreatic Cancer

Niamh EP Mills[1], Bronte B Green, Alice MH Tran[1], Victoria Lee[1], Anna E Howell[1], Terrie-Anne Cock[2], Chris Burns[2], Anaiis Zaratzian[1], Michael Tayao[1], Andrew Da Silva[1], Australian Pancreatic Genome Initiative (APGI), Australian Pancreatic Matrix Atlas (APMA), David Herrmann[1,3], Thomas R Cox[1,3], Marina Pajic[1,3], Paul Timpson[1,3] and Kendelle Murphy[1,3]‡

1. Garvan Institute of Medical Research and The Kinghorn Cancer Centre, Tumour Ecosystems Program, Sydney, NSW 2010, Australia.
2. Amplia Therapeutics Limited, Melbourne, Australia.
3. St. Vincent's Clinical School, Faculty of Medicine, UNSW Sydney, Sydney, NSW 2010, Australia.

Key Words: Pancreatic ductal adenocarcinoma. KRAS inhibition. Focal Adhesion Kinase. Extracellular tumour microenvironment. Combination therapy.

Introduction: Pancreatic cancer (PC) is a genetically complex and treatment-refractory malignancy. PC is predominantly driven by mutations in the Kras oncogene (90%) which allow cancer cells to exhibit immortal proliferative potential. Whilst PC has shown resistance to standard-of-care chemotherapy, new approaches to target mutated Kras show promise. However, pre-clinical investigations have revealed that Kras inhibition leads to enhanced extracellular matrix (ECM) deposition, which drives resistance and restricts treatment efficacy. We have identified Focal Adhesion Kinase (FAK) as a critical regulator of matrix organisation and thus as an important contributor to PC progression.

Objective: Thus, the aim of this study is to determine whether anti-fibrotic FAK targeting can improve the delivery and longevity of Kras Inhibitors in PC.

Methods and Results: We assessed FAK/KRAS inhibitor synergy against tumour cells in a three-dimensional cell assay and used organotypic matrices to optimise drug timing. A subcutaneous syngeneic PDAC mouse model was used to determine how KRAS inhibition influences the ECM by quantifying the expression of picrosirius red, alpha-smooth muscle actin, and FAK activity. To determine whether in vitro synergy translates into in vivo, we tested FAK/KRAS combination in a PDAC mouse model, by assessing tumour growth and quantifying expression of ERK activity (downstream effector of Kras signalling), Ki67 (proliferation), cleaved caspase-3 (apoptosis). PDAC mouse models demonstrated that our KRAS inhibitors have efficacy, increase fibrosis, and activate fibroblasts. Synergy response was heterogeneous across a panel of patient cell lines, suggesting context-specific determinant of combination response. Priming the ECM with a FAK-inhibitor reduced cancer cell invasion and rendered them vulnerable to subsequent KRAS inhibition in vitro.

Conclusions: Combined, this suggests that subtle stromal manipulation via FAK inhibition may allow us to overcome resistance mechanisms to KRAS inhibition.

POSTER SESSION: SCIENTIFIC ABSTRACT #39

Investigating loss of the tumour suppressor gene PTEN as a potential genetic driver of malignant transformation in endometriosis

Anaida Nirula[1], Kristie-Ann Dickson[1], Teagan Fisher[1], Maree Bielski[1], Amani Alghalayini[1] and Deborah J Marsh[1]

1. Translational Oncology Group, Faculty of Science, School of Life Sciences, University of Technology Sydney, Ultimo, NSW 2007, Australia.

Keywords: Endometriosis. PTEN. Ovarian Cancer. CRISPR. AKT

Introduction: Endometriosis is a chronic inflammatory estrogen-dependent condition characterised by the growth of endometrial-like tissue outside of the uterus. People with endometriosis have a ≥ 2-fold increased risk of developing epithelial ovarian cancer compared to those without, most notably ovarian clear cell carcinoma and endometrioid ovarian cancer. Genetic alterations in cancer-associated genes, including *PTEN* which encodes a tumour suppressor protein, have been identified in endometriotic lesions; however, a possible functional role in malignant transformation remains unclear.

Objective: To determine whether CRISPR-engineered loss of PTEN in the 12Z epithelial cell line model of endometriosis leads to malignant-like behaviour *in vitro*.

Methods and Results: *PTEN* was investigated in epithelial (12Z and iEEC16) and stromal (1458 and T-HESC) endometriosis-related cell lines. Sanger sequencing confirmed the wild-type (WT) status of *PTEN* in both 12Z and iEEC16 cells. Using a TaqMan copy-number analysis, loss of one copy of PTEN was observed in both stromal cell lines. CRISPR-Cas9 gene editing was used to generate PTEN knockout (KO) clones in 12Z cells, with single guide RNAs (sgRNAs) targeting exons 5 or 6. Complete loss of PTEN was confirmed by western blotting. Growth rate and clonogenic cell survival assays of exon 5 targeted PTEN KO (PTENe5KO) cells showed a significant increase in proliferation and cell survival compared to 12Z clonal cells. All 12Z PTEN engineered KO clones displayed elevated phosphorylated AKT indicative of activation of pro-survival signalling; however, increased phosphorylation of the downstream target mTOR was not observed. PTENe5KO clones showed greater sensitivity to the AKT inhibitor Afuresertib in MTS cell viability assays.

Conclusions: This study provides evidence that loss of PTEN in 12Z cells correlates with malignant-like behaviour *in vitro*. Response to the AKT inhibitor Afuresertib in PTEN KO cells suggests that activated AKT may be a therapeutic vulnerability in epithelial endometriosis cells with loss of PTEN.

POSTER SESSION: SCIENTIFIC ABSTRACT #40

The Function of Retrotransposon Lx9c11 in the Immune System

Finn O'Loan[1], Cecile King[1]

1. School of Biotechnology & Biomolecular Sciences, UNSW, Sydney, NSW, Australia

Keywords: Immunology, Transposons, Genetics

Introduction: Lx9c11 is a murine LINE-1 retrotransposon critical for the expression of full-length long non-coding RNA, Lx9c11-RegoS. Lx9c11-/- mice feature an exaggerated and lethal immune response to viral infection. This study investigates how Lx9c11 regulates immune cell populations and T cell function during antiviral immune responses.

Objective: Identify the mechanism behind Lx9c11 immune regulation.

Methods and Results: Wild-type and Lx9c11-/- mice were challenged with Coxsackievirus B4 or the viral mimic poly(I:C). Immune cells were analysed by flow cytometry, single-cell RNA sequencing, proliferation assays and RT-qPCR. Lx9c11-RegoS expression was restricted to neutrophils and T cells. Emergency granulopoiesis-associated neutrophils contained the highest proportion of Lx9c11-RegoS-positive cells. The dendritic cell population was reduced by approximately 50% in Lx9c11-/- mice compared with wild-type mice 24 hours after poly(I:C) injection. Lx9c11-/- T cells exhibited increased proliferation.

Conclusion: These findings identify neutrophils and T cells as the primary immune cell types expressing Lx9c11-RegoS and suggest that Lx9c11 contributes to antiviral immune regulation through mechanisms involving neutrophils and T-cells.

POSTER SESSION: SCIENTIFIC ABSTRACT #41

The Vaginal Microbiome, Cervical Length Shortening and Preterm birth risk: A Systematic Review

Jessica Sydney Ramos[1,2], Yafeng Ma[1,2], Therese Becker[1,3,4], Varsha Venkat[2,5], Jon Hyett[2,4,6]

1. School of Clinical Medicine, University of New South Wales, Sydney, NSW, Australia.
2. South Western Sydney Obstetrics Research Unit, Ingham Institute for Applied Medical Research, Liverpool, NSW, Australia
3. Medical Oncology, Ingham Institute for Applied Medical Research, Liverpool, NSW, Australia
4. School of Medicine, Western Sydney University, Sydney, NSW, Australia
5. School of Biomedical Sciences, University of New South Wales, Sydney, NSW, Australia.
6. Fetal-Maternal Unit, Liverpool Hospital, Liverpool, NSW, Australia

Keywords: Maternal microbiome. Cervical length shortening. Preterm birth. 16S Sequencing. Shotgun metagenomics.

Introduction: Preterm birth (PTB) is the leading cause of death in children under the age of five globally. Ultrasound assessment of cervical length (CL) is used to predict risk of PTB at 20 weeks' gestation but performs poorly in the first trimester when alternative strategies for risk mitigation could be applied. Recent investigations in the vaginal microbiome (VMB) have shown potential links between an unbalanced microbial profile and PTB risk.

Objective: The aim is to systematically assess the prognostic value of CL paired with VMB to improve the prediction of PTB.

Methods and Results: A comprehensive search was conducted on PubMed, Cochrane Library, Embase (OVID), Scopus, Web of Science and ProQuest. The keyword search strategy included terms relating to 'prematurity', 'short cervix', 'vaginal microbiota' and '16S sequencing and/or shotgun metagenomic sequencing'. Eligible studies included non-randomised and randomised control trials with inclusion criteria used during two screening stages. A total of 19 studies were included for data extraction relating to interventions, immunological factors, CLs, and metagenomics data. The emerging data supports the association of a short CL and a non-*Lactobacillus* dominated and/or highly diverse vaginal microbiota with high PTB risk. A quality assessment was performed using the Newcastle-Ottawa Scale (NOS) and data analysis is ongoing.

Conclusions: This systematic review identifies an association between CL and VMB in relation to PTB prediction. Current PTB prediction models and screening efficacy may be improved by reporting VMB in relation to CL.

POSTER SESSION: SCIENTIFIC ABSTRACT #42

PocketPCR: Portable PCR with Integrated Fluorescence Detection for viral Screening

Nima Reji[1] and Nham Tran[1]

1. *Discipline of Biomedical Engineering, University of Technology Sydney, Sydney, NSW, Australia.*

Keywords: PocketPCR, point-of-care diagnostics, HPV-16, fluorescence detection, thermal cycling.

Introduction: Polymerase chain reaction (PCR) is widely used for sensitive and specific detection of viral pathogens through amplification of viral nucleic acids. However, conventional PCR systems are often expensive and reliant on specialised laboratory infrastructure, limiting their use in decentralised and point-of-care settings. The open-source PocketPCR offers a low-cost alternative using readily available electronic components and additive manufacturing, with potential applications in field studies, low- and middle-income countries (LMICs), and rural or remote communities.

Objective: This project aimed to design an affordable, portable PCR platform for viral detection using readily available components, additive manufacturing and integrated fluorescence detection.

Methods and Results: An engineering design process incorporating CAD modelling (Fusion 360), additive manufacturing, rapid prototyping and bench testing was used to develop the platform. Thermal performance was improved using an 11,000 RPM high-static-pressure fan, redesigned airflow pathways with honeycomb vents and an elevated base, and a cylindrical twist-lock enclosure around the heating block. Fluorescence detection was integrated using five board-powered blue LEDs, excitation and emission filters, and a sliding viewing cover. Using an identical 40-cycle HPV-16 PCR program, runtime was reduced from 2 h 15 min to 1 h 38 min, with thermal transitions approximately 13 seconds faster per phase. The system clearly distinguished positive and negative controls, while external surfaces remained safe to touch. Fabrication cost was approximately AUD $240.

Conclusions: The redesigned PocketPCR provides a low-cost, portable and self-contained platform for viral detection, with potential applications in field studies, LMICs, and rural and remote settings with limited laboratory infrastructure.

POSTER SESSION: SCIENTIFIC ABSTRACT #43

Medication-related problems and pharmacist recommendations for cardiometabolic medicines in Arabic-speaking patients: A retrospective analysis of home medicines reviews in Australia

Rawan Sawalha[1], Neil J. Spratt[1,2,3], Hassan Hossienzadeh[5], Christopher Levi[2,3,4], and Beata Bajorek[1,2,3].

[1] School of Biomedical Sciences and Pharmacy, University of Newcastle, Callaghan, New South Wales, Australia.
[2] Brain Health Program, Hunter Medical Research Institute, New Lambton Heights, New South Wales, Australia
[3] Hunter New England Local Health District, Newcastle, New South Wales, Australia
[4] School of Medicine and Public Health, University of Newcastle, Callaghan, New South Wales, Australia
[5] School of Medical, Indigenous and Health Sciences, Faculty of Science, Medicine and Health, University of Wollongong, Wollongong, New South Wales, Australia

Medication-related problems (MRPs) are common among patients with cardiovascular disease (CVD) or diabetes and may be further exacerbated in culturally and linguistically diverse populations due to language barriers, health literacy challenges, and culturally influenced beliefs about medicines. However, limited evidence exists regarding MRPs identified through Home Medicines Reviews (HMRs) among Arabic-speaking patients in Australia.

This retrospective study analysed 164 de-identified HMR reports collected between 2022 and 2025 for Arabic-speaking adults living with CVD or diabetes. MRPs and pharmacist recommendations were classified using the validated DOCUMENT framework.

A total of 1,345 MRPs were identified (mean 8.2 ± 3.7 per patient), with MRPs identified in all participants. The most common MRP categories were drug-selection problems (26.8%), monitoring needs (21.2%), and treatment-need problems (16.8%). Cardiometabolic medicines accounted for over half of all MRPs (51.3%) and were implicated in at least one MRP in 95.1% of patients. Drug interactions and monitoring requirements were the most frequently identified issues. Pharmacists commonly recommended monitoring, therapy adjustment, and patient education or counselling.

These findings highlight the high burden of MRPs among Arabic-speaking patients receiving HMR services and demonstrate the important role of pharmacists in identifying and addressing medication-related risks. The study also emphasises the need for culturally responsive medication management strategies and targeted referral pathways to improve medication safety in diverse populations.

POSTER SESSION: SCIENTIFIC ABSTRACT #44

Single-cell and spatial transcriptomic profiling identifies Cancer-associated fibroblast heterogeneity and *MYCN*-associated stromal remodelling in neuroblastoma

Mekonnen Sisay Shiferaw[1], Klaartje Somers[2,3] and Zaklina Kovacevic[1]

1. School of Biomedical Sciences, Faculty of Medicine and Health, University of New South Wales, Sydney, Australia
2. Children's Cancer Institute at Minderoo Children's Comprehensive Cancer Centre, Sydney, NSW, Australia
3. School of Clinical Medicine, University of New South Wales, Sydney, NSW, Australia

Keywords: Neuroblastoma, Tumour microenvironment, CAFs, *MYCN*

Introduction: Despite multimodal therapy, 5-year survival in high-risk neuroblastoma (NB) remains below 50%. *MYCN* amplification drives aggressive tumour biology in a substantial proportion of high-risk NB, yet remains difficult to target directly. Hence, its downstream effects on tumour metabolism and the tumour microenvironment are increasingly recognised as key drivers of progression. Among stromal components, CAFs are emerging as critical regulators of extracellular matrix remodelling, immune organisation, and treatment response.

Objective: This study aimed to characterise CAF heterogeneity, spatial architecture, *MYCN*-associated transcriptional programs, and their relationships with immune profiles.

Methods and Results: scRNA-seq data from 62 NB samples were integrated after stringent quality control. Cell identities were annotated using canonical markers and NBAtlas, followed by targeted downstream scRNA-seq analyses. Visium FFPE spatial transcriptomics was used to define CAF localisation, stromal–immune architecture, and *MYCN*-associated spatial features. Major NB-TME populations comprised neuroblasts, immune cells, and CAFs. Main CAF subtypes include inflammatory (iCAFs), matrix (mCAFs), and myofibroblastic states (myCAFs). Non-*MYCN*-amplified tumours were enriched for iCAF programs, whereas *MYCN*-amplified tumours showed enrichment of myCAF states, reduced immune infiltration, higher immunosuppressive scores, and altered stromal-immune communication. iCAFs were more strongly associated with inflammatory signatures, while mCAFs aligned more closely with broader immunosuppressive programs. COMPASS further supported distinct metabolic features of CAF types between *MYCN*-amplified and non-amplified tumours. Spatial transcriptomics also revealed marked *MYCN*-associated stromal reorganisation, with diffuse stromal infiltration and depleted immune components in *MYCN*-amplified tumours.

Conclusions: Being involved in mediating the immune and metabolic landscapes of NB, this study reveals that CAFs play a crucial role in shaping the NB TME. Understanding CAF heterogeneity and microenvironmental effects of *MYCN* amplification will inform how NB responds to treatment and lead to better patient outcomes. Future studies should prioritise functional validation of CAF states to identify targetable microenvironmental dependencies that may improve patient outcomes.

POSTER SESSION: SCIENTIFIC ABSTRACT #45

Clinical outcomes of children and adolescents with type 1 diabetes mellitus in Ethiopia: evidence on glycaemic achievement and diabetic ketoacidosis

Chalie Marew Tiruneh[1,4], Muhammad Chutiyami[1] (Lecturer), Marilyn Cruickshank[1,2] (Professor), Shannon Lin[3] (Associate Professor), Sewagegn Yeshiwas[5] (Lecturer), Bereket Fantahun[6] (Associate Professor), Lin Perry[1] (Professor)

[1]School of Nursing and Midwifery, Faculty of Health, University of Technology Sydney, Ultimo, New South Wales, Australia
[2]Sydney Children's Hospitals Network, Sydney, Australia
[3]School of Public Health, Faculty of Health, University of Technology Sydney, Ultimo, New South Wales, Australia
[4]Department of Pediatrics and Child Health Nursing, College of Health Sciences, Debre Tabor University, Amhara, Ethiopia
[5]Department of Pediatrics and Child Health, College of Health Science, Addis Ababa University, Addis Ababa, Ethiopia
[6]Department of Pediatrics and Child Health, St Paul Hospital Millennium Medical College, Addis Ababa, Ethiopia.

Keywords: Type 1 diabetes, Children and adolescents, glycaemic achievement, diabetes ketoacidosis. Ethiopia.

Introduction: Type 1 diabetes poses significant challenges for children and adolescents, particularly in resource-limited settings such as Ethiopia. Clinical outcomes are key indicators of the effectiveness of diabetes management, but evidence in these settings is scarce, hindering the development of targeted interventions and policies to improve health outcomes.

Objective: To determine the incidence of diabetic ketoacidosis, rates of achievement of recommended glycaemic levels and factors that predict this.

Methods and Results: A multicentre study combining secondary document analysis and surveys was conducted, recruiting 411 children and adolescents with type 1 diabetes from diabetes care clinics of five hospitals in Ethiopia. Glycaemic status was assessed using internationally recommended targets: for HbA1c (<7%), fasting and random blood glucose ranges (80–144 mg/dL; 140–180 mg/dL, respectively). Factors potentially predictive of outcomes were identified using multivariable logistic regression, with significance set at $p <= 0.05$.

Of 411 participants (51.3% female; mean age 10.9±4.7 years), 405 had HbA1c records. Only 54 (13.3%) met HbA1c target values, and the sample mean was 9.56±2.25%. Fasting and random glucose targets were met in 42.5% and 38.3% of participants, respectively. Hypoglycaemia was common (29.9%). Better glycaemic status was associated with male sex, shorter diabetes duration, no recent hospitalisation, diabetes education attendance, and better HRQoL. Of 9492 person-months of follow-up, 19.7% experienced at least one episode of diabetic ketoacidosis, with an incidence of 8.53 per 1000 person-months.

Conclusions: Low achievement of glycaemic targets and high incidence of diabetic ketoacidosis was revealed in this resource-limited setting, even amongst these clinic attendees. Context-specific evidence is essential to inform targeted interventions to improve outcomes for young people with type 1 diabetes, and study data flag the crucial need to strengthen diabetes education, improve treatment adherence, expand access to care, and integrate psychosocial services into routine clinical management.

POSTER SESSION: SCIENTIFIC ABSTRACT #46

Therapeutic Potential of Adipose-Derived Mesenchymal Stem Cells in Cisplatin-Induced Acute Kidney Injury

Thi Kim Chi Tran[1], Henry H.L. Wu[1,2], Long The Nguyen[1], Carol A. Pollock[1,2], Sonia Saad[1]

1. Renal Research Laboratory, Kolling Institute of Medical Research, Royal North Shore Hospital & The University of Sydney, Sydney, NSW, Australia
2. Department of Renal Medicine, Royal North Shore Hospital, Northern Sydney Local Health District, Sydney, NSW, Australia

Keywords: Mesenchymal Stem Cells, Extracellular Vesicles, Acute Kidney Injury, Mitochondria, Tunnelling nanotubes.

Introduction: Acute kidney injury (AKI) affects an estimated 13.3 million individuals annually, contributing to 1.7 million deaths worldwide, with progression to chronic kidney disease and elevated cardiovascular risks. Cisplatin-induced nephrotoxicity causes AKI in up to 30% of oncology patients, yet no approved pharmacotherapies exist. AKI pathophysiology is driven by proximal tubular cell (PTC) mitochondrial dysfunction, apoptosis, and inflammation. Adipose-derived mesenchymal stem cells (ADMSCs) offer paracrine immunomodulatory and regenerative effects, and may donate mitochondria to injured cells via tunnelling nanotubes (TNTs).

Objective: This study aims to investigate whether ADMSCs and their extracellular vesicles (EVs) ameliorate cisplatin-induced AKI and whether TNT-mediated mitochondrial transfer constitutes a cytoprotective mechanism.

Methods and Results: PTCs were exposed to 300 μM cisplatin (24h), then treated with ADMSC-conditioned medium (CM) or EVs. In vivo, AKI was induced in Sprague–Dawley rats with intraperitoneal cisplatin (5 mg/kg), followed by autologous ADMSC (5×10^6 cells) or EV (100 μg) administration. Injury markers (KIM-1, NGAL, MCP-1, IL-6, Caspase-3, TGF-β, fibronectin) were quantified by qPCR; KIM-1 protein by ELISA; and histopathology by H&E scoring. Mitochondrial transfer was visualised by co-culturing MitoTracker Deep Red labelled ADMSCs with injured PTCs via high-content confocal microscopy.

In vitro, CM treatment significantly attenuated all injury markers; EV treatment selectively reduced KIM-1 and MCP-1. Confocal imaging confirmed TNT formation with MitoTracker signal detected within injured PTCs, demonstrating direct mitochondrial transfer from ADMSCs. In vivo, ADMSC administration significantly reduced injury marker expression, KIM-1 ELISA levels, and histopathological scores. EV treatment produced consistent downward trends, with a notable reduction in KIM-1 protein despite limited sample size.

Conclusions: ADMSCs and their derived EVs attenuate cisplatin-induced AKI in vitro and in vivo. TNT-mediated mitochondrial transfer from ADMSCs to injured PTCs represents a novel cytoprotective mechanism warranting further investigation.

POSTER SESSION: SCIENTIFIC ABSTRACT #47

Co-targeting thrombospondin-1 (THBS-1) to improve KRAS inhibitor efficacy in pancreatic cancer

Victoria Tyma[1,2], Katie Gordon[1,2], Shona Ritchie[1,2], Kendelle Murphy[1,2], Australian Pancreatic Genome Initiative (APGI), Australian Pancreatic Cancer Matrix Atlas (APMA), David Herrmann[1,2], Brooke Pereira[1,2] & Paul Timpson[1,2]

1. Garvan Institute of Medical Research, Sydney, Australia.
2. 2. St. Vincent's Clinical School, University of New South Wales (UNSW), Sydney, Australia.

Introduction: Pancreatic cancer (PC) is one of the deadliest cancers, the fourth leading cause of cancer related deaths in Australia1. Most PC harbours a mutation in the KRAS gene, which is historically challenging to target. However, in the past few years, there has been a major medical advancement in developing highly effective and specific KRAS inhibitors. Mirati's G12D KRAS inhibitor, MRTX1133, can cause a >50% decrease in mouse PC tumour burden2. Pan-KRAS inhibitors act on all KRAS mutations, including RMC-6236 (Revolution Medicines) and BI-2493 (Boehringer Ingelheim)3,4. Despite these exciting results, it is likely tumours will become resistant to KRAS inhibition, partly due to changes in pancreatic tumour microenvironment, including tumour fibrosis and vasculature changes5, 6, 7.

Methods and Results: We aim to explore co-targeting the tumour microenvironment to improve KRAS inhibitor response. From mass spectrometry proteomics data in our lab, we have identified high levels of thrombospondin 1 (THBS-1) in aggressive PC models8, presenting a unique stromal co-target that may overcome challenges with resistance. Preliminary data shows THBS-1 is largely produced by cancer-associated fibroblasts (CAFs), which are responsible for the majority of tumour fibrosis in PC9, 10. We aim to analyse the effect of genetic and pharmacological inhibition of THBS-1 in combination with KRAS inhibition, examining whether we can improve drug response using this dual targeting approach. Ongoing work includes genetic knockdown of THBS-1 in CAFs derived from the genetically engineered KPC mouse model of PC11. With access to LSKL, a potent THBS-1 inhibitor 12, we will assess the functional role of THBS-1 in context of CAF contractility and cancer cell invasion using 3D organotypic matrix contraction and invasion assays13,14, in combination with KRAS inhibitors.

Conclusions: Future work will include trialling our novel combination therapy via subcutaneous and orthotopic (intra-pancreatic) in vivo models. Overall, we aim to assess THBS-1 as a potential co-target to improve the efficacy and durability of these promising KRAS inhibitors.

POSTER SESSION: SCIENTIFIC ABSTRACT #48

Changes in sleep and clinical outcomes in hip osteoarthritis: A longitudinal secondary analysis of a randomised controlled trial

Hanzhi Zhang[1,2] · Fiona Dobson[3] · Kim Allison[4] · Gabrielle Knox[4] · Libby Spiers[4] · Natalia E. Brumley[5] · David Klyne[6] · Paulo Ferreira[2,1] · Michelle Hall[1,2]

1 The University of Sydney School of Health Sciences
2 The University of Sydney Charles Perkins Centre
3 The University of Melbourne Department of Physiotherapy
4 The University of Melbourne Centre for Health Exercise and Sports Medicine
5 Melbourne School of Psychological Sciences
6 The University of Queensland School of Health and Rehabilitation Sciences

Keywords: Pain, Function, Exercise, Sleep

Introduction: Poor sleep, such as difficulty falling asleep and/or staying asleep is one of the most distressful symptoms of hip osteoarthritis (OA). Pain is often considered a major contributor to poor sleep and yet poor sleep is associated with presence and severity of chronic musculoskeletal pain, including in hip OA. Sleep management is not routinely provided in routine care of hip OA. Understanding the association between improved sleep and clinical outcomes (e.g., pain and physical function) could assist to reframe poor sleep as not only a potential symptom of, but a potential contributor to, hip OA symptoms that warrants consideration as a treatment target.

Objective: To investigate whether improvements in sleep quality and duration are associated with improvements pain and physical function in people with hip OA following exercise.

Methods and Results: Data from 185 participants with hip OA, randomized to an exercise intervention were pooled and analysed. Independent variables were 3-month change in sleep quality using the Pittsburgh Sleep Quality Index (PSQI; range 0–21, higher scores indicating poorer sleep) and sleep duration (hours). Outcomes were 3-month change in hip pain severity (11-point scale; range 0-10, with higher scores indicating worse pain) and physical function (Western Ontario and McMaster Universities Osteoarthritis Index; range 0-68, with higher scores indicating more dysfunction). Associations between change in sleep quality and duration and change in outcomes were examined using linear regression models, adjusted for baseline values and covariates and according to baseline sleep quality status (PSQI >5 = poor sleep; ≤5 good sleep). In adjusted models, each 1-point improvement in sleep quality was associated with reduced pain (-0.21 units 95% CI: -0.32 to -0.09) and improved physical function (-1.45 units 95% CI: -2.01 to -0.89). Each 1-hour increase in sleep duration was associated with reduced pain (-0.34 units (95% CI: -0.69 to 0.02) and improved physical function (-3.23 units (95% CI –4.97 to -1.49). There were no statistically significant interactions between baseline sleep quality status (p=0.055 to 0.994) but associations were more pronounced for those with poor sleep.

Conclusions: Improvements in sleep quality and duration are associated with small improvements in pain and physical function following exercise. These relationships were consistent over time but of uncertain clinical relevance.

POSTER SESSION: SCIENTIFIC ABSTRACT #49

Novel β-lactam Combination Therapy with Iminosugar AMP-DNJ to Treat Methicillin Resistant *Staphylococcus aureus*

Wentao Xu[1], Abbey Stewart[1], Farah Lamiable-Oulaidi[2], Ashleigh S Paparella[1]

1. University of Sydney, School of Medical Science, Camperdown, Australia
2. The Ferrier Research Institute, Victoria University of Wellington, Lower Hutt, New Zealand

Keywords: enzyme inhibitors, *Staphylococcus aureus*, antibiotics

Introduction: Methicillin Resistant *Staphylococcus aureus* (MRSA) is a significant and growing threat to human health, responsible for >130,000 attributable deaths in 2021. MRSA's resistance to β-lactam antibiotics operates by targeting penicillin binding proteins (PBP) to eliminate the pathogen is driven by the presence of PBP2a, which has reduced binding affinity to β-lactam that enables it to continue the cross-linking of the cell wall. PBPs, including PBP2a rely on wall teichoic acid (WTA) to regulate their localization and enzyme activity. WTA is synthesised by Tar glycosyltransferases, including TarS and TarM, which are responsible for the glycosylation of WTA and therefore possible targets for MRSA β-lactam combination therapy.

Objective:
1. To discover small molecule inhibitors of TarS and TarM, and determine the binding mechanism.
2. To determine whether iminosugar AMP-DNJ is synergistic with β-lactams.

Methods and Results: To determine potential small molecule inhibitors, a HPLC-based assay was used to test for enzyme activity of TarS and TarM with 23 iminosugars. AMP-DNJ inhibited TarM with an IC_{50} of 1.09 ± 564.40 mM and TarS with an IC_{50} of 728.70 ± 84.13 μM. The binding mechanism of AMP-DNJ with respect to UDP-GlcNAc will be determined from Lineweaver–Burk plots of initial-rate data. The synergism of iminosugar AMP-DNJ with β-lactams was conducted through checkerboard assays with *S. aureus* strand RN4220. The FICI value of ampicillin with DNJ was at 0.53, and AMP-DNJ at 1.06.

Conclusions: The identification of novel small molecules to inhibit the synthesis of WTA may potentiate β-lactams activities against MRSA, the leading cause of Gram-positive antimicrobial resistant deaths. AMP-DNJ is an inhibitor of TarM and TarS and is predicted to be competitive with respect to UDP-GlcNAc. Checkerboard assays were also conducted to determine the FICI value, measuring the synergism of AMP-DNJ and DNJ with β-lactams.

POSTER SESSION: SCIENTIFIC ABSTRACT #50

cGAS Engages Nutrient-Sensing Enzymes To Promote Tumorigenesis

Shreya Mahajan[1], Abhimanu Pandey[1] and Si Ming Man[1*]

[1] *Division of Immunology and Infectious Diseases, The John Curtin School of Medical Research, The Australian National University, Canberra, 2601, Australia.*

Keywords: DNA sensors, cGAS, colorectal cancer, GFPT1, GFPT2

Introduction: Colorectal (also called bowel) cancer is the second leading cause of cancer-related death in Australia and worldwide. DNA liberated from microbes or damaged host cells is detected by innate immune DNA sensors in the gut and can promote inflammation and cancer.

Objective: Here, we investigate the role of cytosolic DNA sensor cGAS, in mice carrying a heterozygous mutation in the adenomatous polyposis coli gene (*Apc*$^{Min/+}$) that mimic human colorectal cancer driven by APC mutants.

Methods and Results: Tumour burden was analysed in 20-week-old littermate *Apc*$^{Min/+}$*cGas*$^{-/-}$ and *Apc*$^{Min/+}$ mice. Immunoblotting, immunohistochemistry and multiplex ELISA assessed mechanisms driving proliferation and inflammatory signalling. cGAS-associated proteins were identified by co-immunoprecipitation followed by mass spectrometry and validated using biochemical and functional assays. We also evaluated the therapeutic potential of cGAS inhibition in human colorectal cancer cell lines and mouse and patient-derived intestinal organoids. We observed *Apc*$^{Min/+}$*cGas*$^{-/-}$ mice had reduced tumour burden, reduced histopathological damage and markedly fewer proliferating intestinal epithelial cells than *Apc*$^{Min/+}$ littermates. Unexpectedly, these tumour-promoting effects of cGAS were independent of its canonical inflammatory and type I interferon signalling functions. Instead, cGAS interacts with the nutrient-sensing enzymes GFPT1 and GFPT2, forming a previously unrecognised tripartite complex. cGAS-GFPT1-GFPT2 complex drives OGT-mediated O-GlcNAcylation of NF-kB (p65), resulting in β-catenin-mediated activation of cell cycle proteins cMyc and Cyclin E, thereby increasing cell proliferation and tumorigenesis. We identified that availability of glucose and glutamine is essential for the formation of cGAS-GFPT1-GFPT2 complex. Pharmacological inhibition of this signalling axis using the cGAS inhibitor RU.521 and/or GFPT1/2 inhibitor DON substantially reduced proliferation of HT-29 cells and restricted growth of mouse and human intestinal organoids.

Conclusions: Together, these findings suggest that cGAS, GFPT1 and GFPT2 could serve as biomarkers and the therapeutic targeting of the cGAS-GFPT1-GFPT2 signalling axis could improve health outcomes in patients with colorectal cancer.

POSTER SESSION: SCIENTIFIC ABSTRACT #51

Combined DNA-PK and BCL-2 Inhibition Overcomes Resistance to venetoclax in AML

Maddison Chambers[1,2], Kasey Miller[1,2], Jonathan Sillar[1,2,3], Joshua S. Brzozowski[1,2], Madeleine Walton-Smith[1,2], Donia Moujalled[4], Anoop Enjeti[1,3], Nicole M. Verrills[1,2], Heather C. Murray[1,2]

1. School of Biomedical Sciences and Pharmacy, College of Health, Medicine and Wellbeing, University of Newcastle, Callaghan, NSW, Australia
2. Precision Medicine Research Program, Hunter Medical Research Institute, New Lambton Heights, NSW, Australia
3. Division of Molecular Medicine, Pathology North, John Hunter Hospital, New Lambton Heights, NSW, Australia
4. Walter and Eliza Hall Institute of Medical Research, Melbourne, VIC, Australia

Keywords: Acute myeloid leukaemia, venetoclax resistance, DNA damage response, targeted therapy

Introduction: Acute myeloid leukaemia (AML) is an aggressive haematological malignancy with poor survival outcomes, particularly in older or medically unfit patients. Although venetoclax, a selective BCL-2 inhibitor, has transformed treatment for this high-risk population, intrinsic and acquired resistance develops in approximately 60% of patients, representing a major barrier to durable clinical response.

Objective: Our lab previously identified upregulation of DNA-PK–dependent DNA repair in kinase-mutant AML, including FLT3-, KRAS-, and NRAS-mutant subtypes that respond poorly to venetoclax. We hypothesised that inhibition of DNA-PK-mediated DNA repair would overcomes resistance and sensitise venetoclax-resistant AML cells to venetoclax.

Methods and Results: Proliferation and apoptosis assays were performed across a panel of human AML cell lines (n = 3 venetoclax-sensitive; n = 4 venetoclax-resistant) treated with venetoclax, the DNA-PK inhibitor M3814, or their combination. Apoptosis signalling was analysed by immunoblotting. In vivo efficacy of the M3814 venetoclax combination was assessed in a venetoclax-resistant, KRAS-mutant AML patient-derived xenograft (PDX) model in immunocompromised mice.

In vitro, the venetoclax/M3814 combination produced synergistic cytotoxicity in all venetoclax-sensitive cell lines and three of four venetoclax-resistant lines. The sole non-responsive resistant line (OCI-AML-3-VenR) harboured a homozygous BAX mutation, suggesting that therapeutic synergy is dependent on intact BAX-mediated apoptotic signalling.

Mechanistically, M3814 suppressed AKT phosphorylation in venetoclax-resistant cells, while combination treatment prevented compensatory AKT reactivation and reduced BAD phosphorylation, consistent with enhanced pro-apoptotic signalling. Co-treatment also induced PARP and caspase-mediated DNA-PK cleavage alongside increased γH2AX accumulation, supporting apoptosis induction with impaired DNA repair and DNA damage accumulation. In the KRAS-mutant venetoclax-resistant PDX model, combination therapy significantly reduced leukaemic burden and significantly improved survival compared with either monotherapy, demonstrating in vivo efficacy.

Conclusions: These findings provide compelling preclinical evidence that DNA-PK inhibition can enhance venetoclax efficacy and overcome therapeutic resistance in AML, supporting clinical evaluation of this rational combination strategy, especially in kinase-mutant disease.