CBSM

2026 Combined Biological Sciences Meeting

Supporting WA Life Science Research 1990-2026

Friday 4th September 2026

The University Club

The University of Western Australia

Program

Editors: Sue Fletcher, Patrica Price & Johannes Debler

32nd Annual Combined Biological Sciences Meeting

The University Club of Western Australia
4th September 2026

Cover Image: 2025 CBSM Image Award
Eric Raes, Minderoo Foundation
Tree-of-Life of an eDNA sample from 2L of seawater from Ningaloo (nyinggulu)

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Student Investigator Award Biodiversity	The Western Australian Biodiversity Science Institute — Leading Knowledge Collaboration, Enabling Smarter Decisions
Student Investigator Award Plant Science	Centre for Crop and Disease Management; GRDC Grains Research & Development Corporation; Curtin University

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CBSM 2025 Award Winners

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OceanOmics	Minderoo Foundation	Lok Ting Kwong
Biological Sciences	CBSM Award	Callum Verdonk
STUDENT SYMPOSIA AWARDS		
Biodiversity	Centre for Crop & Disease Management	Anna Faber
Frontiers in Genetics	Perron Institute	Sonakshi Bassi
Cell & Developmental Biology	Fisher Biotech	Charlotte Sofield
Inflammatory Pathways	Curtin Medical Research Institute	Lidia B Medhin
SENIOR SCIENTISTS POSTER AWARDS		
Agricultural & Plant Science	Decode Sience	Huyen Phan
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Immunology	NCARD	Paige I. Warburton
Biomedical Science	Rowe Scientific	Hilary Hii
STUDENT POSTER AWARDS		
Neuroscience	MNDi Foundation	Helena R Bertazzo
RNA Biology	RNA Society	Saskia Saville
Agricultural & Plant Science	MLS	Ruwani Hapuarachchige
eDNA & Environmental Genomics	Minderoo Foundation	Viet-Cuong Han
Health Sciences - Cancer Biology	CBSM	Amelia Dyton
Biochemistry & Molecular Biology	Professor George Yeoh Poster	Keea Inder-Smith
Cell & Developmental Biology	ANZSCDB	Yasmin Henderson Kelly
IMAGE AWARD		
All posters	The Big Picture Factory	Eric Reas

Early Career Research Development
Seminar Room 4

CONVENORS: Dr Linda Wijaya, Dr Vincent Kuek, Ms Alana Celenza

Dr. Sherif Boulos
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Dr Amanda Cleaver
Head, Research Services & Facilities, The Kids Research Institute Australia

Dr. Vivian Chua
Vice Chancellor's Research Fellow
Lead, Cancer Cell Biology Group, Edith Cowan University

A/Prof Bill Bateman
Wildlife Biologist
School of Molecular & Life Sciences, Curtin University

Mark Fear Mentor Award

In honour of the life and legacy of Dr Mark Fear

Associate Professor Mark Fear was a Senior Scientist at the Fiona Wood Foundation and an Associate Professor at The University of Western Austrlia Burn Injury Research Unit. He played a central role in establishing UWA's basic science in burn injury and scarring. His work led to advances in wound treatment and scar reduction.

Mark was a mentor of quiet strength and commitment, supporting the development of future scientists and clinicians. He guided and encouraged dozens of research students, many of whom now lead important work in science and medicine. His collaborative approach brought together researchers from universities, hospitals and industry to progress burn and wound healing research.

He worked closely with Fiona Wood, united by a shared focus on improving outcomes for people recovering from burn injury. In the lab, he led by example — encouraging those around him to think critically, to aim high and stay focused on work that would make a difference. Students and colleagues will remember his insight, curiosity and the care he brought to each project and conversation.

Professor Jacqueline Batley

The University of Western Australia

Using pan genomics to identify disease resistance genes in Brassica species

Abstract

The *Brassicaceae* contains some of the world's most important economic and agronomic crops, which are utilised as edible and industrial oilseeds and vegetables, along with the model plant *Arabidopsis thaliana* and highly diverse wild species. Pathogens, eg. *Leptosphaeria maculans* (causal agent of Blackleg) severely affect *Brassicaceae* crop production. Plant genomes harbour resistance (R) genes, which play a role in plant immunity. The identification of genes underlying QTL is extremely challenging in complex genomes such as in Brassica species. Advances in next-generation sequencing have enabled development of millions of SNPs. However, as an increasing number of genome sequences become available, there is a growing understanding that the genome of a single individual is insufficient to represent the gene diversity within a species. We examined SNP diversity within resistance genes, and this allelic variation is strongly associated with phenotypic variation. However, we have also observed significant presence/absence and copy number variation of R genes. Molecular analyses of resistance genes in Brassica pan genomes are presented. The difficulties associated with identifying functional gene copies within the highly duplicated Brassica genomes will be discussed. This analysis provides a valuable resource for the identification of R genes for enhanced crop protection through analysis of gene diversity and evolution linked to disease.

Biosketch

Jacqueline Batley is a Professor at The University of Western Australia. She was awarded her PhD (University of Bristol) in 2001, moved to Australia (2002) as a senior research scientist at DPI-Victoria, then led a group at the University of Queensland (ARC QEII Research Fellow), from 2007-2014, before moving as an ARC Future Fellow to UWA. Jacqui has several awards; Nancy Millis Medal from the Australian Academy of Sciences and was named the Western Australian Scientist of the Year in 2025. She is a Clarivate Highly Cited Researcher (since 2022). Jacqui's research applies breakthrough biotechnoloy advances for canola crop improvement, by identifying genomic regions controlling traits. Her work has led to new canola cultivars with enhanced productivity, profit, and yield stability through identification of genes linked to shatter tolerance, blackleg disease resistance and oil quality. She is currently an ARC Laureate Fellow focussing on blackleg resistance in the Brassicaceae.

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Biomedical Sciences

Theatre Auditorium

CHAIR: Associate Professor Briardo Llorente

Speaker	Title	
Amelia Dyton	*Targeting NAD+ metabolism in Down Syndrome-associated Acute Lymphoblastic Leukaemia*	9.40
Christopher Witham	*How We Fight Cancer Using Ubiquitin*	9.55
Linda Wijaya	*Unravelling RASopathies Using iPSC-Derived Neural Disease Modelling*	10.10
Mariana Lizeth Orozco Morales	*Targeting PPARγ To Enhance Tumour Immunogenicity And Immunotherapy*	10.25

Targeting NAD+ metabolism in Down Syndrome-associated Acute Lymphoblastic Leukaemia

Amelia Dyton[1,2], Kunjal Panchal[1], Bree Foley[2], Rishi Kotecha[1], Sébastien Malinge[1]

[1]The Kids Research Institute Australia, [2]The Uniersity of Western Australia

Children with Down Syndrome (DS), the most common chromosomal abnormality in humans, have an over 20-fold increased risk of developing an aggressive type of blood cancer called acute lymphoblastic leukaemia (DS-ALL). Young patients with DS-ALL have significantly lower overall survival rates and a higher risk of developing relapse compared to their non-DS peers. Evidently, there is an urgent need for safer, more targeted therapies for these children. Deregulated cellular metabolism is one of the hallmarks of cancer, the importance of which in the pathogenesis and resistance mechanisms of ALL has become increasingly apparent. Moreover, individuals with DS have systemic disruption of key metabolic pathways, many of which overlap with those seen in leukaemia. However, metabolic dysregulation in DS-ALL remains as of yet an elusive topic. Our lab recently screened >9000 compounds in unique DS-ALL cell lines and identified several families of metabolism-targeting compounds, notably nicotinamide phosphoribosyltransferase (NAMPT) inhibitors, as promising families of drugs. NAMPT inhibition effectively kills ALL cells, both *in vitro* and *in vivo*, due to their dependence on nicotinamide (NAD+) synthesis, but this has never been tested in DS-ALL. Here, we assessed the efficacy of several NAMPT inhibitors in vitro. First, we calculated the IC50 values for several NAMPT inhibitors in nine ALL cell lines, including three DS-ALL cell lines uniquely available in our lab. Next, we tested potential synergy between our most promising compound, OT-82, and standard-of-care (S.O.C) agents. Finally, we pre-treated ALL cells with NAD+ before OT-82 treatment, to validate the specificity of this compound. We found that DS-ALL cells were particularly sensitive to NAMPT inhibition, while our synergy assays revealed an additive effect between OT-82 and S.O.C drugs *in vitro*. These pre-clinical findings identify OT-82 as a promising new therapy for further investigation *in vivo* and demonstrate that metabolism is a potential target in DS-ALL.

How We Fight Cancer Using Ubiquitin

Christopher M. Witham[1,2], Nishi Kumari[3], Sarah C. E. Wright[1,2], and Pieter J. A. Eichhorn[1,2]

[1] Curtin Medical School, Curtin University, [2] Curtin Medical Research Institute, Curtin University, [3] Cancer Science Institute of Singapore, National University of Singapore

Ubiquitin is a small, highly conserved protein found in all eukaryotes. The covalent attachment of ubiquitin to substrate proteins is a critical post-translational modification that regulates numerous cellular processes, including protein localisation, signal transduction, and protein degradation. This process, known as ubiquitination, is mediated by a sequential enzymatic cascade comprising E1 activating, E2 conjugating, and E3 ligase enzymes. Conversely, deubiquitinating enzymes (DUBs) remove ubiquitin, providing dynamic control over protein stability and function. Dysregulation in protein ubiquitination has been implicated in tumour initiation, progression, and therapeutic resistance, making it an attractive target for cancer therapy, as demonstrated by the clinical success of proteasome inhibitors such as bortezomib, carfilzomib, and ixazomib. The functional versatility of ubiquitin is largely attributable to a highly conserved, solvent-accessible surface that enables transient, low-affinity interactions with a wide variety of proteins. Interestingly, this interface is amenable to engineering, resulting in the development of ubiquitin variants (UbVs) with high affinity and selectivity for individual ubiquitin-binding proteins. UbVs therefore represent a promising strategy for selectively modulating DUB activity. In this study, we investigate engineered UbVs targeting the deubiquitinases USP10 and USP15, both of which have context-dependent roles in tumour progression and therapy resistance. To enable therapeutic intracellular delivery, we generated in vitro transcribed (IVT) mRNA encoding for either a USP10 or USP15 targeting UbV and established efficient expression in therapy-resistant breast cancer cells following extensive optimisation of mRNA design and lipid nanoparticle (LNP)-mediated delivery. We are now evaluating whether transient UbV expression can restore sensitivity to PI3K pathway inhibition and enhance the efficacy of cereblon-mediated targeted protein degradation therapies. Collectively, this work establishes an RNA-based platform for the intracellular delivery of engineered ubiquitin variants and highlights their potential as a versatile therapeutic strategy for targeting dysregulated DUBs in cancer.

Unravelling RASopathies Using iPSC-Derived Neural Disease Modelling

Linda K. Wijaya[1,2], Catherine A. Forbes[1], Emma Kuzminski[1,2], Nicole C. Shaw[1,2], Paula Gomez[1], Kevin Chen[1], Mitchell Hedges[1], Gareth Baynam[3], Timo Lassmann[1,2], Vanessa S. Fear[1,2].

[1]The Kids Research Institute Australia, Perth, [2]UWA, [3]Rare Care Centre, Perth

The RASopathies are a group of developmental disorders caused by germline variants in genes regulating the RAS/MAPK signalling pathway. RASopathy syndromes, such as Cardiofaciocutaneous syndrome (CFC), Noonan syndrome (NS), and others, share overlapping clinical phenotypes, including developmental delay, cardiac malformations, and craniofacial dysmorphology, making diagnosis challenging. While pathogenic variants are identified in about half of patients, many remain undiagnosed due to novel variants or variants of uncertain significance (VUS). Each VUS requires functional validation to determine pathogenicity, a process that can take more than 5 years, if achieved at all. There is an urgent need to accelerate variant interpretation to support diagnosis. To address this, we developed a laboratory platform combining precise CRISPR-Cas9 gene editing in induced pluripotent stem cells (iPSCs) with neural disease modelling to fast-track genetic variant interpretation. In this study, BRAF variants, the most common cause of CFC, and present in a subset of NS cases, were investigated. Three iPSC lines harbouring a CFC-associated pathogenic variant (BRAF p.Leu499Glu), an NS-associated pathogenic variant (BRAF p.Trp532Cys), a VUS (BRAF p.Phe548Ser), and isogenic controls, were generated. These lines were differentiated into neural progenitor cells to assess neurodevelopmental phenotypes. Transcriptomic profiling, protein expression, and cell-based signalling analyses were performed to evaluate biological processes and variant-specific effects on RAS/MAPK pathway regulation. Our results reveal variant-specific biological processes and differential MAPK activation, providing support data for the classification of the BRAF VUS. This study demonstrates the utility of CRISPR-Cas9 gene editing and iPSC-based disease models for accelerating the interpretation of RASopathy variants.

Targeting PPARγ To Enhance Tumour Immunogenicity And Immunotherapy

M.L. Orozco Morales[1,2], J. McQuillan[1,3], B.J. Vitali[1], J. Lu[1], A. Scaffidi[3], J. King[3], V. Fear[1], M. Piggott[3], W.J. Lesterhuis[1,2]

[1]The Kids Research Institute Australia, [2]School of Biomedical Sciences and [3]School of Molecular Sciences, The University of Western Australia

Immunotherapy has transformed cancer treatment, yet many cancers remain resistant. Emerging insights into the tumour microenvironment reveal that altered metabolism supports tumour proliferation and suppresses anti-cancer immunity. Peroxisome proliferator-activated receptor gamma (PPARγ) is a key regulator of energy and lipid metabolism under normal physiological conditions. In many cancers, PPARγ becomes activated, either due to the abundance of fatty acids in the tumour microenvironment or through mutations in *PPARG* or *RXR* genes, promoting tumour growth and dampening anti-cancer immunity, highlighting PPARγ as a potential therapeutic target. To evaluate the role of PPARγ in tumour immunogenicity, we used genetically modified mouse models (*Pparg*$^{+/-}$ or *Pparg*^{LoxP} Cre mice), alongside CRISPR-engineered cancer cell lines. Subcutaneous tumour growth was assessed in wildtype C57BL/6 and genetically modified mice, with or without immune checkpoint therapy. Flow cytometry, RNA sequencing, and untargeted metabolomics were used to characterise immune and metabolic changes. Deletion of PPARγ led to an increased anti-tumour immune response and improved responsiveness to immune checkpoint therapy. Transcriptomic profiling of spleens and tumours revealed downregulation of E2F, G2M, and Myc-associated pathways. Metabolomic analysis of *Pparg*$^{+/-}$ spleens showed increased biosynthesis of arginine, glycine, and alanine. Immune profiling demonstrated enrichment of CD4^{+} and CD8^{+} T cells and activated B cells, indicating a more immunostimulatory environment. Our findings validate PPARγ as a promising therapeutic target for enhancing tumour immunogenicity. Inhibiting PPARγ activity may reprogramme metabolic and immune networks to improve the efficacy of immunotherapy.

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Plants and Genetics

Seminar Room 4

CHAIR: Professor Lee Hickey

Speaker	Title	
Daniel Edge-Garza	*Marker-Assisted Seedling Selection for Improved Apple Breeding Efficiency in Australia*	9.40
Kristina Gagalova	*Beyond RNAseq: Integrating Transcriptomics, Proteomics, and QTL Mapping to Reveal Driving Infection Mechanisms in Patastagonospora nodorum - Wheat pathosystem*	9.55
Ulduz Vafadarshamasbi	*Toward robust chromosome-scale genome assembly with iterative Hi-C scaffolding and validation*	10.10
Zeeshan Ahmed	*Stable Inheritance and Quantitative Phenotyping of Variegation in Anigozanthos flavidus 'Stripie': A Model for Investigating Photosynthetic Compensation Mechanisms*	10.25

Marker-Assisted Seedling Selection for Improved Apple Breeding Efficiency in Australia

Daniel Edge-Garza[1], Sultan Mia[1], Kristen Brodison[2], Dario Stefanelli[3]

[1]Department of Primary Industries and Regional Development, Jandakot,
[2]Department of Primary Industries and Regional Development, Bunbury,
[3]Department of Primary Industries and Regional Development, Manjimup

Breeding new horticulture cultivars is a lengthy and costly process, with substantial resources required to raise and evaluate large seedling populations. As part of the Next Generation Orchards project (a national project aimed to improve selection efficiency of various breeding programs in Australia), funded by the Department of Primary Industries and Regional Development (DPIRD) and Horticulture Innovation Australia (HIA), we are implementing marker-assisted seedling selection (MASS) to identify seedlings unlikely to meet breeding objectives before field establishment. Marker information was prioritized according to the objectives of individual crosses, allowing selection decisions to reflect the specific breeding goals of each family. In Western Australia MASS was implemented in the Australian National Apple Breeding Program and of the apple seedlings screened, 22% were culled, reducing demands on field space and other resources that can instead be reallocated to novel breeding objectives and further improved efficiency of the program. The available marker tests could have resulted in culling more than 90% of the seedlings; however, such stringent selection would risk removing the genetic diversity and novel phenotypes necessary for cultivar development. This highlights the importance of balancing predicted genetic merit with the need to maintain sufficient population size for discovery and selection of novel breeding material. The DNA data also provided benefits beyond trait selection. In one family, marker information confirmed the paternal origin of seedlings for which two potential paternal parents had previously been identified, enabling their correct assignment to the appropriate family for subsequent evaluation. Our experience demonstrates that MASS can contribute to improving efficiency by reducing unnecessary resource use while retaining sufficient genetic diversity for discovery. Future implementation will benefit from explicitly integrating marker priorities with the breeding objectives of individual crosses when designing selection strategies.

Beyond RNAseq: Integrating Transcriptomics, Proteomics, and QTL Mapping to Reveal Driving Infection Mechanisms in *Patastagonospora nodorum* - Wheat pathosystem

Kristina K. Gagalova[1], Lukas Hunziker[1], Fiona Kamphuis[1], Eiko Furuki[1], Kasia Rybak[1] and Huyen Phan[1]

[1]Centre for Crop and Disease Management, Curtin University

Parastagonospora nodorum, the causal agent of Septoria nodorum blotch (SNB), poses a severe threat to global wheat production. Quantitative molecular resistance offers durable, broad-spectrum defense against SNB; understanding its multi-layered host regulation is crucial for breeding resistant wheat cultivars. Uncovering quantitative resistance mechanisms remains difficult because single-layer approaches, such as transcriptomics (RNAseq), may generate long candidate gene lists that require further effort to navigate the multi-gene pathway dynamics. We investigated whether integrating transcriptomic and proteomic data can effectively resolve host pathway regulation and pinpoint key single-gene candidates driving host infection. Additionally, we combined these pathway-derived candidate genes with mapped quantitative trait loci (QTL) derived from an infected mapping population to anchor host defense signals directly to genomic regions governing disease response and study the concordance of gene candidates across the methods. We profiled time-course transcriptomic and proteomic datasets from two elite wheat cultivars exhibiting contrasting disease responses, following *P. nodorum* infection. To bridge these layers, we compared major multi-omics integration strategies, including Partial Least Squares (PLS) regression, co-expression clustering, network analysis, and AI-driven embeddings. These multi-layer pathway outputs will be then cross-referenced against mapped QTL intervals to determine how candidate pathway genes overlap with genomic regions underlying pathogenicity. Integrated transcriptome-proteome profiling revealed tight molecular layer and identified four primary coordinated host defence pathways: early immune signalling, redox homeostasis, intracellular transport, and stress-response networks. Cross-referencing these integrated multi-layer networks against QTL mapping is currently underway and will enable us to systematically overlap pathway dynamics with genomic loci and obtain a concordance overview of the methods. This study demonstrates the value of integrating transcriptomic, proteomic and genomic approaches to better understand wheat defense against SNB and has the potential to identify high-confidence molecular targets for marker-assisted breeding of disease-resistant varieties.

Toward robust chromosome-scale genome assembly with iterative Hi-C scaffolding and validation

Ulduz Vafadarshamasbi[1], Irina kuznetsova[1], Mario Fruzangohar[2], Adam Sparks[1]

[1] Centre of Crop and Disease Management, Curtin University, [2] Biometry Hub, School of Agriculture, Food and Wine, Adelaide University

Chromosome-scale genome assemblies have become fundamental resources for plant genomics, enabling studies of genome evolution, structural variation, comparative genomics, and molecular breeding. Despite advances in long-read sequencing technologies, reconstructing large and repeat-rich genomes into accurate chromosome-scale assemblies remains challenging. Although Hi-C sequencing is widely used for chromosome-scale scaffolding, practical guidance for systematic scaffold validation and evidence-based assembly decision-making remains limited. To address this gap, we developed the HiC-Scaffold framework, a modular and reproducible computational workflow that integrates assembly quality assessment, Hi-C scaffolding, comparative assembly evaluation, and iterative validation. The framework incorporates predefined quality-control checkpoints that combine complementary sources of evidence to support transparent and reproducible assembly decisions rather than relying on conventional assembly statistics or a single scaffolding strategy. The framework was demonstrated using a large and repeat-rich barley genome as a representative case study, integrating approximately 76 Gb of PacBio HiFi sequencing and 940 million Hi-C paired-end reads. Comparative evaluation of alternative scaffold configurations, together with iterative Hi-C contact-map validation, supported evidence-based selection of the final chromosome-scale assembly. Application of the framework supported construction of a chromosome-scale assembly that anchored 4.20 Gb to the seven barley chromosomes, achieved a scaffold N50 of 613 Mb, and recovered 99.5% complete BUSCO orthologues, while Hi-C contact maps confirmed high structural integrity. By embedding quality assurance throughout the assembly process, the HiC-Scaffold framework provides a practical and reproducible strategy for generating reliable chromosome-scale genome assemblies and can be readily adapted to other complex genomes, including future chromosome-scale, telomere-to-telomere, and pangenome initiatives.

Stable Inheritance and Quantitative Phenotyping of Variegation in *Anigozanthos flavidus* 'Stripie': A Model for Investigating Photosynthetic Compensation Mechanisms

Zeeshan Ahmed[1], Cornelia Hooper[1], Ian Small[1], David Field[2], Agyeya Pratap[3], Praful Umaretiya[4], Digby Growns[4]

[1]ARC Centre of Excellence Plant energy Biology, School of Molecular Sciences, UWA, [2]Department of Applied Biosciences, Macquarie University, [3]Australian Plant Phenomics Network UWA , [4]DBCA, Kings Park & Botanical Gardens, Perth

Anigozanthos flavidus 'Stripie' is the only known variegated Kangaroo Paw among WA native *Anigozanthos* species. Unlike most variegated plants, which typically exhibit reduced growth due to loss of photosynthetically active tissue, 'Stripie' maintains stable variegation while growing as vigorously as fully green plants. This unusual phenotype suggests an underlying energy-compensation mechanism with potential relevance to plant productivity and photosynthetic efficiency. The stability, inheritance, and quantitative assessment of variegation in 'Stripie' had not been formally investigated. This study aimed to determine whether the phenotype is stable under commercial propagation, genetically heritable, and measurable using objective phenotyping methods. 'Stripie' plants were propagated through tissue culture following flower bud induction over three years. To assess inheritance, 'Stripie' was crossed with the tetraploid cultivar 'Early Spring' to produce a triploid F_1 population, and self-pollinated progeny were evaluated for segregation of variegation. Variegation was quantified using multispectral imaging (PlantEye, TraitFinder) from five scans of three plants. NDVI vegetation indices was used to calculate green and white leaf area. Tissue culture propagation produced 20 plants in 2022, 100 in 2023, and 400 in 2024. All plants displayed stable, true-to-type variegation with no observed reversion. The triploid 'Early Spring' × 'Stripie' F_1 retained visible variegation, confirming transmission through sexual reproduction. Selfed progeny segregated in a 1:2:1 ratio of hyper-variegated, variegated, and green phenotypes, consistent with a single heterozygous locus exhibiting incomplete dominance and dosage-dependent expression. TraitFinder analysis provided reproducible quantification of variegation, with representative scans recording upto 70.8% green and 29.2% white leaf area. Variegation in *A. flavidus* 'Stripie' is stable, heritable, and quantifiable. Future transcriptomic, proteomic, and metabolomic analyses will investigate the molecular basis of the apparent photosynthetic compensation mechanism, providing insights to support breeding and potentially improve photosynthetic efficiency in horticultural and crop species.

Minghao Zheng

Microbiology and Genomics

Seminar Room 1

CHAIR: Professor Ryan Lister

Speaker	Title	
Dilruka Kasturiarachchi	*DBHS Proteins as a Model for Studying Biomolecular Condensate Assembly and Maturation*	9.40
Marhami Fahriani	*Beyond Enterococcus faecalis and Enterococcus faecium: Genomic insights into other enterococci causing bacteraemia*	9.55
Roland Politan	*Engineering phosphate-controlled PHB production in Vibrio natriegens from renewable feedstocks*	10.10
Xavier Barton	*Do Ticks Care About the Environment?*	10.25

DBHS Proteins as a Model for Studying Biomolecular Condensate Assembly and Maturation

Dilruka V Kasturiarachchi[1], Andrew C Marshall[1], Charles S Bond[1]

[1] School Molecular Sciences, The University of Western Australia

Biomolecular condensates, formed through liquid-liquid phase separation (LLPS), enable membraneless compartmentalization and are essential for cellular organization. Dysregulated LLPS can drive aberrant "maturation" from dynamic liquid condensates to solid, irreversible assemblies, a process implicated in neurodegenerative disease and cancer. The DBHS (Drosophila Behaviour/Human Splicing) family, comprising SFPQ, NONO and PSPC1, are RNA-binding paralogues that undergo LLPS and scaffold the paraspeckle, a nuclear membraneless organelle. Dysregulation of DBHS proteins is linked to Amyotrophic Lateral Sclerosis (ALS), triple-negative breast and prostate cancers. Prior work using truncated protein constructs showed that DBHS proteins function obligately as dimers and preferentially heterodimerize; here, we extend these findings to full-length SFPQ, NONO and PSPC1 to ask how condensate composition (homo- vs heterodimeric) shapes phase behaviour, maturation and downstream biochemical function. We hypothesized that heterodimerization alters the biophysical properties of full-length DBHS proteins relative to their homodimeric counterparts. These full-length proteins were recombinantly expressed and mixed pairwise at 37°C to drive heterodimerization, monitored by analytical size-exclusion chromatography (SEC) and complemented with biological small-angle X-ray scattering (Bio-SAXS). All three heterodimer combinations formed completely within 30 minutes. By analytical SEC, two of three heterodimers eluted at volumes matching their larger homodimeric subunit, while PSPC1-NONO eluted earlier than either homodimer, indicating a larger hydrodynamic radius. Bio-SAXS corroborated this: all heterodimers showed modestly increased radius of gyration (Rg) relative to homodimers, with PSPC1-NONO showing the largest increase, suggesting a more extended, potentially stable conformation. These results show that full-length DBHS heterodimerization measurably alters protein conformation, supporting the hypothesis that heterodimer preference reflects increased conformational stability. *In vitro* phase separation and maturation of homo- and heterodimeric species are being characterized by turbidity assays and fluorescence imaging. Ongoing phase separation assays will test whether these differences translate into altered condensate formation and maturation kinetics, informing how composition tunes paraspeckle assembly and disease-relevant aggregation.

Beyond *Enterococcus faecalis* and *Enterococcus faecium*: Genomic insights into other enterococci causing bacteraemia

Marhami Fahriani[1], Geoffrey W Coombs[1,2,3], Denise A Daley[2,3], Christopher A Mullally[1], Shakeel Mowlaboccus[1,3] on behalf of the Australian Group on Antimicrobial Resistance

[1]School of Medical, Molecular and Forensic Sciences, Murdoch University, [2]Department of Microbiology, PathWest Laboratory Medicine-WA, Fiona Stanley Hospital, [3]Australian Group on Antimicrobial Resistance, Fiona Stanley Hospital

Enterococci cause approximately 10% of bacteraemia globally, but surveillance predominantly focuses on *Enterococcus faecalis* and *Enterococcus faecium*. Non-*faecium* non-*faecalis* (NFF) enterococci are increasingly recognised as opportunistic pathogens, yet routine laboratory methods may misidentify these species and underestimate their epidemiological and antimicrobial resistance (AMR) burden. NFF enterococci may also harbour AMR genetic determinants shared with *E. faecalis* and *E. faecium*, suggesting a role as reservoirs of transferable AMR genes. This study aimed to characterise the genomes of NFF enterococci causing bacteraemia in Australia and to identify their AMR genetic determinants. Antimicrobial susceptibility testing and whole-genome sequencing (WGS) were performed on 833 NFF enterococcal bacteraemia isolates collected across Australia over 10 years. Phylogenetic reconstruction was performed using IQ-TREE 2, whilst AMR genetic determinants and mobile genetic elements were identified using AMRFinderPlus and MobileElementFinder, respectively. WGS confirmed 729 isolates as NFF enterococci, whereas 13.4% (n=104) were misidentified as *E. faecalis*, *E. faecium* or incorrect NFF species. The 47 isolates initially reported as *E. casseliflavus* were reclassified into *E. innesii* (n=27), *E. entomosocium* (n=18), and Candidatus *E. testudinis* (n=2). Ampicillin resistance (7.7%, n=57) and benzylpenicillin resistance (10.4%, n=72) were associated with mutations in the *pbp* gene and its promoter, whilst ciprofloxacin resistance (4.3%, n=25) was linked to amino acid substitutions in ParC, ParE, GyrA, and GyrB. Resistance to erythromycin (27.9%, n=176), high-level gentamicin (2.2%, n=16), and tetracycline (20.5%, n=108) was associated with *ermA/ermB*, *aac(6')-Ie/aph(2")-Ia*, and *tetM/tetS/tetO/tetL*, respectively. Multiple AMR genes were carried on mobile genetic elements, including Tn*554*, Tn*5405*, Tn*6000*, Tn*5801*, and Tn*916*, previously reported in other enterococci and Gram-positive bacteria. NFF enterococci are clinically important but under-recognised causes of bacteraemia and may act as reservoirs of transferable AMR genes, supporting the need for improved species-level identification and ongoing genomic surveillance.

Engineering phosphate-controlled PHB production in *Vibrio natriegens* from renewable feedstocks

Roland J Politan[1], Joshua Z Wong[1], Kathryn M Morris[1], Bassey Friday Bassey[1], Ben RK Roller[2], Gavin Flematti[1], Simona Della Valle[1,*], and Georg Fritz[1,*]

[1]School of Molecular Sciences, The University of Western Australia, [2]Division of Microbial Ecology, Centre for Microbiology and Environmental Systems Science, The University of Vienna

Plastic pollution has become one of the defining environmental challenges in this generation. The very durability that makes these materials useful renders them persistent pollutants, and growing concern over their environmental burden has intensified the search for biodegradable alternatives. Polyhydroxybutyrate (PHB) is a biodegradable polyester and a leading candidate to replace petrochemical plastics. *Vibrio natriegens* is a natural PHB producer and is exceptionally well-suited for next-generation biomanufacturing due to its nonpathogenicity, rapid growth, genetic tractability, and feedstock flexibility. The regulatory circuits that govern native PHB accumulation in *V. natriegens*, and whether these can be exploited to build an efficient, self-triggering bioprocess, is yet to be characterized. Using a luminescence biosensor of the endogenous *phaBAPC* operon, we found that its promoter is induced by phosphate, but not nitrogen, depletion, a previously unreported regulatory pattern for a *Vibrio* species. Because the cells deplete phosphate as they grow, the switch from growth to PHB production is dynamic and self-actuating and requires no chemical inducer or engineered regulatory circuit. Integrating additional *phaBAPC* copies into competing biosynthetic and neutral loci, which simultaneously deletes those loci, led to a three-fold increase in PHB titer using acetate as sole carbon source. Process optimization towards a residual-phosphate induction regime further improved production, and a kinetic model predicted the initial phosphate concentration maximizing volumetric titer. The phosphate-starvation strategy drove PHB synthesis from six renewable carbon sources, with sugars and glycerol outperforming acetate, which we nonetheless adopted as a sustainable, non-food feedstock. Kinetic modeling validation near this optimum yielded 0.123 g/L PHB from acetate. Under low-phosphate conditions, the strain accumulated up to 69.1% PHB per cell dry weight from acetate alone, without complex nutrient supplementation. Together, these results demonstrate phosphate depletion as an autonomous trigger for sustainable two-stage PHB production in *V. natriegens*.

Do Ticks Care About the Environment?

Xavier Barton[1], Joseph B. Fontaine[2], Shanan S. Tobe[1], Siobhon L. Egan[1], Charlotte L. Oskam[1]

[1]School of Medical, Molecular and Forensic Sciences, College of Environmental and Life Sciences, Murdoch University, [2]School of Environmental and Conservation Sciences, College of Environmental and Life Sciences, Murdoch University

Ticks pose a global threat through pathogen transmission to humans, companion animals and livestock. Population connectivity and landscape movement patterns shape how introduced tick species or novel pathogens might spread following an incursion. Western Australia's most common human-biting tick, *Amblyomma triguttatum* (ornate kangaroo tick), offers a valuable model for understanding these processes on the Swan Coastal Plain, a biogeographic region where tick population structure remains uncharacterised. Landscape and movement patterns of this species are not currently characterised, limiting our ability to anticipate and model novel tick or pathogen incursion events into the region. We addressed this gap by generating the first population-level genomic data for *A. triguttatum*, sampling populations across the Swan Coastal Plain. Using 379 *A. triguttatum* specimens, we employed double digest Restriction-site Associated DNA sequencing (ddRADseq) to characterise population structure and measured the effect of environmental factors, including land use, soil type and climate, as predictors of that structure. Population structure analysis identified four main genetic clusters, comprising a primary split between populations north and south of the Swan River and Perth city, with secondary splits within each of these two groups. Vegetation coverage and hard barrier coverage (built and water) showed opposing threshold effects on genetic distance between individuals. Genetic distance dropped sharply once vegetation coverage exceeded approximately 65%, indicating that dense vegetation supports gene flow, while genetic distance rose sharply once hard barrier coverage exceeded approximately 25%, indicating that urban and water features restrict gene flow. Soil type and climate showed no statistically significant effect on genetic structure. This research contributes foundational data on how terrestrial pathogen vectors move across the Australian landscape and interact with landscape features, supporting the development of predictive models with implications for public health, agriculture and wildlife management.

Notes

Dame Juliet Gerrard

The University of Auckland

Tales of the unexpected: a science career across universities, business, and government with some lessons learned along the way.

Abstract
In this talk I'll discuss some highlights, low lights, and quirky twists and turns in a protein science career, which started in Oxford, took a sharp left to Lincoln, New Zealand, and wound its way up to Auckland. Surprisingly, it included a six year stint as the New Zealand Prime Minister's Chief Science Advisor, providing evidence for policy across a wide range of different topics, including an all-consuming pandemic and a volcanic eruption. I'll reflect on what I learned along the way, especially about the tricky business of providing scientific evidence to politicians and policymakers.

Biography
Dame Juliet Gerrard trained at Oxford and moved to New Zealand in 1993. Her research career has been broad and interdisciplinary, centred on protein science. She is currently a professor at the University of Auckland (based across the Faculties of Science and Engineering). From 2018-2024 Juliet served as the Prime Minister's Chief Science Advisor to three Prime Ministers (Jacinda Ardern, Chris Hipkins, Chris Luxon) during a tumultuous time. She provided science advice on a broad range of topics, including plastics, cannabis, AI in healthcare and food waste. She also had a front row seat during three emergencies - the Christchurch mosque shooting, the volcanic eruption of Whakaari | White Island, and the COVID-19 pandemic. She will reflect on her experiences in this and other roles in this presentation.

The Australian Society for Microbiology

Theatre Auditorium

CHAIRS: Dr Lucy Furaro & Dr Chris Mullaly

	Keynote Presentation	
Dr Christine Carson	*Challenges to Addressing the Complexity of Antimicrobial Resistance Through Innovation in Diagnostics and Therapeutics*	1.30
	Student Presentations	
Dávid Szabó	*A Dual Promoter System and Translational Derepression Activates Horizontal Gene Transfer in Mesorhizobium japonicum R7A*	2.00
Princy Shoby	*Bacteriophages Against Multidrug-Resistant Enterococcus faecium*	2.15
Vanessa Tenaglia	*PilA Is Essential for Streptococcus pyogenes Strain M1 Attachment to Primary Tonsil Epithelial Cells*	2.30
Tim Cusens	*Rapid, Non-ureolytic Calcium Carbonate Precipitation by Vibrio natriegens in Synthetic Seawater*	2.45
	Tea Break	3.00
	Invited Presentations	
Dr Rutendo Mapengo	*Two clades, One threat: The Genomic Epidemiology of Candidozyma auris in Western Australia, 2018–2024*	3.30
Dr Chris Mullaly	*Transforming neonatal sepsis diagnosis: developing rapid molecular approaches for early pathogen detection from low blood volumes*	4.00

Dr Christine Carson

Senior Research Fellow, The University of Western Australia
Chief Scientific Officer Cytophenix

Challenges to Addressing the Complexity of Antimicrobial Resistance Through Innovation in Diagnostics and Therapeutics

Abstract

Antimicrobial resistance (AMR) is a critical global health challenge, driving urgent demand for faster diagnostic tests and novel therapeutic strategies to preserve antibiotic efficacy and reduce mortality from resistant infections. Despite discoveries and advances in antimicrobial agents and rapid diagnostic tests, translation into clinical practice remains limited by fragmented funding, weak market incentives, and slow adoption of new products in healthcare settings. Here, I review the landscape of successes and challenges using examples from new and emerging antimicrobial agents, including new antibiotics and phage therapy, and recent diagnostic innovations such as rapid phenotypic susceptibility testing platforms. Areas of focus include market drivers, regulatory pathways, reimbursement models, and laboratory workflow integration. Evidence highlights that while technological capability exists, systemic barriers including insufficient and sporadic investment, misaligned reimbursement structures, and regulatory complexity, hinder widespread implementation. Innovation in diagnostic and therapeutic countermeasures to address AMR are happening, but face significant systemic headwinds. Several rapid diagnostic platforms demonstrate significant time-to-result reductions, yet adoption rates remain low in most regions. New, approved antimicrobials deliver promising clinical activity but adoption remains slow. Technological solutions to AMR are possible and some are maturing, reaching the patients who need them. Achieving sustainable impact requires coordinated investment, aligned regulatory frameworks, better market incentives, and health system readiness to change practice. Bridging these gaps will accelerate deployment of life-saving diagnostics and treatments and mitigate the devastating effects of AMR.

Biography

Dr Christine Carson is a Senior Research Fellow at The University of Western Australia and CSO at Cytophenix, a UWA spinout company. As a highly experienced microbiologist she bridges research and industry, with an extensive background in developing novel diagnostic tools, focusing on antimicrobial susceptibility testing. By improving analytical and diagnostic tools as a countermeasure to antimicrobial resistance, she seeks a better way to quantify and analyse antimicrobial susceptibility and develop fast, antimicrobial susceptibility tests to tailor antibiotics to infections.

A Dual Promoter System and Translational Derepression Activates Horizontal Gene Transfer in *Mesorhizobium japonicum* R7A

Dávid Szabó[1,2], Tahlia Bastholm[1,2], and Joshua Ramsay[1,2]

[1]Curtin Medical Research Institute, Curtin University, [2]School of Diagnostic and Therapeutic Sciences, Curtin University

Heritable differentiation of cells is well documented in multicellular organisms, but less thoroughly explored in prokaryotes. In populations of the nitrogen-fixing bacterium *Mesorhizobium japonicum* R7A, ~2% of cells enter an epigenetically differentiated state termed 'R7A*'. R7A* cells become active for quorum sensing and can participate in horizontal transfer of the integrative and conjugative element ICE*Ml*Sym^{R7A}, which encodes genes for nitrogen fixation. The R7A* state is maintained by a highly stable epigenetic switch. In R7A* cells, the master regulator QseC2 silences transcription of the quorum sensing and ICE transfer repressor *qseM*. Transient overexpression of *qseC2* in R7A cells is sufficient to activate the R7A* state in perpetuity, even after the *qseC2* overexpression vector is removed. Surprisingly, *qseC2* mRNA abundance is similar in R7A and R7A* cells, suggesting R7A* activation may involve regulation of *qseC2* translation. In this work, 5′ONT-cappable seq was used to map mRNA transcriptional start sites across the R7A and R7A* transcriptomes. This revealed that *qseC2* mRNA is transcribed from distinct transcriptional start sites in R7A and R7A* cells. In R7A cells, the 5′UTR of *qseC2* mRNA contains an inverted repeat sequence overlapping the ribosomal binding site. In R7A* cells, transcription of *qseC2* initiates 18bp downstream, resulting in a shorter 5′UTR that omits half the inverted repeat. Here, we show that translation of R7A* *qseC2* mRNA is ~4-fold higher than R7A *qseC2* mRNA, which contains the 5′UTR inverted repeat. We also show that QseC2 binding to the promoter region simultaneously blocks transcription of the longer R7A mRNA and stimulates transcription of the shorter R7A* mRNA, thereby positively autoregulating its own translation. This work demonstrates how transcriptional and translational regulation can work together to establish an epigenetic fuse that, once activated, stably maintains itself through transcriptional activation and translational derepression.

Bacteriophages Against Multidrug-Resistant *Enterococcus faecium*

Princy Shoby[1], Shakeel Mowlaboccus[1,2], Geoffrey Coombs[1], Lucy Furfaro[3]

[1]School of Medical, Molecular and Forensic Sciences, Murdoch University, [2]School of Biomedical Sciences, UWA, [3]Division of Obstetrics and Gynaecology, Medical School, UWA

Enterococcus faecium is part of the human gastrointestinal tract microbiome, however, can become pathogenic when the opportunity arises. Antimicrobial resistance in *E. faecium* has resulted in WHO high-priority status. Obligately lytic phages are therapeutic alternatives to address multidrug-resistant (MDR) infections. To build an arsenal of effective options against the increasingly resistant invasive *E. faecium* infections, the study aimed to isolate and characterise obligately lytic bacteriophages of *E. faecium*. Four phages were isolated and characterised phenotypically and genotypically against a clinical invasive biobank of MDR *E. faecium* isolates, including extensive host range and infection kinetics, sequencing, virion stability and electron microscopy. Isolated phages ΦPS-Efm-1, ΦPS-Efm-2, ΦPS-Efm-3 and ΦPS-Efm-5 were confirmed by sequencing as obligately lytic, with combined activity against all (n=23) tested clinical *E. faecium* isolates, despite the presence of at least one anti-phage defence system in the bacterial hosts. No association between phage activity and host sequence types was identified. Phage virion stability was observed across pH 4-8 and clinically-relevant temperatures, with varying storage temperatures resulting in different recovery rates. All phages belonged to *Caudoviricetes*, with genome sizes ~ 140-155 kb. The characteristics of ΦPS-Efm-1, -2, -3 and -5 highlight the therapeutic potential of the phage candidates against critical infections. Additionally, the ~40 min latent periods (time from phage adsorption to release of progeny phages) could enable formulating rapid-acting therapeutic phage cocktails. To the best of our knowledge, these are the first documented obligately lytic phages isolated in Australia against clinical *E. faecium*.

PilA Is Essential for *Streptococcus pyogenes* Strain M1 Attachment to Primary Tonsil Epithelial Cells

Vanessa Tenaglia[1,2], Jasreen Kular[2], Jua Iwasaki[1], Wenna Lee[1], Timothy C. Barnett[1,2]

[1]Wesfarmers Centre of Vaccines and Infectious Disease, The Kids Research Institute Australia, [2]School of Biomedical Sciences, The University of Western Australia

Host cell attachment and colonisation are critical steps in the pathogenesis of Streptococcus pyogenes (Strep A). The pilus of Strep A, specifically the tip subunit PilA, has been shown to be important in epithelial attachment in in vitro models (1-4). However, the limitations of these studies are that they have used cell lines, a limited number of Strep A strains (M types) and very high multiplicities of infection(1-4). Thus, we aimed to use a primary tonsil cell culture model to investigate the role of PilA in tonsil attachment and determine if PilA is essential for attachment in a diverse range of M types. PilA isogenic knockout mutants were generated in strains M1, M1UK, M5, M18, M49 and M53, using allelic recombination. Confluent monolayers of primary tonsil epithelial cells were infected with wild type and PilA knockout mutants. After 30 minutes of incubation, cells were trypsinised, lysed and cell lysates plated to determine colony forming units per millilitre. Attachment in M1 and M1UK PilA mutant strains was completely abolished ($p<0.05$) compared to wild-type. No significant difference in attachment was observed between wild-type and PilA mutants for the other M types investigated (M18, M5, M53, M49). Additionally M protein and FCT region isogenic knockout mutants were generated for a subset of the non-pilA essential strains (M18 and M53). For M18 neither the FCT region nor M protein mutant had a significant change in attachment, however in the M53 M protein and FCT knockout mutants, attachment was significantly reduced ($p<0.05$) compared to the wild-type. Furthermore, PilA is an essential adhesin for tonsil epithelial cell attachment in only M1 and M1UK strains. M protein and a protein encoded in the FCT region of M53 is also important for attachment, highlighting strain-specific attachment mechanisms. Future studies will further characterise essential adhesins and their host receptors.

Rapid, Non-ureolytic Calcium Carbonate Precipitation by *Vibrio natriegens* in Synthetic Seawater

Tim Cusens[1], Roland Politan[1], Susanne Gebhard[2], Georg Fritz[1]

[1]School of Molecular Sciences, The University of Western Australia, [2]Institute of Molecular Physiology, Johannes Gutenberg-Universität

Microbially induced calcium carbonate precipitation (MICP) shapes marine and sedimentary carbonate formation and is increasingly harnessed for biotechnology applications, such as carbon sequestration and biocement. Most engineered systems rely on ureolysis, which alkalinises bulk media by releasing ammonia, an environmental nutrient pollutant. Non-ureolytic, heterotrophic pathways are more benign but typically slow. Here, we investigated whether the fast-growing marine bacterium *Vibrio natriegens* could precipitate $CaCO_3$ without urea, how precipitation couples to bulk media chemistry, and the role of central carbon metabolism in MICP. Cultures were grown in defined synthetic seawater (MBLK) with acetate or glucose as sole carbon source and 15–120 mM Ca^{2+}; growth, acetate consumption, bulk pH, soluble Ca^{2+}, and mineralogy were tracked over three days. *V. natriegens* supported two distinct MICP modes: a classical urease-dependent pathway on glucose plus urea, and a rapid, ammonia-free pathway on acetate that removed most soluble Ca^{2+}within two days (~60mM to ~10 mM), faster than the ureolytic route. When grown on calcium acetate, bulk pH transiently fell while Ca^{2+}, was precipitated and acetate flux was high, decoupling mineral formation from bulk saturation and implicating cell-associated microenvironments as the locus of control for acetate-dependent MICP. A ΔaceA strain and a laboratory evolved strain with improved growth rate on acetate shifted the timing of precipitation, indicating that carbon metabolism offers genetic control over the process. Dose-response experiments defined an approximately 2:1 acetate:Ca^{2+}, ceiling, above which precipitation plateaued, indicating an alkalinity-limited regime determined by acetate availability. Together these results establish V. natriegens as a fast, genetically tractable, urea-free chassis for marine biomineralisation and identify carbon flux and acetate:calcium stoichiometry as quantitative handles for engineering precipitation rate and yield, providing a foundation for seawater-grown, low-carbon engineered materials.

Rutendo Mapengo
Department of Microbiology, PathWest Laboratory Medicine and Fiona Stanley Hospital Network

Two clades, One threat: The Genomic Epidemiology of *Candidozyma auris* in Western Australia 2018–2024

Abstract

Candidozyma auris (formerly Candida auris) is a multidrug-resistant fungal pathogen, reported in over 60 countries and associated with nosocomial outbreaks. A limited number of mainly travel-associated cases have been reported in Australia. Emergence of multidrug-resistant *C. auris,* including azole and echinocandin resistance, is worrisome. Resistance has been linked to *ERG11* and *FKS1* mutations. We describe the molecular epidemiology of *C. auris*, using WGS for isolates collected between 2018 and 2024 in Western Australia. Nineteen cases of culture-confirmed *C. auris* infection or colonisation were identified through laboratory surveillance. Isolates were identified by Bruker MALDI-TOF, then sequenced using MiSeq Illumina. Phenotypic characterisation included pseudo-hyphae production and aggregate formation. Antifungal susceptibility testing was performed using commercial broth microdilution. Molecular analysis was based on WGS data; bioinformatics and phylogenetic analysis confirmed clade assignments. Cases had a median age of 54 years (IQR: 48 – 71). Phylogenetic analysis showed 19 isolates that belong to two clades: clade III South African and clade I South Asian. Fourteen isolates were from screening swabs while five were from clinical samples and these patients were treated. Among those treated, mortality rate was 60% (3/5). Fluconazole resistance was detected in 94.7% (18/19) of isolates (provisional Centre for Disease Control and Prevention) MIC breakpoints (≥32 µg/mL) and 17 of these possessed ERG11 gene mutations. Screening prevented the spread of *C. auris* in Western Australia. MALDI-TOF allows rapid identification while WGS provides insights into the origin, clade, and resistance patterns.

Biography

Rutendo Mapengo (PhD, Clinical Microbiology and Infectious Diseases) is a Medical Scientist at PathWest's Microbiology Laboratory, specialising in pathogen genomics and infectious disease surveillance, applying whole-genome sequencing, molecular diagnostics, and high-throughput bioinformatics to investigate outbreaks and antimicrobial resistance. Rutendo uses phylogenetic interpretation to support public health responses. Previously, at Monash University and the National Institute for Communicable Diseases she contributed to large-scale genomic studies and the development of diagnostic approaches for emerging fungal pathogens.

Dr. Chris Mullaly

School of Medical, Molecular & Forensic Sciences, Murdoch University; Wesfarmers Centre for Vaccines & Infectious Diseases, The Kids Research Institute AU

Transforming neonatal sepsis diagnosis: developing rapid molecular approaches for early pathogen detection from low blood volumes

Abstract

Neonatal sepsis remains a major cause of morbidity and mortality worldwide, requiring rapid identification and treatment to improve clinical outcomes. Despite advances in neonatal care, diagnosis remains dependent on conventional blood culture, which can require 24–72 hours for pathogen identification. This challenge is amplified in neonates, where small blood volumes, low bacterial concentrations, and prior antibiotic exposure reduce diagnostic sensitivity. Consequently, many infants receive prolonged broad-spectrum antimicrobial therapy despite no confirmed infectious cause, contributing to antimicrobial resistance and disruption of the developing microbiome. To address these limitations, we developed a rapid diagnostic workflow combining short-duration culture enrichment with multiplex quantitative PCR (qPCR) for sensitive pathogen detection from low-volume blood samples. Using only 0.5 mL of blood, this combined culture–molecular approach demonstrated high analytical sensitivity and specificity, enabling detection of clinically relevant bacterial concentrations after only five hours of enrichment. The workflow provides pathogen identification within approximately 8–10 hours, representing a substantial improvement over current culture-based timelines. Rapid molecular detection of bloodstream infections has the potential to transform neonatal sepsis management by enabling earlier targeted antimicrobial therapy, improving antimicrobial stewardship, and reducing unnecessary antibiotic exposure. Ongoing clinical evaluation will determine the impact of this approach for improving diagnostic pathways and outcomes in vulnerable newborn populations.

Biography

Dr Christopher Mullally is an early-career molecular microbiologist at Murdoch University. His research focuses on developing innovative molecular approaches to improve the diagnosis and treatment of infectious diseases. His work combines pathogen genomics, microbiome analysis, and rapid diagnostic technologies, with a particular interest in neonatal sepsis and antimicrobial resistance.

ANZSCDB
Australia and New Zealand Society for Cell and Developmental Biology Inc.

Seminar Room 4

CHAIRs: Dr Liisa Hirvonen & Dr David Martino

	Keynote Presentation	
Dr Ben Dwyer	*The WA Organoid Innovation Hub: integrating patient-derived models and phenotypic screening to improve cancer therapy*	1.30
	Student Presentations	
Beatriz Da Silva Campos	*Magnetic isolation of CD26 subpopulations reveals distinct roles in RECELL®-mediated wound repair*	2.00
Dane Webster	*Mechanically Induced Paraspeckle Formation in Osteocyte Health*	2.15
Jacob Byrne	*ATR inhibition overrides radiation-induced G2 arrest to drive post-mitotic death in medulloblastoma cells*	2.30
Keea Inder-Smith	*Will Nano-Infrared Light the Way to Unravel Iron-Driven Lipid Alterations in Pancreatic Tissue?*	2.45
	Tea Break	3.00
	Invited Presentations	
Dr Ben Padman	*Volume electron microscopy reveals a new class of mitochondrial membrane contact site*	3.30
Dr Jasreen Kular	*Lysosomal Transporters: The Ins and Outs of Osteoclasts*	4.00

Dr Ben Dwyer
Curtin Medical Research Institute & Curtin University Liver Cancer Collaborative

The WA Organoid Innovation Hub: integrating patient-derived models and phenotypic screening to improve cancer therapy

Abstract
Patient-derived organoids (PDOs) are a powerful tool for cancer therapy development, faithfully recapitulating the genetic, histological and functional heterogeneity of the tumours from which they are derived. The Western Australian Organoid Innovation Hub at Curtin Medical Research Institute provides integrated, end-to-end high throughput drug development capabilities incorporating liquid handling, imaging and bioprinting. Automated liquid handling with the Revvity Zephyr G3 and Integra Viaflo384 systems enables reproducible, high-throughput assay establishment. High content imaging with the Revvity Operetta CLS system supports fluorescent and label-free phenotypic drug development in organoids, capturing thousands of parameters at single cell resolution enabling comprehensive, unbiased biological profiling. Precision bioprinting using the Inventia RASTRUM and Cellink BIO X6 systems, supports construction of fully defined, synthetic models that more accurately recreate the tumour microenvironment compared to traditional three-dimensional culture substrates. Together, these platforms support drug screening and repurposing, biomarker discovery and mechanistic investigation of therapeutic vulnerabilities in cancer. This talk will outline the capabilities available at the Hub and discuss how researchers can access these tools to advance their own cancer research using worked examples in liver and breast cancers

Biography
Dr Benjamin Dwyer is a Senior Research Fellow and Director of the WA Organoid Innovation Hub at Curtin Medical Research Institute at Curtin University. He received his PhD from the University of Western Australia in 2015 and undertook postdoctoral research at the University of Edinburgh (2015-2020), where he discovered key mechanisms controlling cholangiocarcinoma development and was part of a management team for macrophage therapy clinical trial in liver cirrhosis patients. He developed patented GMP-compatible methods to engineer and cryopreserve therapeutic macrophages, which are in clinical trial through University of Edinburgh spin-out company Resolution Therapeutics in the UK and Europe, of which he is a member of the founding team. He holds a FHRI WANMA Emerging Leaders Fellowship and leads a team developing patient-derived organoid models of primary liver cancer in the Liver Cancer Collaborative and RNA therapies for triple negative breast cancer in the Australian Centre for RNA Therapeutics in Cancer.

Magnetic isolation of CD26 subpopulations reveals distinct roles in RECELL®-mediated wound repair

Beatriz Da Silva Campos[1], Lucy Barrett[1], Fiona Wood[1,2], Mark Fear[1,2] , Andrew Stevenson[1]

[1]The University of Western Australia, [2]Fiona Wood Foundation

Introduction. RECELL® is an autologous cell suspension therapy that achieves wound healing comparable to split-thickness skin grafting while requiring substantially less donor skin, reducing donor-site morbidity. Despite these advantages, hypertrophic scarring remains a major challenge. CD26+ fibroblasts contribute to extracellular matrix deposition and fibrotic repair; however, their role within RECELL® suspensions remains unknown. Identifying whether CD26+ fibroblasts influence regenerative outcomes may enable targeted cell modulation. While conventional cell-sorting approaches could achieve selective depletion, their lengthy processing times limit intraoperative application. Magnetic-activated cell sorting (MACS) offers a rapid, clinically translatable alternative. **Problem Statement**. We investigated CD26 subpopulations in RECELL® mediated wound healing and assessed whether selective CD26+ modulation could influence regenerative outcomes in wound repair. **Procedures**. MACS separated RECELL® cell suspensions into CD26+ and CD26- populations, with sorting confirmed by flow cytometry. Functional differences were assessed using scratch wound and Scar-in-a-Jar assays. Therapeutic efficacy was evaluated in a two-month porcine full-thickness excisional wound model. Histological analyses included haematoxylin and eosin (H&E), Picrosirius Red (PSR), and immunofluorescence for CD31 and α-SMA to assess collagen architecture and vascular remodelling. **Results**. MACS generated distinct CD26+ and CD26- populations. *In vitro*, CD26+ fibroblasts exhibited slower wound closure and greater collagen deposition than CD26- cells. *In vivo*, collagen fibre composition and organisation were comparable across groups. However, CD26+ treatment demonstrated persistent vascular remodelling, characterised by increased endothelial cell abundance and a greater proportion of immature vessels, whereas CD26- treatment showed vascular resolution comparable to RECELL®. **Conclusions**. MACS is a feasible technique for modifying RECELL® cell suspensions. Although depletion of CD26+ cells did not improve healing beyond standard RECELL®, enrichment of CD26+ cells consistently promoted features associated with delayed wound remodelling. These findings demonstrate the influence of CD26+ fibroblasts in a clinically relevant porcine wound model and support further investigation of targeted cell selection to optimise regenerative therapies.

Mechanically Induced Paraspeckle Formation in Osteocyte Health

Dane Webster[1], Yu Suk Choi[1], Nathan Pavlos[2], Dominique Blache[4], Song Zhang[1], Luoyang Ding[4], & Archa Fox[1, 2, 3]

[1]School of Human Sciences, [2]School of Biomedical Science and [3] School of Agriculture and Environment, The University of Western Australia, [4]RNA Innovation Foundry (RIF), [5]Australian Centre for RNA Therapeutics in Cancer (ACRTC)

Master regulators of homeostatic bone health that influence the dynamic balance between bone formation and bone resorption are promising therapeutic targets for skeletal diseases. Paraspeckles, nuclear regulatory hubs built upon Neat1_2 lncRNA, are increasingly implicated in the molecular regulation of bone homeostasis. In osteoblasts, it has been established that paraspeckles can promote osteogenesis via RUNX2-related pathways when Neat1_2 expression is upregulated in mechanical stress. Currently, the relevance of paraspeckles in osteocyte health specifically is unknown, despite osteocytes being known as the primary mechanosensory and most-cell abundant in bone.This talk presents my PhD work on the role of paraspeckles in osteocyte health in two different mechanical stressors using MLO-Y4 cells: hypo-osmotic stress and GelMA based manipulation of matrix stiffness.When MLO-Y4 cells are grown in hypo-osmotic media, hereafter hypo-osmotic stress, we observe increases in cell and nuclear area, as well as significant growth arrest. In opposition to the established mechanosensitive, pro-osteogenic role in osteoblasts, Neat1_2 and paraspeckles are completely ablated in osteocytes within 2 hours of hypo-osmotic stress. At this 2-hour time point, we also observe a mechanically induced ~2-fold upregulation of Collagen and Osteocalcin mRNA, and a 0.3-fold reduction in Rankl mRNA; however, this is dynamically regulated to ~0.5-fold mRNA downregulation of all 3-bone gene expression at 24 hours of hypo-osmotic stress. To investigate paraspeckle function, we then overexpressed Neat1_2 to sustain paraspeckles under hypo-osmotic stress in which prevented the induction of bone gene mRNA response at the 2-hour time point. In contrast to hypo-osmotic stress, preliminary experiments with 3D GelMA suggest Neat1_2, paraspeckles, and bone gene mRNA levels all increase when osteocytes are seeded within stiffer matrix showing similarity to the paraspeckle role in osteoblasts. Overall, this work elucidates mechanical and osmolarity driven paraspeckle regulation and the effect of this on bone gene expression in MLO-Y4 osteocyte-like cells.

ATR inhibition overrides radiation-induced G2 arrest to drive post-mitotic death in medulloblastoma cells

Jacob Byrne [1], Annabel Short [1,2], Hetal Dholoria [1,3,4], Jacqueline Whitehouse [1,2], Meegan Howlett [1,2], Jessica Buck [1,2], Chloe Buckingham [1], Nicholas G Gottardo [1,3,4], Raelene Endersby [1,2].

[1]WA Comprehensive Kids Cancer Centre, The Kids Research Institute Australia, [2]Centre for Child Health Research, UWA, [3]Department of Paediatric and Adolescent Oncology/Haematology, Perth Children's Hospital, [4] Medical School – Paediatrics, The University of Western Australia

Survival outcomes for children with high-risk medulloblastoma remain poor, and existing treatments are inadequate. Ionizing radiation (IR) is a critical therapeutic component but is associated with debilitating late effects. High-throughput drug screening has identified DNA damage response kinases as promising therapeutic targets, with ATR inhibitor elimusertib demonstrating radiosensitizing activity in preclinical models. Defining the mechanistic basis of ATR inhibitor–mediated radiosensitization is essential for informing the safe advancement of this combination into clinical evaluation. Here, we aim to define how ATR inhibition alters cell-cycle progression and DNA damage responses following irradiation in medulloblastoma models to support the optimization and future clinical translation of this combination. Medulloblastoma cells were treated with ATR inhibitor elimusertib and IR, alone and in combination. Matrix-based combination assays with mathematical modelling quantified drug–radiation interactions. Cell-cycle kinetics and checkpoint integrity were examined using FUCCI live-cell time lapse imaging and flow cytometry to quantify S-phase arrest, mitotic entry, and apoptosis. DNA damage response signaling and apoptotic markers were evaluated by western blotting. ATR inhibition and IR exhibited robust synergy across wide dose ranges. FUCCI imaging revealed that IR induced a sustained G2 arrest (42.5h vs 15.8h in controls), with most cell death occurring prior to, and not after, mitosis. ATR inhibition accelerated S/G2 transit (9.9h) with limited cytotoxicity, particularly when administered in G1. In contrast, combined treatment abrogated the IR-induced G2 checkpoint (12.2h), promoting premature mitotic entry and resulting in substantial post-mitotic death in G1. These findings were validated by flow cytometry. Western blot analysis demonstrated suppression of ATR signaling, increased DNA damage accumulation, and elevated apoptotic marker expression. Taken together, these data indicate that ATR inhibition converts radiation-induced checkpoint arrest into a lethal mitotic transition, providing a mechanistic basis for its potent radiosensitizing activity and rationale for clinical evaluation.

Will Nano-Infrared Light the Way to Unravel Iron-Driven Lipid Alterations in Pancreatic Tissue?

Keea R Inder-Smith[1,2], Cyril DS Mamotte[1,2], Jitraporn Vongsvivut[3], Hendrik Vondracek[4], Gianfelice Cinque[4], David Paterson[3], Gorcin Kurejsepi[1,2,5], Mark J Hackett[2,6] and Ross M Graham[1,2]

[1]School of Diagnostic & Therapeutic Sciences, Curtin University, [2]Curtin Medical Research Institute, [3]ANSTO - Australian Synchrotron, [4]Diamond Light Source, Didcot, UK; [5]Ozgene Pty Ltd, [6]School of Molecular and Life Sciences, Curtin University

Obesity has reached epidemic proportions worldwide, and is associated with fat accumulation within pancreatic tissue. Dysregulated iron metabolism can damage tissues, and is hypothesised to cause fatty replacement of pancreatic tissue. This may see 'benign' pancreatic fat deposition progress to diabetes, steatopancreatitis, and possibly pancreatic carcinoma. As both iron and lipid dysregulation are implicated in pancreatic disease, clarifying how these two pathways interact is essential in advancing therapeutics for fatty pancreas related disease states. Mice were placed on either standard or high-fat diets with or without 2% carbonyl iron for six months. Iron loading and distribution were examined using x-ray fluorescence microscopy (XFM) at the Australian Synchrotron. Lipid analysis was performed using atomic force infrared microspectroscopy (AFM-IR) at the Diamond Light Source, UK. AFM-IR was used to target insulin producing β-cells within pancreatic islets, in a first attempt to optimise this technique for tissue analysis. XFM found pancreatic tissues load evenly with iron, no variation in irons distribution were found. AFM-IR data in combination with serial sections, aimed to identify subcellular organelle distribution. However, topology maps revealed little regarding organelles in sectioned tissue. Spectroscopy yielded expected and unexpected results. The addition of iron into high-fat diet decreased free fatty acids and lipid content and increased the carbonyl content of lipids present, consistent with previous data. Unexpectedly however, spectroscopy found high fat group total lipid content to be lower than control levels. This study has revealed current advantages and limitations of this nano scale method. Currently, identifying organelle targeted post-measurement is difficult. Results generated are robust, but come from unknown locations within a cell. AFM-IR is an exciting avenue that can be employed by biologists for tissue lipid analysis, and has the potential to give reliable and consistent results in the future with further optimisation.

Dr Ben Padman

The University of Western Australia Centre for Child Health Research (affiliated with the The Kids Research Institute Australia)

Volume electron microscopy reveals a new class of mitochondrial membrane contact site

Abstract

The structural form and biochemical functions of a mitochondrion are inseparable characteristics, maintaining a tenuous balance between mitochondrial homeostasis and pathophysiology. Our understanding of mitochondrial structure has come far since their initial description as “thread” (mitos-) “grains” (-chondros), but the primary research methods for studying their structure remain the same; microscopy. At the turn of the century, the biological research community was introduced to a new suite of three-dimensional (3D) electron microscopy methods, providing newfound capabilities that were deemed impossible just decades ago. This presentation will demonstrate the application of these 3D methods in an exploratory investigation into mitochondrial ultrastructure. Through a comprehensive analyses of 3D mitochondrial morphology and organelle interactions, the presentation will also reveal the existence of a previously undescribed category of membrane contact sites, called mitochondrial intrusions

Biography

Dr. Padman is a Research Fellow at The Kids Institute (TKI) and the University of Western Australia in Perth (Western Australia). A specialist in advanced imaging methods, Dr. Padman’s research seeks to elucidate the complex relationships between mitochondrial structure and function that underlie rare metabolic disorders. His current work focuses on exploring inter-organellar communication networks that modulate the mitochondrial interactome, by utilizing cutting-edge imaging techniques across the spectrum—from light and electron microscopy, to ion beams and x-rays. Believing that “a picture is worth a thousand words,” Dr. Padman is dedicated to making each image a revelation.

Dr Jasreen Kular

Bone Biology & Disease Laboratory, School of Biomedical Sciences, The University of Western Australia

Lysosomal Transporters: The Ins and Outs of Osteoclasts

Abstract

Osteoclasts are giant bone-digesting cells that harbour specialized organelles, 'secretory lysosomes,' that give rise to the ruffled border, the osteoclasts' bone-resorbing apparatus. Fusion of secretory lysosomes with the ruffled border equips its membrane folds with molecular machinery required to dissolve bone mineral and degrade the underlying organic matrix. Understanding the nature and contribution of secretory lysosome proteins holds potential to discover new regulators of osteoclast function and bone homeostasis. Using proteomics, we recently resolved the molecular landscape of osteoclast secretory lysosomes (*Ng, Ribet et al., Nat Commun, 2023*). To validate the osteoclast secretory lysosome proteome, we interrogated the functional contribution(s) of proteins predicted to participate in secretory lysosome membrane transport in osteoclasts. Here we report on another identified lysosome-resident transporter, major MFSD12, a little-characterised twelve transmembrane domain protein implicated in lysosomal import of the amino acid cysteine. We show that MFSD12/Mfsd12 is robustly expressed both in human and mouse osteoclasts during RANKL-induced differentiation, reflecting the cell's high intracellular demand for cysteine. To investigate the potential physiological role of Mfsd12 in osteoclasts and bone homeostasis, we generated global Mfsd12 knockout (Mfsd12KO) mice using CRISPR-Cas9. Although born at sub-Mendelian ratios and reduced in size, Mfsd12KO mice were viable to 30 weeks. MicroCT analysis revealed a dramatic low bone mass phenotype consistent with osteoporosis. Strikingly, gross inspection of Mfsd12KO viscera uncovered a multisystem disorder, including pale, reduced kidneys harbouring renal calculi, and abnormally enlarged livers. Ultrastructural analyses revealed a profound lysosomal storage disorder characterised by distorted lysosomes, some bearing crystals. Our findings unmask MFSD12 as an essential regulator of lysosome function whose deficiency produces a multisystem lysosomal storage disorder that affects bone homeostasis, kidneys and the liver, phenotypes that bear resemblance to clinical features observed in patients afflicted with the rare lysosomal storage disorder cystinosis.

Biography

Dr Kular is a Research Fellow in the Bone Biology and Disease Laboratory at UWA. Her research focuses on uncovering the molecular mechanisms that regulate secretory lysosomes and their role in controlling osteoclastic bone resorption.

The George Yeoh Award

Seminar Room 2

CHAIRS: Muhammad Ahmed & Dr Richard Allcock

	Keynote Presentation	
A/Professor Briardo Llorente	*Reshuffling the genome of the fastest-growing bacterium*	1.30
	Student Presentation	
Bac Dao1	*Isoform-aware deep learning enables transcriptome-wide prediction of RNA modifications from sequence and structure*	2.00
Callum Flanagan	*BAP1 is tumour suppressor gene that drives invasion in mesothelioma*	2.15
Joey Lye	*Disease model for Usher syndrome type IIA hearing loss*	2.30
Saskia Saville	*Developing a Programmable Cas13d RNA Therapeutic Platform for Hepatocellular Carcinoma*	2.45
	Tea Break	3.00
	Keynote Presentation	
Dr Jessica Kretzmann	*Encoding Structures: DNA Design and Biological Function*	3.30
Professor Ryan Lister	*Single-Cell Discovery and Characterisation of Epigenome Editing Tools*	4.00

Associate Professor Briardo Llorente

Australian Genome Foundry, Associate Investigator at the Australian Research Council Centre of Excellence in Synthetic Biology, Group Leader and Associate Professor, Macquarie University

Reshuffling the genome of the fastest-growing bacterium

Abstract

CGenome organization is thought to constrain bacterial physiology. Here, we establish a high-throughput system for generating extensive genome architectural diversity and apply it to probe physiology under altered genome organization. We engineered the fast-growing bacterium Vibrio natriegens to undergo stochastic duplications, translocations, inversions, and deletions across chromosome-scale regions, generating populations with a wide range of genome architectures. We find that multiple distinct genome architectures sustain stable physiology and, in some cases, enhanced growth, providing a striking example of phenotypic robustness. These results indicate that bacteria can tolerate substantial chromosomal restructuring more readily than previously appreciated and further suggest that genome reconfiguration may contribute to phenogenetic drift, enabling evolutionary exploration while preserving phenotype. Our work provides a framework for advancing the understanding and engineering of bacterial genomes.

Biography

Briardo is the Chief Scientist of the Australian Genome Foundry (AGF), an Associate Investigator at the Australian Research Council Centre of Excellence in Synthetic Biology (CoESB), and Group Leader and Associate Professor at Macquarie University in Sydney, Australia. Briardo is interested in tackling major challenges through synthetic biology while pushing the boundaries of genomic, metabolic, and intercellular engineering. His team engages in diverse research areas, ranging from re-engineering symbiosis and genomes to enhancing biomanufacturing and developing improved crops and medicines. Before joining the AGF and CoESB, Briardo was a CSIRO Synthetic Biology Future Science Fellow. He also served as a Marie Curie Fellow and a Juan de la Cierva Fellow at Barcelona's CRAG and worked as a visiting researcher at the University of Aarhus in Denmark and the Salk Institute in California. Briardo completed his PhD at the INGEBI and the University of Buenos Aires, Argentina

Isoform-aware deep learning enables transcriptome-wide prediction of RNA modifications from sequence and structure

Bac Dao[1], Marcell Szikszai[1,2], Max Ward[1,2], Archa Fox[1], Nikolay Shirokikh[1]

[1]Australian Centre for RNA Therapeutics in Cancer, School of Human Sciences and [2]School of Physics, Mathematics and Computing, The University of Western Australia,

The epi-transcriptome, comprising diverse RNA modifications, is a fundamental regulator of RNA metabolism and cellular function. Transcript fate and function is adjusted, often controlled, by its modification profile. A central question in synthetic biology and therapeutic applications and diagnostics of RNA is therefore how to predict the likelihood of presence of a modification in any given transcript of an arbitrary sequence. Although recent computational approaches have improved the prediction of RNA modification occurrence for certain types of RNA modifications, most existing tools rely on short-read sequencing-derived training data, where ambiguous read mapping limits the accurate assignment of modification sites to individual transcript isoforms. Moreover, conventional models typically address only a single modification type at once, largely overlook the regulatory role of RNA secondary structure, and fail to leverage the predictive power of modern large-scale RNA foundation models. Here, we present the first isoform-aware deep learning framework for transcriptome-wide prediction of RNA modifications in the human transcriptome using isoform-resolved ground-truth labels derived from nanopore direct RNA sequencing. By integrating sequence representations from best-performing RNA foundation model with explicit RNA secondary structure information, our framework captures both local and long-range regulatory determinants of RNA modification landscapes at the transcript level. To enhance generalizability across biological contexts, we incorporate cell-specific embeddings that enable the model to learn context-dependent epi-transcriptomic patterns across diverse cell types. Our framework achieves or surpasses state-of-the-art performance for m6A prediction across multiple cell lines. Furthermore, through a multi-task learning strategy, the model jointly learns multiple RNA modification types within a shared latent representation, enabling knowledge transfer from abundant modifications to rarer ones, which substantially improves predictive performance across all of the epi-transcriptome. Collectively, our work enables new, epi-transcriptome-aware approaches to the design of expressed synthetic RNA, empowering biotechnologists and therapists with more flexible and function-rich RNA solutions.

BAP1 is tumour suppressor gene that drives invasion in mesothelioma

Callum Flanagan[1,2], Alistair Nash[1,2], Ebony Rouse[1,3], Alec Redwood[1,3], Bruce Robinson[1,2,3,4], Jenette Creaney[1,2,3,4]

[1]National Centre for Asbestos Related Diseases and [2]Medical School, The University of Western Australia, [3]Institute for Respiratory Health, Perth, [4]Department of Respiratory Medicine, Sir Charles Gairdner Hospital

Pleural mesothelioma is an aggressive cancer arising from mesothelial cells lining the pleural cavity, where patients face dismal prognoses and have poor treatment options. Loss of BRCA1-associated protein-1 (BAP1) function is common, happening in approximately two out of three mesotheliomas. BAP1 is a tumour suppressor gene with numerous functions, including DNA repair, cell cycle control and epigenetic regulation of gene expression. In other cancers, BAP1 loss can promote or inhibit invasion while the effect in mesothelioma is unclear. Patients with mesothelioma whose tumours show BAP1 loss have a better prognosis than those with functional BAP1. BAP1 was hypothesised to promote tumour invasion through epigenetic regulation of gene expression. Therefore BAP1 loss was expected to reduce invasive capacity. Two cell lines with BAP1 knockout induced using CRISPR, six cell lines with functional BAP1 and four with BAP1 loss were utilised. Capacity for invasion was assessed using scratch wound migration, 2D transwell invasion and 3D spheroid invasion assays. Expression of invasion-related genes and proteins was assessed using RNA-seq and immunoblotting. Two repressive chromatin marks (H2AK119ub1 and H3K27me3) were mapped in the BAP1 wildtype and knockout cell lines to identify differentially enriched genes using cleavage under targets and release using nuclease (CUT&RUN). Cell lines with BAP1 loss migrated slower than those with functional BAP1. BAP1 knockout significantly reduced invasion in 2D transwell invasion assays and completely prevented invasion in 3D spheroid invasion assays. Cell lines with functional BAP1 showed higher expression of invasion related gene-signatures and proteins compared to BAP1 loss cell lines. CUT&RUN demonstrated widespread accumulation of repressive marks around invasion-related genes in the BAP1 knockout cell lines. Despite playing an important role in tumour suppression, BAP1 has been demonstrated to promote invasion in mesothelioma, likely contributing to the poor clinical outcomes associated with BAP1 retention.

Disease model for Usher syndrome type IIA hearing loss

Joey Lye[1,2], Fiona K Leith[1,2], Josh CW Gacer[1,2], Sharon L Redmond[1,2], Samuel McLenachan[2,3], Fred K Chen[2,3,4], Marcus D Atlas[1,2,5], Elaine YM Wong[1,2,5]

[1] Ear Science Institute Australia, [2]Centre for Ear Sciences, Medical School, The University of Western Australia, [3]Lions Eye Institute, [4]Royal Perth Hospital, , [5]Curtin Medical School, Curtin University

Hearing loss is a disabling sensory disorder and it has been predicted to affect over 2.5 billion people worldwide by 2050. Usher syndrome (USH) is the most common autosomal recessive inherited disease that causes both blindness and deafness. *USH2A* is one of the USH causative genes and is responsible for up to 50% of all USH cases. Usherin is a large transmembrane protein encoded by the *USH2A* gene and this protein is essential for maintaining the stereocilia integrity in developing cochlear hair cells. Mutations are found along the entire *USH2A* gene and USH remains incurable with no treatment available. In this study, in collaboration with Lions Eye Institute, USH2A patient-derived induced pluripotent stem cells (iPSCs) were used to generate inner ear organoids harbouring biallelic mutations c.949C>A and c.1256G>T to model the USH2A disease mechanism in the inner ear. Inner ear organoids containing sensory hair cells were generated through a controlled stepwise differentiation method. We performed gene expression and immunofluorescence analysis of early otic cell fate markers to examine the effect of USH2A mutations during inner ear development. Our results demonstrated that USH2A inner ear organoids were able to form otic vesicles, a sensory epithelium that gives rise to the inner ear during embryogenesis. Furthermore, hair cell and neuronal markers were also expressed in iPSC-derived inner ear organoids, which may provide insights the role of *USH2A* at later developmental stages. This preclinical model will be used for high-throughput drug screening and to develop gene therapeutic approaches to correct *USH2A* mutations for hearing restoration.

Developing a Programmable Cas13d RNA Therapeutic Platform for Hepatocellular Carcinoma

Saskia Saville[1], Nina Tirnitz-Parker[1], Rodrigo Carlessi[1]

[1]Curtin Medical Research Institute, School of Diagnostic and Therapeutic Sciences, Curtin University

Hepatocellular carcinoma (HCC) remains a major cause of cancer mortality, particularly for patients who present beyond curative surgical options. Although systemic and immunotherapy combinations have improved treatment, response rates remain below 30% and resistance is common. Many important HCC drivers, including WNT/beta-catenin and RAS/MAPK signalling, remain difficult to suppress with conventional drugs. HCC is well suited to RNA therapeutics because liver-directed lipid nanoparticle (LNP) delivery is feasible and clinically validated through many approved RNA medicines. We aim to develop a programmable, fully RNA-based CRISPR Cas13d therapeutic platform that can be rapidly redirected to different HCC transcripts by changing the guide RNA. Cas13d is an RNA-guided ribonuclease that recognises and degrades target RNA. Our approach delivers Cas13d as transient mRNA with synthetic guide RNAs using LNPs. This platform is being developed in two therapeutic modes: precision knockdown of oncogenic transcripts, and tumour-selective cell killing through collateral RNA cleavage triggered by highly expressed HCC-enriched RNAs. We first optimised the RNA and lipid nanoparticle delivery conditions using a tuneable destabilised GFP (d2EGFP) reporter system and live-cell imaging. This enabled Cas13 targeting dynamics to be assessed across different levels of target expression using d2EGFP knockdown as a readout. Notably, targeting highly expressed d2EGFP transcripts induced stronger collateral RNA cleavage activity and greater cell death than targeting lowly expressed d2EGFP transcripts. We then applied the system to an array of HCC-relevant targets in a panel of HCC cell lines. Several individual guide RNAs and combinations reduced proliferation and induced cell death, with viability reduced by up to 88% compared with scrambled guide controls. These findings support Cas13d as a flexible and programmable RNA therapeutic platform for HCC. By combining hepatocyte-directed LNP delivery with rapidly interchangeable guide RNAs, this approach addresses a key translational barrier for personalised RNA oncology.

Invited Presentation

Dr Jessica Kretzmann

School of Molecular Sciences, The University of Western Australia

Encoding Structures: DNA Design and Biological Function

Abstract

DNA origami has enabled the construction of DNA nanostructures with unprecedented structural control over size, shape and surface functionality. Using one long strand of ssDNA, and hundreds of short DNA oligos, user-defined complex structures can be formed via sequence-dependent self-assembly in a one-pot annealing reaction. Shapes such as cubes, bricks, cylinders and triangles can be formed, all the way to programmed higher ordered assemblies, molecular rotors, transport pores, and logic gates. Traditionally, DNA origami has utilized bacteriophage-derived sequences as the single-stranded DNA scaffold strand. However, it is now possible to create complex, customizable scaffolds, and even encode for, and fold, mammalian genes into DNA origami. So, what other functions can we encode for?

Herein, recent work in the development of DNA origami for biotechnology applications will be discussed, such as the genetic encoding of DNA origami for mammalian gene expression, and the creation of tools to probe the intracellular fate and functionality of DNA origami objects

Biography

Jessica completed her PhD degree in Chemistry from the University of Western Australia, spending part of her doctoral studies as a Fulbright Scholar at the University of Massachusetts, Amherst (2018). Her PhD focused on the design and evaluation of new materials for the delivery of gene therapies. From there, Jessica was an Alexander von Humboldt Postdoctoral Fellow at the Technical University of Munich, where she worked on the design and implementation of DNA origami for biotechnological applications (2020 – 2022) with Prof Hendrik Dietz.

Jessica returned to UWA through a Forrest Fellowship in late 2022, and now as a DECRA and NHMRC Fellow is establishing her group in DNA nanotechnology.

Professor Ryan Lister

Harry Perkins Institute of Medical Reserch & The University of Western Australia

Single-cell discovery and characterisation of epigenome editing tools

Abstract

Precise manipulation of the epigenome holds great promise for controlling gene expression and cell identity, yet current epigenome editing tools are frequently limited by transient effects, variable efficacy, and poorly understood context dependency. Overcoming these limitations requires both the discovery of more effective epigenetic effectors and a systematic understanding of how genomic and chromatin context shapes their activity. We have developed a massively parallel single-cell screening platform that enables the interrogation of hundreds of epigenome effectors at endogenous target loci across multiple human cell types. By reading out transcriptional consequences at single-cell resolution, we identify potent effectors for gene activation and repression and assess their efficacy and generalizability at scale. Through comprehensive epigenomic characterisation of the chromatin reconfiguration induced by effectors, across diverse genomic and chromatin contexts, we aim to identify the principles governing where and when epigenome editing is effective. These insights will further inform how multiple epigenomic layers coordinate to regulate transcription, aiding the design of flexible and robust editing strategies tailored to the endogenous chromatin state of a given cell type. This work provides a foundation for the rational deployment of epigenome editing tools in gene regulation, cell engineering, and diseaseBiography

Biography

Ryan Lister leads a research group investigating the epigenome and cell identity, at the University of Western Australia and the Harry Perkins Institute of Medical Research. After receiving his PhD from UWA in 2005, Ryan undertook postdoctoral studies at The Salk Institute for Biological Studies, where he developed and applied new techniques to map the epigenome and transcriptome. Having returned to UWA in 2012, Ryan's laboratory is focused upon understanding the role of the epigenome in regulation of cell identity, and developing molecular tools to precisely edit the epigenome and transcriptional activity to control cell state and functions.

The Western Australian Biodiversity Science Institute

LEADING KNOWLEDGE COLLABORATION
ENABLING SMARTER DECISIONS

Seminar Room 1

CHAIR: Professor Liz Watkin

	Keynote Presentation	
A/Professor Stephanie Godfrey	*Parasites in wildlife - a neglected aspect of conservation biology*	1.30
	Student Presentations	
Cameron Dodd	*Taxonomy and Conservation Genomics of the Kultarrs (Antechinomys spp.)*	2.00
Heather Russell	*Insights from our spiny relatives: using spiny mice fibroblasts as a blueprint for skin regeneration*	2.15
Pia Dethlefsen	*Assessing the effects of fragment length on detection success of airborne eDNA*	2.30
Shengyao Lin	*Population Genomics and Landscape Genetics of Uperoleia daviesae (Anura: Myobatrachidae) Highlights the Vulnerability of Naturally Fragmented Short-Range Endemics to Urban Development*	2.45
	Tea Break	3.00
	Invited Presentations	
Dr Alexandre Siqueira	*The rise and fall of the world's greatest marine biodiversity hotspot*	3.30
A/Professor Holly Kirk	*The devil is in the details: translating ecological knowledge for nature positive cities*	4.00

Associate Professor Stephanie Godfrey

Department of Zoology, University of Otago

Parasites in wildlife - a neglected aspect of conservation biology

Abstract

Parasites are a dominant component of biodiversity and a major force in ecological and evolutionary processes - yet they remain neglected in conservation management. But this tune is changing. Over the last ten years, there has been a surge in publications on this research topic, the IUCN Parasite Specialist Group has been established, and a growing number of threat-listings and recovery programmes for parasite species are underway. Despite this heightened interest in parasite conservation - we still have very little understanding of how to effectively conserve parasites, and how to implement this in a landscape of limited conservation funding and negative public perception. In this talk, I will address the progress that has been made in this field, with reference to case studies that build our understanding of how to incorporate parasites into conservation planning.

Biography

Stephanie Godfrey is an Associate Professor in the Department of Zoology at the University of Otago. Her parasitological interests centre on two key themes; (1) the role of animal behaviour in parasite transmission, particularly using social network models to understand contact patterns in wildlife populations; and (2) the conservation biology of native and endemic parasites, with a focus on recognising parasites as integral components of biodiversity. Stephanie's work challenges traditional views of parasites solely as threats, instead highlighting their ecological importance and the need to consider them in conservation planning. Her research combines field ecology, behavioural modelling, and conservation science, contributing to a more nuanced understanding of host-parasite relationships in Australasia's unique fauna.

Taxonomy and Conservation Genomics of the Kultarrs (*Antechinomys* spp.)

Cameron S. Dodd[1,2], Linette S. Umbrello[2], Andrew M. Baker[3,4], Kenny J. Travouillon[2], Michael Westerman[5], Renee A. Catullo[1,2]

[1]University of Western Australia, [2]Western Australian Museum, [3]Queensland University of Technology, [4]Queensland Museum, [5]La Trobe University

Accurate and thorough taxonomy is an important component of the conservation of wild populations as it allows researchers to better prioritize management practices and allocate resources. Unfortunately, despite extensive taxonomic work to date, many species remain undescribed, even in well-studied groups like mammals. This is particularly true in Australia where many small mammals live in isolated areas and occur in very low densities, making them incredibly difficult to study. One particularly diverse group is the carnivorous marsupials (Family Dasyuridae) which has seen an almost 50% increase in the number of species since 2000. Here we used an integrative approach to investigate the taxonomy of the genus *Antechinomys*, a group of small dasyurid marsupials which superficially resemble hopping-mice. Using genome wide genomic data and museum specimens, we document two previously unrecognized species of kultarr, found across arid Australia. We also identify an additional population in Southwest WA which has not been caught alive in 50 years and may represent a fourth taxon. In addition, we present the first draft genome for this genus and use it to investigate the genomic diversity and richness in the now three species. We show a substantial decline in allelic diversity in one semi-arid species, *A. laniger*, as well as high, but differently distributed, diversity in the two arid-zone species: *A. auritus* and *A. spenceri*. Our results show the importance of comprehensive taxonomic assessments of all modern mammal groups and highlights the utility of using genomic data to assess the population health of species which are hard to study in the field.

Insights from our spiny relatives: using spiny mice fibroblasts as a blueprint for skin regeneration

Heather Russell, Robyn Allen, Runshi Zheng, John Henderson, Mike Adam, Zahra Massoud, Ahmed Muhammad, Ashley Seifert, Fiona M. Wood, Sofia Ferreira-Gonzalez, Mark W. Fear and Andrew W. Stevenson

[1]The University of Western Australia, [2]Fiona Wood Foundation, [3]University of Edinburgh, [4]University of Kentucky

Scarring is a major challenge following injuries such as burns. Unlike healthy skin, scar tissue does not restore function and can have lasting physical and psychological impacts on patients. Despite advances in wound care, there are currently no treatments able to prevent scarring. This is largely due to our lack of understanding of true regeneration and the mechanism involved. Spiny Mice (spp *Acomys*) are remarkable mammals capable of regenerating their skin and several internal organs (incl. heart, spinal cord, skeletal muscle) without forming scars, even in adulthood. Due to their close phylogeny to humans compared to other regenerative vertebrates, they pose unprecedented potential uncover regenerative mechanisms that could improve human wound healing. Key cellular derived or repair and consequently scarring. are dermal fibroblasts know to over produce collagen in response to injury. Emerging evidence suggests that fibroblasts exist as distinct subpopulations with specialised roles in either fibrosis or regeneration but retain plasticity across these subtypes. In this study we aim to identify regenerative fibroblast states in the spiny mouse that could serve as a blueprint for promoting regenerative healing in humans. We characterise human, pig, mouse (classical models of skin fibrosis) and spiny mouse dermal fibroblast to elucidate differences in phenotype and behaviour across these scarring and regenerative species. We found that all species contain diverse fibroblast populations; however, the spiny mouse possesses a greater proportion of regeneration-associated fibroblasts rather than a unique fibroblast subtype. Remarkably, spiny mouse fibroblasts also displayed spontaneous lineage plasticity in culture, differentiating into neuron-, cartilage-, and epithelial-like cells through dedifferentiation to progenitor-like intermediate state not observed in the other species. Understanding the mechanisms that enable this exceptional cellular plasticity could identify new therapeutic targets to shift human fibroblasts toward regenerative states, ultimately reducing scarring and improving skin function after injury.

Assessing the effects of fragment length on detection success of airborne eDNA

Pia Dethlefsen[1], Joshua Newton[1], Angus D'Arcy Lawrie[1], Marina de Oliveira[1]

[1]Trace and Environmental DNA Laboratory (TrEnD), School of Molecular and Life Sciences, Curtin University

Airborne environmental DNA (eDNA) offers a non-invasive approach for monitoring elusive terrestrial fauna, but detection is constrained by rapid degradation caused by ultraviolet radiation, oxidation, desiccation and atmospheric dispersal. This project investigates how target fragment length influences the detectability of airborne eDNA using the greater bilby (Macrotis lagotis), an elusive and conservation-significant marsupial, as a model species. Six species-specific mitochondrial assays were developed using one forward primer and six reverse primers designed to generate amplicons of 142, 306, 394, 696, 1218 and 1,915 base pairs. Candidate primers were assessed for melting temperature, GC content, secondary structure and specificity against mitogenomes for 65 marsupials occupying a similar distribution. Airborne eDNA was collected from a bilby enclosure at Perth Zoo using five fan-assisted filter boxes, each containing two sterilized filters and operating for 24 hours. Ten enclosure surface swabs were also collected to provide a comparison matrix. DNA was extracted from the environmental samples, and assay validation was undertaken using synthetic modified bilby DNA, museum tissue in a quantitative PCR platform. Primer design, field sampling and DNA extraction were completed successfully, producing a controlled sample set for comparing amplification and quantification across six fragment lengths. The nested assay design enables direct testing of the prediction that shorter DNA fragments are detected more consistently than longer fragments in airborne samples. This work will improve understanding of airborne DNA degradation and inform assay design and sampling strategies for non-invasive monitoring of bilbies and other difficult-to-detect terrestrial species.

Population Genomics and Landscape Genetics of *Uperoleia daviesae* (Anura: Myobatrachidae) Highlights the Vulnerability of Naturally Fragmented Short-Range Endemics to Urban Development

Shengyao Lin[1], Peter J. McDonald[2], Alistair Stewart[2], Carolyn J. Hogg[3], Luke W. Silver[3], Sam C. Banks[4], Graeme R. Gillespie[2], Raphael K. Didham[1], and Renee A. Catullo[1]

[1]School of Biological Sciences, The University of Western Australia, Department of Lands, Planning and Environment, Northern Territory Government, [3]School of Life and Environmental Sciences, The University of Sydney, [4]Research Institute for the Environment and Livelihoods, Charles Darwin University

Urbanisation and land use change threaten short-range endemic amphibians. Uperoleia daviesae, the Howard River toadlet, is a threatened frog species endemic to sandsheet heath, a unique, naturally patchy mosaic of habitats near Darwin in Australia's Northern Territory. We generated a chromosome-level genome assembly and performed genome-wide SNP analyses using data from 115 individuals across 15 sites to assess dispersal patterns, genetic diversity, anthropogenic impacts, and conservation targets. Our findings reveal a history of past connectivity, followed by recent genetic subdivision, partly due to urban expansion. Regions 2 (north of Girraween and Humpty Doo) and 3 (south of Bees Creek and Lloyd Creek) contain most of the genetic diversity within the species. The two isolated populations, Region 1 (north of Howard Springs) and Region 4 (near Wickham and Weddell), each harbour unique alleles. We recommend treating local populations as a metapopulation and developing targeted conservation actions in each region. In-situ conservation actions, such as establishing protected areas or translocation, should be considered to maintain gene flow and genetic diversity. These findings provide a foundation for evidence-based management of U. daviesae and contribute to broader discussions on the conservation of short-range endemic species in increasingly urbanised landscapes.

Invited Presentation

Dr Alexandre Siqueira

ARC DECRA and Vice-Chancellor's Research Fellow, Edith Cowan University

The rise and fall of the world's greatest marine biodiversity hotspot

Abstract

Habitat condition and area shape global species distributions, with shallow-water reefs hosting a disproportional share of marine biodiversity. Although reef area is a well-established predictor of marine species richness, its historical context is less well understood. In this talk, I will reveal how the rise of tropical marine biodiversity is closely tied to reef expansion in space and time. During the Early/Mid Miocene (23 to 11.6 million years ago), Indo-Pacific reefs reached unprecedented size and thickness, surpassing any reef systems in the past 66 million years. These massive reefs, likely driven by unique environmental, biotic, and tectonic conditions, fostered the expanding diversity and functional evolution of marine fish and coral assemblages. These findings underscore the importance of historical reef contexts and the implications of ongoing reef losses for tropical marine biodiversity.

Biography

Alexandre Siqueira is a marine biologist fascinated by the processes shaping coral reef biodiversity through time. His research integrates phylogenetics, palaeontology, and ecology to identify the evolutionary and ecological mechanisms underpinning reef fish diversity and ecosystem functions. He completed his PhD at James Cook University in 2021, focusing on the trophic evolution, macroecology, and biogeography of coral reef fishes. Following 3.5 years as a Postdoctoral Fellow at the Reef Function Hub, he is now an ARC DECRA and Vice-Chancellor's Research Fellow at Edith Cowan University, investigating the characteristics of ancient reefs that influenced fish productivity

Dr Holly Kirk
Curtin University

The devil is in the details: translating ecological knowledge for nature positive cities

Abstract

The intersection of ecological theory with urban planning and design has great potential for the delivery of biodiversity conservation benefits during urban development. Achieving healthy urban ecosystems that support diverse flora and fauna means designing landscapes that provide for multiple ecological processes and account for the complexity of resource provision for multiple species. Designing for biodiversity from the beginning of the urban development process helps ensure that key requirements for nature can be included synergistically while meeting other development needs. The Biodiversity Sensitive Urban Design (BSUD) framework provides a method for improving the inclusion of ecological knowledge within urban design. The flexible framework can be applied across different development types, translating ecological needs into urban design actions using a set of five key principles. In this session I will explore the intersection of ecological theory with urban design and planning, reflecting on my applied research work on a range of urban case studies. I will draw on how we can use spatial ecology to identify key areas to protect and enhance habitat for biodiversity conservation in cities, explore how mapping biodiversity can help with target setting, and demonstrate ways to use ecological theory to enhance landscape design. I'll also reflect the need for properly planned monitoring programs to help measure the impact of on-ground action, including ways to empower the local community in learning about the everyday nature around them, while collecting valuable urban biodiversity data.

Biography

Dr Holly Kirk is Associate Professor of Urban Biodiversity, researching and teaching in the School of Design and the Built Environment at Curtin University. She specialises in the application of ecology to urban planning and design. An expert in spatial modelling and animal movement, for the last six years Dr Kirk has been using this skill to plan cities that support and enhance urban nature. She works closely with industry and government, understanding how Biodiversity Sensitive Urban Design (BSUD) can be implemented in different urban developments: from greenfield residential sites to high-rise inner-city buildings.

CBSM

Centre for Crop and Disease Management

GRDC GRAINS RESEARCH & DEVELOPMENT CORPORATION

Curtin University

Formal Dining Room

CHAIR: Professor Jacqueline Batley

	Keynote Presentation	
Professor Lee Hickey	*Using AI to develop future crops*	1.30
	Student Presentations	
Leon Lenzo	*Fair-Weather Friends: Asymmetric Priority Effects Challenge Host Immunity in Polymicrobial Foliar Disease of Wheat*	2.00
Naomi Gray	*Pangenomics Provides Insight into Global Fungicide Resistance*	2.15
Clementine Wu	*Tracing the B-genome origin of Rlm6-mediated blackleg resistance in Brassica napus*	2.30
Yutathkarn Coles	*Transposon-Driven Genome Remodelling Underpins Adaptation of Pyrenophora teres f. teres*	2.45
	Tea Break	3.00
	New Investigator Presentations	
Dr Farley Kwok van der Giezen	*Understanding and engineering RNA editing in energy transducing organelles*	3.30
Dr Bryn Funnekotter	*Frozen for the Future: The Science Behind Kings Park's Cryopreservation Biobank*	3.45

Profesor Lee Hickey

ARC Future Fellow, ARC Training Centre in Predictive Breeding, University of Queensland

Using AI to develop future crops

Abstract
Modern crop breeding generates vast amounts of genomic, phenotypic, and environmental data, yet turning that data into better varieties faster remains a central challenge. This talk explores how artificial intelligence and machine learning are being integrated into predictive breeding pipelines at The University of Queensland to accelerate genetic gain. Drawing on our work in speed breeding, genomic prediction, and haplotype based selection, I'll show how AI can help breeders predict performance, prioritise crosses, and stack favourable traits across cereals, legumes, and other crops. Crucially, I'll argue that the most powerful gains come not from data alone, but from grounding these models in biological knowledge, embedding what we understand about crop physiology, genetics, and gene function so that predictions are both accurate and interpretable. From forecasting disease resistance to optimising selection decisions, these tools are reshaping how we design the crops of the future, offering a practical pathway to more resilient, productive, and climate ready agriculture.

Biography
Professor Lee Hickey is a plant breeder, crop geneticist, and ARC Future Fellow at the University of Queensland, where he directs the ARC Training Centre in Predictive Breeding. Lee is one of the pioneers of speed breeding — a technique that fast-tracks the development of new crop varieties and is now used by breeding programs worldwide. His work integrates AI, genomics, and genome editing to build better crops for farmers. He is equally passionate about nurturing the next generation of plant scientists, currently mentoring 18 PhD students with many former graduates now working at leading breeding programs and research institutes around the world. His research has featured in the BBC, National Geographic, and the New York Times, and is backed by more than 100 scientific publications.

Fair-Weather Friends: Asymmetric Priority Effects Challenge Host Immunity in Polymicrobial Foliar Disease of Wheat

Leon Lenzo[1], Evan John[2], Jason Bradley[3], Geoff Thomas[3], Dion Bennett[1] and Kar-Chun Tan[1]

[1]Centre for Crop and Disease Management, Curtin University, [2]Department of Plant Pathology, INRES, University of Bonn, [3]Department of Primary Industries and Regional Development, WA

In plant and animal pathosystems, the host is commonly challenged by more than one pathogen simultaneously. Most pathosystem studies involve a single pathogen, but where multiple pathogens have been studied, synergy, antagonism and priority effects have frequently been detected, interactions which threaten persistent host immunity. *Parastagonospora nodorum* and *Pyrenophora tritici-repentis* cause septoria nodorum blotch and tan spot of wheat, respectively. Field co-infection between these fungal pathogens is widely established, however interaction dynamics remain poorly understood. Using a newly developed digital PCR method, we studied co-infection in naturally infected wheat fields, inoculated field trials and controlled environment artificial infections. Natural infection revealed a clear majority of symptomatic infections involved both pathogens, with host leaves resistant to both pathogens showing significantly higher disease and pathogen biomass under co-infection compared to single species. Controlled inoculum field experiments showed *P. tritici-repentis* priority was most similar to natural and controlled mixed infection, suggesting *P. tritici-repentis* priority occurs in natural systems. Laboratory experiments showed that consecutive infection where *P. tritici-repentis* was followed by *P. nodorum* in a resistant host resulted in a significant increase in disease and biomass for both pathogens. When *P. nodorum* established on the host first, *P. tritici-repentis* colonisation was highly reduced regardless of host resistance in both controlled field studies and laboratory experiments. To our knowledge this is the first description of asymmetric priority effects overcoming host resistance in a plant pathosystem. Resistance breeding strategies evaluating single pathogen challenges may inadvertently select for cultivars vulnerable to sequential co-infection, necessitating integrated disease complex approaches for durable resistance development.

Pangenomics Provides Insight into Global Fungicide Resistance

Naomi Gray[1], Chala Turo[1], Jackson Greene[1], Mohitul Hossain[1], Pavel Misiun[1], Kristina Gagalova[1,2], Huyen Phan[1], Manisha Shankar[3], Hossein Golzar[3], Romain Valade[4], Kar-Chun Tan[1], Keelan Hasset[1], Elzette Palmiero[1], Pao Theen See[1], Simon Elwood[1], Lincoln Harper[1], Wesley Mair[1], Richard Oliver[1], Fran Lopez-Ruiz[1], James Hane[1]

[1]Centre for Crop Disease Management (CCDM), Curtin University, [2]Analytics for the Australian Grains Industry (AAGI), [3] Department of Primary Industries & Regional Development, [4]Arvalis

Fungal pathogens comprise some of the most destructive diseases on crops globally, causing yield losses in major crops capable of feeding over 600 million people. One of the main methods of disease control is through the application of various fungicides. However, this control can be lost due by the emergence of fungicide resistance mutations among pathogen populations. Fungal pathogens possess a high degree of genome plasticity which enables them to rapidly adapt to different fungicides. Due to their genome variability, it is advantageous to study multiple individuals from a species as a collective pangenome. This allows the monitoring of crop diseases at a population level. We applied a tool called FRAST (Fungicide-Resistance Allele Screening Tool), developed at the Centre for Crop and Disease Management, to a global pangenome collection of 18 major cereal pathogens. In total, 2422 genomes across Africa, Asia, Australia, Europe, the Middle East, North America, and South America were surveyed. Our results revealed that mutations that confer resistance to DMIs (a class of fungicide, also known as azoles) were present in all species and geographical regions surveyed in this study. Australia notably possessed the highest frequency of fungicide resistance mutations. The fungal pathogens *Parastagonospora nodorum*, *Pyrenophora teres. f. teres,* and *Pyrenophora tritici-repentis* possessed intense levels of fungicide resistance mutations globally, with some resistance mutations present in 100% of the population. The DMI class of fungicides is a relatively cheap chemical and is therefore often prophylactically applied. These results reflect the economically motivated decisions that growers make around the world to minimise cost and disease severity with incomplete knowledge of the local pathogen resistance profiles. This survey demonstrates the importance of surveying pathogen population genomes to enable growers to make educated disease management decisions.

Tracing the B-genome origin of *Rlm6*-mediated blackleg resistance in *Brassica napus*

Clementine Wu[1], Junrey Amas[1], Nur Shuhadah Mohd Saad[1], Angela Van de Wouw[2], David Edwards[1,3], and Jacqueline Batley[1,3]

[1]School of Biological Science, The University of Western Australia, [2]Faculty of Science, University of Melbourne, [3]UWA Institute of Agriculture

Blackleg, caused by the fungal pathogen *Leptosphaeria maculans*, is a major disease in canola (*Brassica napus,* AACC) responsible for substantial yield losses. The deployment of major resistance genes remains an important strategy for managing the disease. One of these genes is *Rlm6*, which mediates recognition of the corresponding pathogen avirulence gene (*AvrLm6*). Previous studies indicate that *Rlm6* was introduced from the B-genome of the related species *Brassica juncea* (AABB) through interspecific hybridisation. However, the causal gene remains unresolved. In this study, phenotypic and genomic analyses were integrated to identify a candidate gene for *Rlm6* in *B. napus* and investigate its putative B-genome origin. Selected *B. napus* lines were phenotyped following inoculation with *L. maculans* for resistant and susceptible responses. Genome-wide association studies identified a candidate gene, designated as *pg_16,* which showed sequence segregation between resistant and susceptible lines. Further comparative analysis using the *Brassica* 90K SNP array identified a distinct genomic region in which resistant *B. napus* lines shared B genome-derived SNPs, which were absent in susceptible lines, suggesting a *B. juncea* introgression. Time-point experiments show that *pg_16* is highly activated at 8 days post-inoculation (DPI), providing key insights into its expression kinetics. Together, the phenotypic and genotypic analyses support the hypothesis that the *Rlm6*-associated region in resistant *B. napus* germplasm likely originated from the B-genome of *B. juncea*. Further characterisation of the introgressed region and functional validation of *pg_16* will be required to confirm the molecular identity of *Rlm6*. This study highlights the contribution of interspecific introgression to blackleg resistance and the broader value of related *Brassica* species as sources of resistance diversity for canola improvement

Transposon-Driven Genome Remodelling Underpins Adaptation of *Pyrenophora teres* f. *teres*

Yutathkarn Coles[1], Jade Davis[1], Ciara Gifford[1], Lilian Sanglard[1], Mark Gibberd[1], Fatima Naim[1]

[1]Centre for Crop and Disease Management, Curtin University

Fungal plant pathogens can rapidly adapt to environmental conditions including host resistance, owing to diverse and independent genomic mechanisms that also shape pathogen lifestyle. Net form net blotch (NFNB), caused by *Pyrenophora teres* f. *teres*, is a major foliar disease of barley. A major NFNB epidemic in 2022 resulted in the widespread failure of an elite barley cultivar. Despite a wealth of knowledge in fungal evolutionary mechanisms, the genetic basis of the aggressive virulence observed during this epidemic was unknown. To understand how *P. teres* f. *teres* overcame host resistance over a short period, we combined long-read genome assemblies and disease phenotyping across nine Western Australian isolates and compared these to other publicly available genomes dating back four decades. Comparative genomics between isolates revealed substantial transposon-mediated genome remodelling in recent isolates, evidenced by an expanded transposon repertoire and co-localisation with candidate effectors. We found that previously uncharacterised *Starship* large mobile elements have proliferated across the *Pyrenophora* genus. These Starships carry candidate effectors between geographically distinct *P. teres* f. *teres* populations and were enriched in contemporary isolates, with one virulence-rich element sharing a genetic origin with *P. tritici-repentis*. Simultaneously, a *hAT-Tnp dimer* transposon-flanked architecture was identified within a chromosome 3 subtelomeric virulence quantitative trait locus. This region encoded various putative effectors, manually curated using RNA sequencing data, that were embedded within repeat-rich islands and enriched in contemporary isolates. The unique and dynamic genomic architecture of *P. teres* f. *teres* has undergone substantial remodelling, permitting widespread acquisition of genes with effector-like properties and we hypothesise these features as the most likely cause of the widespread change in pathogenicity of NFNB on recent barley cultivars.

Invited Presentation

Dr Farley Kwok van der Giezen

Research Associate, ARC Centre of Excellence in Plants for Space

Understanding and engineering RNA editing in energy transducing organelles

Abstract

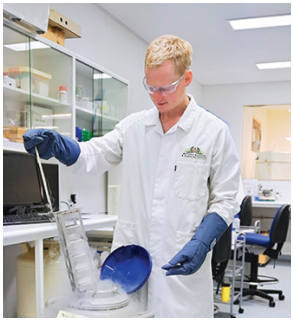
The plastid and the mitochondria are energy transducing organelles. They are descendants of free living cyano- and α-proteobacteria whose cellular activities provide the foundation for all aerobic life on Earth. Despite billions of years of co-evolution with the nucleus, they have retained minimal versions of their bacterial-like genomes. The organelle genomes encode highly conserved core components of the photosynthetic and respiration machineries. Plants have evolved a curious mechanism to fix mutations in the organelle genomes when they arise. Rather than fixing mutations in their DNA, they fix mutations in their RNA. This is a process we call RNA editing. RNA editing in plants is catalysed by a large family of RNA binding proteins called pentatricopeptide repeat (PPR) proteins. In plants, PPR proteins recognise and bind to specific sequences and catalyse the conversion of cytidine to uridine (C-to-U), and in seedless plants they can also convert uridine to cytidine (U-to-C). For the most part, RNA editing occurs in gene coding sequences to restore codons to their evolutionary conserved state, but some RNA editing sites are more consequential than others. There are many cases where an RNA editing event is necessary to create a start codon, or to remove a premature stop codon. These types of editing events are abundant in the organelle transcripts of ferns and appear to be more conserved than other editing events. It is conceivable that in some plant species, RNA editing is emerging as a 'gate' for protein translation – a transcript must be fully processed before translation can initiate. This kind of 'switch' mechanism would be ideal for building molecular logic gates. I will present our research into the mechanisms and evolution of RNA editing in plant organelle transcriptomes, and how we have taken inspiration from nature to engineer and optimise synthetic RNA editing proteins for targeted base editing in chloroplasts and mitochondria.

Biography

Dr Farley Kwok van der Giezen is a Research Associate at the ARC CoE in Plants for Space. Farley completed his PhD at the University of Western Australia, and studies the evolution, mechanisms and engineering of RNA editing proteins in plant chloroplasts and mitochondria. He is currently serving as the chair of the Perth Protein Group, and co-chair of the WA RNA Salon.

Dr Bryn Funnekotter
Kings Park Science, Department of Biodiversity, Conservation and Attractions, WA

Frozen for the Future: The Science Behind Kings Park's Cryopreservation Biobank



Abstract
The Kings Park Science Conservation Biotechnology program supports the conservation of threatened Western Australian plant species through the integration of plant tissue culture and long-term cryopreservation. The program currently maintains over 50 plant species in tissue culture, including 36 species listed as threatened. Long-term preservation is achieved through cryopreservation, where tissue-cultured plant material, seeds, pollen, and other propagules are stored in liquid nitrogen at –196°C. To date, 44 species have been successfully cryopreserved, providing a secure conservation resource that can be recovered and propagated when required. Current research focuses on developing species-specific cryopreservation protocols while investigating the physiological responses of plants to cryogenic storage and recovery processes. This presentation will cover some of the findings seen with thermal analysis to understand ice formation and effectiveness of the cryoprotective agents used, lipidomics studies to assess membrane compositions and membrane leakage, changes to the redox environment and the effect cryopreservation has on mitochondrial function.

Biography
Dr Bryn Funnekotter is a Research Scientist at Kings Park Science, which is a part of the Department of Biodiversity, Conservation and Attractions in Perth, Western Australia. Bryn's research focus is to enhance and solidify the use of cryopreservation and plant tissue culture to conserve Australia's unique plant species. He has a keen interest in studying the fundamental science underpinning conservation biotechnologies. By pinpointing and understanding the limitations of current methodologies, more efficient, wider-reaching protocols will enhance our ability to conserve our threatened species.

Plenary Presentation
Theatre Auaditorium
CHAIR: Sue Fletcher

Professor Mirko Uljarević

The Kids Research Institute Australia

Advancing Understanding of Neurodevelopmental and Neuropsychiatric Conditions through Novel Assessments

Abstract
This talk will provide an overview of the major challenges facing research and clinical practice involving children and adolescents with neurodevelopmental conditions, including the limitations of current categorical and dimensional frameworks and assessments. This will be followed by an overview of the latest advances in the measurement development space, illustrating how new assessments hold the potential to transform how we understand, conceptualise, research, and support children, and a discussion of the planned future work in this space.

Biography
Professor Uljarević is a Professor at CliniKids and The Kids Research Institute. Prior to the current role, he was an Associate Professor of psychiatry and behavioral sciences at Stanford University and Director of the Stanford Program for Psychometrics and Measurement-Based Care. His research takes a life-span perspective and is driven by the urgent need to improve outcomes for people with autism and other neuropsychiatric (NPD) and neurodevelopmental (NDD) conditions. His primary research interest has focused on combining cutting-edge psychometric procedures and a big data approach to better understand the structure of clinical phenotypes across autism and other NPD and NDD, and on using this knowledge to improve existing and develop new clinical assessments that are more effective for characterizing clinical and cognitive features, tracking the natural and support-related change and for use in genetic and neurobiological studies. His current work is supported by the Future Health Research and Innovation (FHRI) Fund Distinguished Fellowship and focuses on the development of the new generation of assessments to facilitate screening, characterization, and intervention matching across clinical and research contexts.

Poster Locations

STUDENT POSTERS	POSTER SESSION	LOCATION
Health Sciences	A	Lower Colonnades
Immunology	B	
Gene Therapy	C	
Frontiers in Genetics	D	
Biochemistry and Molecular Biology	E	Foyer Wall 1
~Omics	F	Foyer Wall 2
Cell and Developmental Biology	G	Foyer Centre
Microbiology	H	Upper Colonnades
Biodiversity	I	
Professional Scientists: Biological Sciences	J	Seminar Room 3
Professional Scientists: Biomedical Sciences	K	
Plant Science; Pathology & Crop Protection	L	
Plant Science - Food Science and Applied Biology	M	

Poster Presentations-Authors

NAME		POSTER#
Adams	E	**A6**
Adhikari	B	**M1**
Ahmed	Z	**M5**
Alam	MJ	**H7**
Ale	S	**I3**
Alkresheh	R	**G10**
Aquino	G	**G5**
Ayres	C	**B2**
Bacal	C	**K1**
Bartholomeusz	Z	**H12**
Bhalla	N	**A9**
Bharwad	H	**B5**
Brown	I	**F2**
Brown	R	**K4**
Brundrett	M	**J13**
Busby	K	**B7**
Byrne	J	**G8**
Chambers	W	**B10**
Cheasley	D	**K3**
Chia	C	**G3**
Chua	V	**K6**
Churchill	E	**A7**
Cusens	T	**H10**
Dao	B	**F1**
de Jong	I	**E5**
Dow	A	**G1**
Drysdale	S	**F5**
Dyton	A	**G2**
Emery	C	**A4**
Fahriani	M	**I4**
Farhan	W	**B9**
Fisher	B	**A2**
Form	A	**H1**
Form	A	**H2**
Gacer	J	**D3**
Galopin	R	**D6**
Gifford	C	**L3**
Gomes	A	**A1**
Harding	A	**E1**
Hegde	KR	**M2**
Herbst	C	**L1**
Hii	H	**G7**
Hu	S	**G11**
Ingram	H	**C1**
Javed	MA	**I5**
Jelaca	C	**B3**
Joy	T	**A11**
Kar	S	**H9**
Knowling	E	**E2**
Kurejsepi	G	**E4**
Kuznetsova	I	**J7**
Lacquio	M	**F3**
Lai	A	**D1**
Leith	F	**C2**
Lenzo	L	**L6**
Li	Z	**E6**
Lim	A	**B1**
Lim	C	**H4**
Link	S	**D7**
Mahmud	S	**J12**
March	M	**D2**
Marshall	A	**J3**
Marshall	L	**J8**
McNess	R	**M3**
Misiun	P	**L8**
Mohd Saad	NS	**J11**
Mukherjee	S	**J2**
Nakazwe	Z	**H13**
Negahdar	NZI	**B8**
Ninan	J	**B6**
Peh	C	**B4**
Petrucci	M	**D5**
Pullakhandam	A	**J4**
Qian	P	**D8**
Qin	B	**I1**
Rapanaro	G	**G6**
Rashid	S	**F4**
Rudler	D	**J6**
Ryan	E	**E3**
Sahchithananthan	S	**I2**
Saleem	A	**L10**
Sbrana	L	**H6**
Schreurs	J	**C4**
Seeam	T	**G12**
Sexauer	D	**A5**
Shirokikh	N	**J10**
Singh	D	**L4**
Smith	T	**C3**
Sonam	U	**L9**
Sturrock	R	**A10**
Sudeep	SM	**F6**
Szabó	D	**H5**
Templeman	A	**H3**
Tenaglia	V	**H11**
Trotter	J	**A8**
Tse	SW	**J1**
van der Heyde	M	**J9**
Vaz Pereira	M	**L7**
Verdonk	C	**J5**
Wallis	M	**G9**
Webster	D	**G4**
Wilson	C	**A3**
Witham	C	**K2**
Worth	L	**D4**
Xu	Y	**M4**
Xue	S	**K5**
Yan	Z	**B11**
Yee	N	**H8**
Zhang	C	**L2**
Zhu	J	**L5**

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Combined Biological Sciences Meeting 2026

	Time	Session	Speaker	Title				
	8-8:40	Early Career Researcher Development Session (Seminar Room 4)	Dr. Vivian Chua Dr. Sherif Boulos Dr. Amanda Cleaver A/Prof Bill Bateman					
	8-8:30	**Registration and Breakfast (sponsored by Illumina)**						
	08:45	Welcome		Opening address				
	09:00	Plenary (Theatre Auditorium) Chair: Dr Farley Kwok van der Giezen	Prof. Jacqueline Batley	Using pan genomics to identify disease resistance genes in Brassica species				
New Investigator Symposia	09:40	New Investigator Session Biomedical Sciences (Theatre Auditorium) Chair: Associate Professor Briardo Llorente	Amelia Dyton Christopher Witham Linda Wijaya Mariana L Orozco Morales	Targeting NAD+ metabolism in Down Syndrome-associated Acute Lymphoblastic Leukaemia How We Fight Cancer Using Ubiquitin Unravelling RASopathies Using iPSC-Derived Neural Disease Modelling Targeting PPARg To Enhance Tumour Immunogenicity And Immunotherapy				
		New Investigator Session Plants and Genetics (Seminar Room 4) Chair: Professor Lee Hickey	Daniel Edge-Garza Kristina Gagalova Ulduz Vafadarshamasbi Zeeshan Ahmed	Marker-Assisted Seedling Selection for Improved Apple Breeding Efficiency in Australia Beyond RNAseq: Integrating Transcriptomics, Proteomics, and QTL Mapping to Reveal Driving Infection Mechanisms in Patastagonospora nodorum - Wheat pathosystem Toward robust chromosome-scale genome assembly with iterative Hi-C scaffolding and validation Stable Inheritance and Quantitative Phenotyping of Variegation in Anigozanthos flavidus 'Stripie': A Model for Investigating Photosynthetic Compensation Mechanisms				
		New Investigator Session Microbiology and Genomics (Seminar Room 1) Chair: Professor Ryan Lister	Dilruka Kasturiarachchi Marhami Fahriani Roland Politan Xavier Barton	DBHS Proteins as a Model for Studying Biomolecular Condensate Assembly and Maturation Beyond Enterococcus faecalis and Enterococcus faecium: Genomic insights into other enterococci causing bacteraemia Engineering phosphate-controlled PHB production in Vibrio natriegens from renewable feedstocks Do Ticks Care About the Environment?				
	10:40	**Morning Tea (sponsored by ECU Centre for Precision Health)**						
	11:00	(Upper and Lower Colonnade)	**Poster Session**					
	11:30	Plenary (Theatre Auditorium) Chair: Professor Mike Bunce	Dame Juliet Gerrard	Tales of the unexpected: a science career across universities, business, and government with some lessons learned along the way.				
	12:10	**Lunch (sponsored by Curtin MRI)**						
	01:00	(Upper and Lower Colonnade)	**Poster Session**					
Specialist Symposia	01:30	Breakout sesion	**Microbiology**	**Cell and Developmental Biology**	**Frontiers in Genetics**	**Biodiversity**	**Plant Science**	
		Organiser	Theatre Auditorium	Seminar Room 4	Seminar Room 2	Seminar Room 1	Formal Dining Room	
		Chair	Dr. Lucy Furfaro Dr. Chris Mullaly	Dr. Liisa Hirvonen Dr. David Martino	Muhammad Ahmed Dr. Richard Allcock	Prof. Liz Watkin	Prof. Jacqueline Batley	
		Keynote Speaker	Dr. Christine Carson	Dr. Ben Dwyer	A/Prof Briardo Llorente	A/Prof Stephanie Godfrey	Prof. Lee Hickey	
	02:00	Student Speakers	Dávid Szabó Princy Shoby Vanessa Tenaglia Tim Cusens	Beatriz Da Silva Campos Dane Webster Jacob Byrne Keea Inder-Smith	Bac Dao Callum Flanagan Joey Lye Saskia Saville	Cameron Dodd Heather Russell Pia Dethlefsen Shengyao Lin	Yutathkarn Coles Yubei (Clementine) Wu Leon Lenzo Naomi Gray	
	03:00	**Afternoon Tea (sponsored by Department of Energy and Economic Diversification)**						
	03:30	Invited Speakers	Dr. Rutendo Mapengo Dr. Chris Mullaly	Dr. Ben Padman Dr. Jasreen Kular	Dr. Jessica Kretzmann Prof. Ryan Lister	Dr. Alexandre Siqueira A/Prof Holly Kirk	Dr. Farley Kwok van der Giezen Dr. Bryn Funnekotter	
	04:30	Plenary (Theatre Auditorium) Chair: Professor Sue Fletcher		Advancing Understanding of Neurodevelopmental and Neuropsychiatric Conditions through Novel Assessments				
	05:10	Scientific Awards		Scientific award ceremony and closing address				
	5:30 - 7:30	**Sundowner (sponsored by Murdoch University)**						