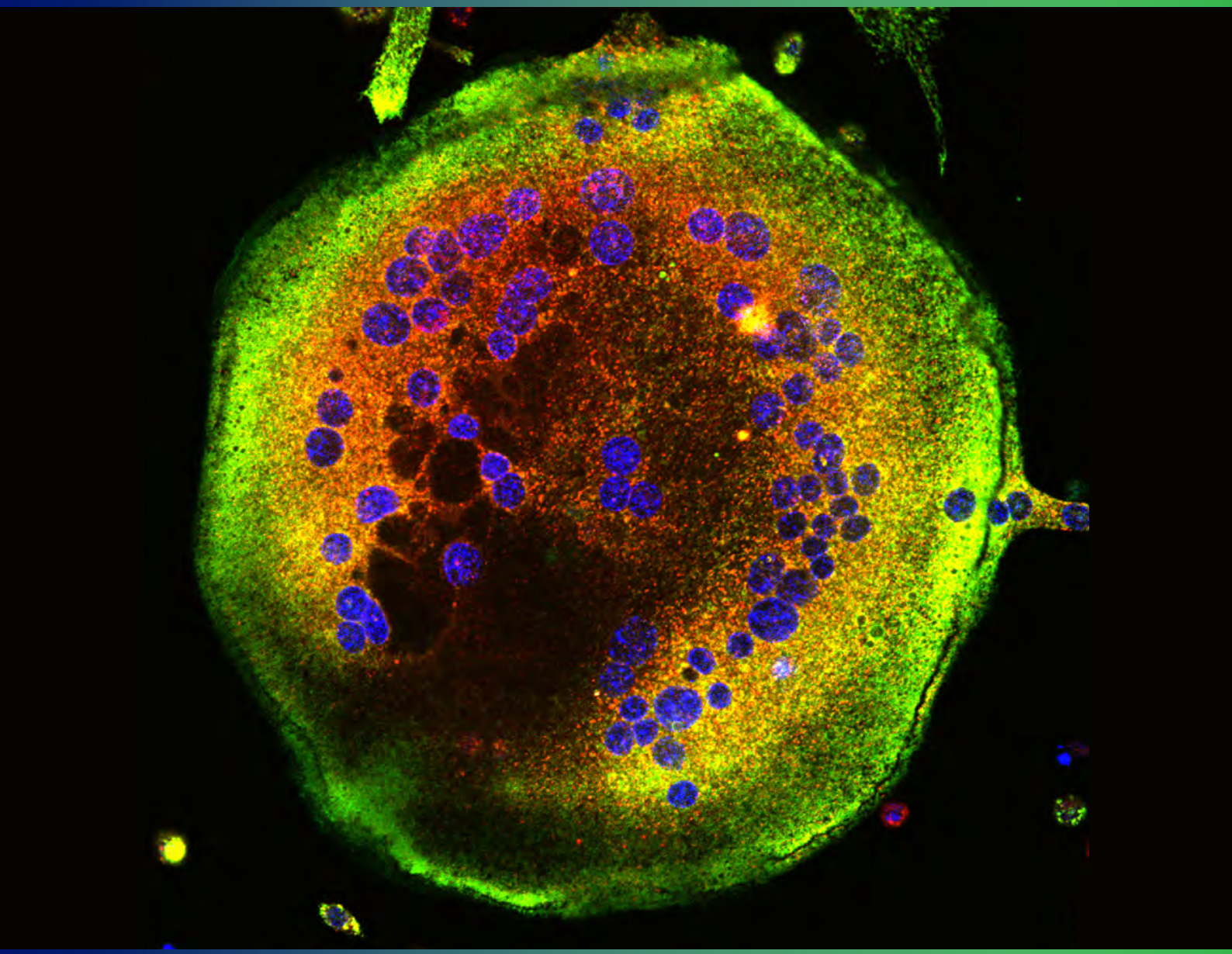
CBSM

2019 Combined Biological Sciences Meeting



The University Club
30th August 2019

Promoting biological science in Western Australia

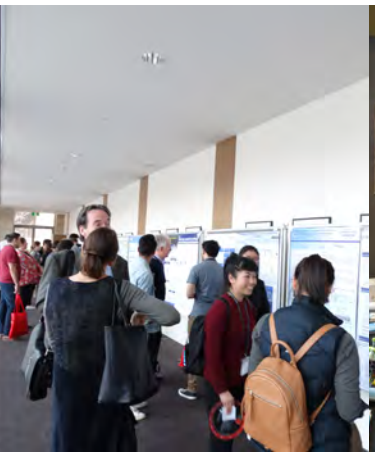
Program & Abstracts

Editors: Sue Fletcher, Jessica Cale & Clayton Fragall

29th Annual Combined Biological Sciences Meeting

The University Club

30th August 2019





Cover Image

Osteoclast differentiated from Raw cells transfected with GFP-slc23a2

L.M.G Scientific Services Award

Amy Ribet
School of Biomedical Sciences, Faculty of Health and Medical Sciences
The University of Western Australia

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Royal Society of Western Australia BIODIVERSITY	Student Oral Presentation

Poster Presentation Awards
Australian and New Zealand Society for Cell and Developmental Biology (ANZSCDB)
Australian Society for Biochemistry and Molecular Biology (ASBMB)
Australasian Society for Immunology (ASI) WA Branch
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The ASI WA organizing committee warmly invites you to participate in the

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21st-22nd October 2019
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This year's themes include:

Early life immunity
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Dr. Kylie Quinn
Vice-Chancellor's Research Fellow, RMIT

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Registration deadline 20th September. All inclusive 2-day costs:-

$45 for student ASI members
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Registration and abstract submission is via
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Any questions contact: Bree.Foley@telethonkids.org.au

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CBSM 2018 Award Recipients

CBSM 2018 New Investigator Awards

Department of Health Western Australia Award	Ms Kelly Martinovich	Telethon Kids Institute
Edith Cowan University Award	Ms Nicole Bzdyl	Marshall Centre for Infectious Diseases, The University of Western Australia

CBSM 2018 Student Oral Awards

Curtin Health Innovation Research Institute Award	Ms Lelinh Duong	School of Pharmacy and Biomedical Sciences, Curtin University
Edith Cowan University Award	Ms Alice Michie	School of Biomedical Sciences, The University of Western Australia
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Centre for Comparative Genomics, Murdoch University Award	Ms Janya Grainok	Centre for Comparative Genomics, Murdoch University

Life Technologies 2018 Manuscript Award

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L.M.G. Scientific Services 2018 Image Award

Amy Ribet
'Osteoclast differentiated from Raw cells transfected with GFP-slc23a2'
School of Biomedical Sciences, Faculty of Health and Medical Sciences
The University of Western Australia
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CBSM 2018 Award Recipients

CBSM 2018 Student Poster Awards		
Australian and New Zealand Society for Cell and Developmental Biology (ANZSCDB)	Tenielle George	Curtin Health Innovation Research Institute, Curtin University
Australian Society for Biochemistry and Molecular Biology (ASBMB)	Conrad Hogg	Laboratory of Cancer Medicine, Harry Perkins Institute of Medical Research
Australian Society for Immunology (ASI) WA branch	Jennifer Currenti	School of Human Sciences, The University of Western Australia
Australian Society for Microbiology (ASM) WA branch	Melissa Koh	Marshall Centre for Infectious Diseases, The University of Western Australia
Centre for Crops & Disease Management (CCDM)	Emma Bonello	Centre for Rhizobium Studies, Murdoch University
Human Genetics Society of Australasia	Isabel Hemming	Brain Growth and Disease Laboratory, Harry Perkins Institute of Medical Research
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Qiagen	9
Rowe Scientific	22
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Thermo Fisher	12, 13

Dr Joshua Lewis

Edith Cowan University

Writing a successful fellowship application, lessons learned the hard way!

Biography

Dr Joshua Lewis heads the disorders of mineralisation research group with a focus on the shared epidemiology of osteoporosis and cardiovascular disease, including biomarkers, nutrition and lifestyle factors and genetic factors. His research aims to develop better ways to identify and prevent disease before the onset of clinical symptoms. He holds a highly competitive National Health and Medical Research Council Career Development Fellow at the School of Medical and Health Sciences, Edith Cowan University and is an author on >120 publications in leading journals such as Nature, Nature Genetics, Journal of Bone and Mineral Research, Archives of Internal Medicine and the American Journal of Clinical Nutrition. He has been awarded prestigious national and international awards and honours for his research that regularly receives media attention and has attracted almost 2 million dollars in funding from 21 local and national grants and scholarships. He is regularly on grant review panels, including the NHMRC project and investigator grant review panels. He will discuss his experiences in writing fellowship applications and some of the lessons he has learned along the way.

Notes

Professor Sarah Bekessy

RMIT University

The critical role of 'everyday nature' for the future of cities

Abstract
A perfect storm of ideas is generating unprecedented enthusiasm for embracing nature in cities. Re-enchanting urban residents with nature can deliver a remarkable range of health benefits, while creating cities that are more resilient to climate change. Creating 'every day nature' in cities presents opportunities to reverse the fate of many threatened species and connect people with Indigenous history and culture. But it's more than just urban greening; it's generating daily doses of biodiversity. The future of livable cities may well depend on this new conceptualization, but a major shift in the way nature is conceived of and planned for is required.

Biography
Professor Sarah Bekessy leads the Interdisciplinary Conservation Science research group at RMIT University. She is interested in the intersection between science and policy in environmental management and is currently involved in an interdisciplinary range of research projects, including an ARC Future Fellowship titled 'Socio-ecological models for environmental decision making' and an ARC linkage project titled 'Designing green spaces for biodiversity and human well-being'. She leads projects in two National Environment Science Program Hubs (Threatened Species Hub and Clean Air and Urban Landscapes Hub) and is a Chief Investigator in the European Commission funded project Urban Greenup, which seeks to evaluate nature-based solutions for cities. She co-developed the Biodiversity Sensitive Urban Design protocol that has now been used by numerous developers, governments and non-government organisations to design innovative urban biodiversity strategies.

Plenary Presentation

CHAIR: Associate Prof Charlene Kahler

Professor Mark Nicol

The University of Western Australia

Early life microbial exposures and health outcomes in young African children

Abstract

Our exposure to microbes in early life shapes our immune system, impacts on growth and neurodevelopment and affects our risk of infection. Mark will draw from his experiences studying children participating in a birth cohort in a low-income South African community to explore how detailed study of these microbial exposures can help us better understand the role of the microbiome in early child health. In particular, he will describe recent work exploring the relationship between the nutritional content of breast milk, the breast milk microbiome and lactational outcomes, and discuss how the microbiome of the upper airways may modulate the risk of pneumonia in children.

Biography

Mark Nicol is Professor of Microbiology in the Division of Infection and Immunity within the School of Biomedical Sciences at UWA. He also holds an honorary appointment at the University of Cape Town. He studied medicine and medical microbiology at the University of the Witwatersrand and completed his PhD in childhood tuberculosis in Cape Town. His group uses modern molecular tools to study the complex microbial communities in the human body, investigating how imbalances in these communities cause illness. He also has an interest in developing and evaluating better diagnostic tests for infections, particular for diseases of poverty. Much of his work focuses on respiratory infections in children, such as pneumonia and tuberculosis.

Professor Alpha Yap

The University of Queensland

Tissue forces and epithelial homeostasis

Abstract
Epithelia constitute many of the principal barriers in metazoan bodies. They demarcate tissue compartments and protect the internal milieu of the body from the external environment. Epithelia are also common sites for disease, notably cancer and inflammation. Yet, the incidence of epithelial disease is remarkably low, given their constant exposure to injurious agents. This implies that mechanisms must exist for epithelia to detect potential homeostatic disturbances and deal with them. Recently, it has become increasingly apparent that epithelia use mechanosensing as a mechanism to support homeostasis. Cells constantly exert contractile forces on their neighbours through their cell-cell junctions, and possess mechanotransduction pathways at those junctions that detect changes in force. Importantly, altered contractility is a hallmark of many forms of cellular disturbance from apoptosis to transformation. Mechanosensing may then be an early-warning system that allows epithelia to detect, and respond to, homeostatic challenges. Conversely, defects in mechanotransduction may predispose epithelia to disease.

Biography
Alpha Yap is a Professor and Group Leader at the Institute for Molecular Bioscience, The University of Queensland. After training in Internal Medicine, Endocrinology and Cell Physiology, he undertook post-doctoral research with Barry Gumbiner at Memorial Sloan-Kettering Cancer Center before returning to Australia to establish his independent research group. His laboratory studies how tissue mechanics and mechanotransduction participate in epithelial morphogenesis and homeostasis, work that has led them into the rapidly-developing field of mechanobiology. Collaborating across disciplines with colleagues from physics, developmental biology and mathematics, they have been instrumental in discovering how mechanical forces are generated, and sensed, to coordinate cell behaviour in tissues. Alpha Yap was Chair of the 2011 Gordon Research Conference on Cell Contact & Adhesion and of the 2016 GRC on Signaling by Adhesion Receptors. He currently serves on the editorial boards of several journals, amongst them Molecular Biology of the Cell, Developmental Cell and Current Biology. He was the recipient of the 2013 President's Medal of the Australia and New Zealand Society for Cell and Developmental Biology and is a Senior Principal Research Fellow of the National Health and Medical Research Council of Australia.

Notes

GOVERNMENT OF WESTERN AUSTRALIA

Department of Health

Department of Health WA

New Investigator session

Theatre Auditorium

CHAIR: Dr May Aung-Htut
Dr Jessica Buck

Speaker	Title	Time
Ms Shelley Waters	*Digging deep to reveal novel CMV variants and associations with patient outcome*	9:40
Ms Jennifer Currenti	*Deep sequence analysis of HIV adaptation following vertical transmission reveals the impact of immune pressure on the evolution of HIV*	9:55
Dr Lucy Furfaro	*Group B Streptococcus colonisation of Western Australian pregnant women*	10:10
Dr Rachael Zemek	*Sensitisation to immune checkpoint blockade through activation of a STAT1/NK axis in the tumour microenvironment*	10:25

Digging Deep to Reveal Novel CMV Variants and Associations with Patient Outcome

Shelley Waters[1], Silvia Lee[1,2], Kylie Munyard[1], Ibnu Ariyanto[3], Nina Kresoje[4,5], Silvana Gaudieri[6],, Shay Leary[6], Ashley Irish[7] Patricia Price[1], Richard Allcock[4,5]

[1]School of Pharmacology & Biomedical Science, Curtin Health Innovation Research Institute, [2]Department of Microbiology, Pathwest Laboratory Medicine, [3]Virology & Cancer Pathobiology Research Center, Universitas Indonesia, Jakarta, Indonesia, [4]LotteryWest State Biomedical Facility – Genomics, [5]School of Human Sciences, University of Western Australia, [6]Institute for Immunology & Infectious Diseases, Murdoch University, [7]Renal Unit, Fiona Stanley Hospital

Human cytomegalovirus (CMV) is a beta-herpesvirus carried by ~83% of the world's population. Acute CMV infections are asymptomatic in healthy individuals, but can be severe in newborns, transplant recipients and HIV patients. CMV is most commonly genotyped based on the UL55 gene which encodes glycoprotein B (gB) required for viral entry into host cells. gB genotypes define four sub-types but these do not associate with severity of infection or patient outcome, and show no geographical patterns. Through evolution, CMV has acquired homologs of host genes that may help the virus escape immune surveillance and/or explain why extremely small amounts of virus can trigger dramatic clinical and immunological sequelae. For example, the US28 gene encodes a chemokine receptor similar to CX3CR1 and can alter cell to cell spread or act as a co-receptor for HIV. We address the possibility that US28 genotypes may affect pathogenicity. We developed a NGS protocol to sequence very small amounts of CMV DNA in the presence of large quantities of human genomic DNA, using Ampliseq technologies on an Ion Proton platform. We sequenced 60 samples (saliva, blood and urine) from Australian transplant recipients, neonates and healthy adults, and Indonesian HIV patients. The sequencing captured 5 CMV genes with a mean coverage depth of 11,734. Preliminary analysis of US28 using proprietary software (VGAS) has revealed novel variants in immunocompromised hosts when compared to healthy adults. We identified 62 variants, 19 of which were non-synonymous. Five of these were predicted to be "possibly" or "probably" damaging by PolyPhen-2. They can now be modelled against the structure of CX3CR1 to further understand their possible phenotypic effects. We are currently assessing genotype associations with disease, immunological measurements and patient outcome. We are also analysing the remaining 4 genes, UL18, UL40 (NK receptor homologues), UL111a (Il-10 homologue) and US2 (degrades MHC-I).

Deep Sequence Analysis of HIV Adaptation Following Vertical Transmission Reveals the Impact of Immune Pressure on the Evolution of HIV

Currenti J[1], Chopra A[2,3¶], John M[2,4¶], Leary S[2¶], McKinnon E[2], Eric Alves[1], Pilkinton M[3], Smith R3, Barnett L[3], McDonnell WJ[3], Lucas M[5], Noel F[6], Mallal S[2,3], Conrad JA[7], Kalams S[3&], and Gaudieri S[1,2,3&]

[1]Human Sciences, UWA, Australia, [2]IIID, Murdoch University, Australia, [3]Infectious Diseases, Vanderbilt University, USA, [4]Clinical Immunology, RPH, Australia, [5]Medicine, UWA, Australia, [6]GHESKIO Centre, Haiti, [7]Chemistry, Vanderbilt University, USA, [¶&]Equal contribution

Human immunodeficiency virus (HIV) is responsible for more deaths than any other infectious disease worldwide, with the only treatment option being lifelong antiretroviral therapy. HIV changes as a result of an individual's immune response via modifications (adaptations) that affect its recognition by the immune response and impact an individual's disease outcome. These adaptations are specific to an individual's immune response and can be transmitted. To design an effective vaccine, it is vital to understand the complex interactions between the immune response and HIV (host-viral interactions). We sequenced the HIV strains present in 26 mother-to-child transmission pairs. The resultant sequences and previously determined adaptations were used to determine what/if adaptations were present, and create a score depicting the level of adaptation in subjects' HIV strains. We showed that the dynamics of HIV adaptations following mother-to-child transmission provides insight into host-viral interactions. Transmitted viruses were commonly already adapted to the child's immune response, however we found evidence of new adaptation in the child. These specific immune responses in the child suggest that the immune response to HIV may be different in children than in adults. Analysis of HIV sequences in mother/child pairs revealed HIV transmission dynamics, identifying potential benefits of specific adaptations for the virus. It is hoped a better understanding of these dynamics can be used to inform vaccine design.

Group B Streptococcus Colonisation of Western Australian Pregnant Women

Lucy Furfaro[1], Elizabeth Nathan[1, 2], Barbara Chang[3] and Matthew Payne[1]

[1]Division of Obstetrics and Gynaecology, University of Western Australia. [2]Women and Infants Research Foundation, King Edward Memorial Hospital. [3]Marshall Centre for Infectious Diseases Research and Training, University of Western Australia.

Streptococcus agalactiae, or Group B Streptococcus (GBS), is a leading neonatal pathogen that causes sepsis, meningitis and pneumonia. Globally, strategies have been implemented to address vertical transmission, and in Western Australia (WA) culture-based screening at 35-37 weeks gestation is part of routine care to guide antibiotic administration. In this study we aimed to describe antenatal GBS colonisation within a large population in WA; this is a population that has received little attention in previous research. A cohort of 814 pregnant women attending antenatal clinics (2015-2017) provided self-collected vaginal and rectal swabs at ≤22 weeks (n = 814) and ≥33 weeks (n = 567) gestation. These were assessed for GBS presence using culture and PCR and serotyping was conducted using molecular methods. Lifestyle questionnaires and medical data were collected. We observed an overall GBS colonisation rate of 24%, with 10.6% of positive participants transiently colonised. Ethnicity (Aboriginal, Torres Strait Islander and African), maternal age ≥25 years, vitamin use, frequent sexual intercourse (≥5 times/week) and use of sex toys were associated with GBS colonisation following logistic regression analysis. The dominant serotypes identified were Ia (27.9%), III (20.9%), II (16.3%), V (15.8%), Ib (8.4%), VI (5.1%), IV (2.8%), NT (1.9), VIII (0.5%) and IX (0.5%) at visit one, with V (18.9%) preceding serotype II (18.2%) at visit two. Serotype VII was not detected. This is the first cohort study to assess GBS colonisation in WA pregnant women and will be highly beneficial for guiding future clinical practice and therapeutic options, in particular, selection of suitable vaccine candidates.

Sensitisation to Immune Checkpoint Blockade through Activation of a STAT1/NK Axis in the Tumour Microenvironment

Rachael M. Zemek[1,2,3], Emma De Jong[3], Wee Loong Chin[1,2,4], Iona S. Schuster[1,5,6], Vanessa S. Fear[1,2], Thomas H. Casey[1,2], Cath Forbes[1,2], Sarah J. Dart[1,2], Connull Leslie[7], Ayham Zaitouny[1,8], Michael Small[1,8], Louis Boon[9], Alistair R.R. Forrest[10], Daithi O Muiri[10], Mariapia A. Degli-Esposti[1,5,6], Michael J. Millward[1,4], Anna K. Nowak[1,2,4], Timo Lassmann[3], Anthony Bosco[3], Richard A. Lake[1,2], W. Joost Lesterhuis[1,2,3]

[1]UWA, [2]National Centre for Asbestos Related Diseases, [3]Telethon Kids Institute, [4]Sir Charles Gairdner Hospital, [5]Lions Eye Institute, [6]Monash University, [7]PathWest Laboratory Medicine, [8]CSIRO, [9]Bioceros, [13]Harry Perkins Institute of Medical Research

Introduction: Treatment with antibodies that block immune checkpoints have shown efficacy in some cancers, resulting in complete regression of tumours in some patients, but no response in others. It is not well understood what biological processes contribute to an effective response to immune checkpoint blockade (ICB). This lack of understanding hinders the development of rational combination treatments to improve responses to ICB. **Problem Statement:** We identified what determines the response to ICB and a way to induce it, improving the response to ICB. **Procedure:** We set out to define the pre-treatment microenvironment associated with an effective outcome by using the fact that genetically identical mice bearing the same monoclonal cancer cell line-derived tumours have a clear divergence in response, similar to that seen in patients. We compared the cellular composition and gene expression profiles of responsive and non-responsive tumours from mice before ICB, using RNA sequencing, flow cytometry and immunohistochemistry We validated the targets *in vivo* and in publicly available RNA sequencing datasets from patients treated with ICB. **Results:** We found that responsive tumours were characterised by an inflammatory gene expression signature consistent with up-regulation of signal transducer and activator of transcription 1 (STAT1) and Toll-like receptor 3 (TLR3) signalling and down-regulation of interleukin-10 (IL-10) signalling. In addition, responsive tumours had more natural killer (NK) cells, which were necessary for response. We therefore rationalised a pre-treatment combination using the STAT1-activating cytokine interferon-γ, the TLR3 ligand poly(I:C), and an anti–IL-10 antibody. This combination sensitised tumours to ICB by attracting NK cells into the tumour, resulting in increased cure rates. **Conclusion:** Our results identify a pre-treatment tumour microenvironment that predicts response to ICB, which can be therapeutically attained. These data suggest a biomarker-driven approach to patient management to establish whether a patient would benefit from treatment with sensitising therapeutics before ICB.

Notes

Edith Cowan University

New Investigator session

Case Study Room

CHAIR: Dr Megan Lloyd

Speaker	Title	Time
Mrs Julie Sartori	*The Placenta Project: Characteristics in Assisted & Non-Assisted Pregnancy for a Western Australian Cohort*	9:40
Dr Kritu Panta	*Dengue antibody and Zika*	9:55
Dr Kai Chen	*Pseurotin A Prevents Estrogen Deficiency-induced Bone Loss by Inhibiting Osteoclastogenesis via Suppressing Reactive Oxygen Species Level*	10:10
Dr Pauline Zaenker	*Identification and Validation of a Diagnostic Autoantibody Signature for Primary Cutaneous Melanoma*	10:25

The Placenta Project: Characteristics in Assisted and Non – Assisted Pregnancy for a Western Australian Cohort

Julie Sartori[1], Anna, C Callan[1], Peter Roberts[1], Michelle Tickner[1], Michelle Cannon[1], Julie Quinlivan[2], David Coall[1].

[1]Edith Cowan University, [2]Professional Service Review, Australia

Over 350 000 babies are born in Australia each year, a majority of which are conceived by natural conception. However, an increasing infertility rate (currently above 20%) has seen a rise in the number of Australian couples conceiving via Assisted Reproductive Technologies [ART]. Previous studies have revealed that routine ART procedures such as in-vitro fertilisation (IVF) and intracytoplasmic sperm injection (ICSI) can be associated with poorer pregnancy outcomes such as preterm birth and low birth weight. To investigate this further, we examined maternal, fetal and placental characteristics from assisted and non-assisted pregnancies. As part of the Placenta Project, 630 pregnant women were recruited from private and public hospitals in Perth, Western Australia. Of these, 538 women conceived via natural conception and 92 conceived using ART. Data collected from participants included a survey questionnaire (Maternal Health Questionnaire) which included paternal demographic data, Gross Placental Examination, Digital Placental Imaging, Maternal and Natal Health Records. Statistical analyses were conducted using SPSS. When age matching assisted and non-assisted pregnancies there were few differences between these subsets. When comparison across the entire non-assisted cohort, infant birth weight, placental shape, and umbilical cord insertion remained similar. However, the parents of pregnancies resulting from ART were older (maternal mean 34.3 years and paternal mean 36.2 years) which is consistent with national data. Further to this, there were key aspects (placental morphology, maternal mental health and morning sickness) that showed clinically significant differences that would benefit from further investigation. These findings highlight the importance of research to promote a greater understanding of both natural and assisted pregnancies.

Dengue Antibody and Zika

Kritu Panta[1], David Smith[1 2], Allison Imrie[1 2]

[1]School of Biomedical Sciences, The University of Western Australia, [2]Pathwest Laboratory Medicine

Background: Dengue virus (DENV) and Zika Virus (ZIKV) are closely related Flaviviruses with more than 50% homology in amino acid polyprotein sequence. Structural similarity between DENV and ZIKV results in high immunological cross-reactivity. Studies using monoclonal antibodies in mice models have shown that pre-existing DENV antibodies may either cross-protect, or enhance, ZIKV infection. We studied the effects of human DENV antisera on in-vitro ZIKV replication. Previous studies have investigated Zika infection in dengue endemic countries with high seroprevalence and where the sequence of DENV infections is not known; our study population of returned WA travelers with well characterized monotypic DENV infection allows the influence of pre-existing DENV antibodies, induced by of each of the four serotypes, to be assessed. Associations between pre-existing DENV humoral immunity and ZIKV replication kinetics may be therefore be defined. **Method:** Serum samples were collected longitudinally from travelers with well-defined DENV infection from 1 week to 5 years after diagnosed acute infection. The capacity of anti-DENV antisera (defined by haemagglutination inhibition and focus reduction neutralization test) to enhance or neutralize ZIKV replication was assessed. **Result:** Monotypic, cross-reactive anti-DENV antisera collected less than 2 months post onset of acute DENV infection may enhance ZIKV replication; antisera collected more than 2 years after infection do not enhance ZIKV replication. Monotypic DENV antisera cross-recognized ZIKV at high magnitude, however ZIKV neutralization was strain-specific. **Conclusion:** ZIKV replication is neutralized in a strain-specific manner by anti-DENV antisera with differential cross-recognition; specificity and magnitude varied across DENV-infected individuals. Time post-DENV infection influenced ZIKV enhancement. These findings have direct implications for DENV vaccine design, especially for populations where DENV and ZIKV circulate.

Pseurotin A Prevents Estrogen Deficiency-induced Bone Loss by Inhibiting Osteoclastogenesis via Suppressing Reactive Oxygen Species Level.

Kai Chen[1], Pengcheng Qiu[2], Jennifer Tickner[1], Shunwu Fan[2], Xianfeng Lin[2], and Jiake Xu[1]

[1]School of Biomedical Sciences, The University of Western Australia, [2]Department of Orthopaedic, Sir Run Run Shaw Hospital, Medical College of Zhejiang University, Hangzhou, Zhejiang, China

Introduction. Excessive osteoclast activity plays an essential role in postmenopausal osteoporosis. Growing evidence shows that intracellular reactive oxygen species (ROS) accumulation is a critical factor in the development of osteoporosis by triggering osteoclast formation and function. Pseurotin A (PA) is a bioactive secondary metabolite originally isolated from *Aspergillus fumigatus* that has been shown to have antioxidant activity. However, its effects on osteoclasts remain unknown. **Problem Statement.** We hypothesized that PA may have therapeutic effects on bone loss in ovariectomized (OVX) mice by inhibiting hyperactive osteoclasts via targeting ROS. **Procedures.** PA was added to osteoclast culture medium *in vitro* to identify whether PA can inhibit osteoclast formation. Western Blot (WB) assay and qPCR were used to investigate the cell signalling pathways. Furthermore, OVX osteoporosis mice models were administrated with or without PA to determine its effects *in vivo*. ROS fluorescence probes were used to detect the ROS intensity both in the cells and bone tissues. **Results.** PA was found to inhibit osteoclast formation, osteoclast-specific genes expression, and resorptive activity in a dose-dependent manner. Mechanistically, these effects were achieved by suppressing receptor activator of nuclear factor-κB (RANKL)-induced mitogen-activated protein kinases (ERK, p38, and JNK) and NF-κB pathway, which lead to the downregulation of NFATc1, the master transcriptional regulator of osteoclastogenesis. Additionally, PA was further determined to significantly attenuate the RANKL-induced intracellular ROS production in pre-osteoclasts. *In-vivo* results indicated significantly increased ROS level in the bone marrow microenvironment as well as dramatically increased numbers of osteoclasts on the bone surface, which caused bone loss in the OVX model group; whereas PA supplementation could effectively prevent these changes. **Conclusions.** PA may be a novel alternative therapy for osteoclast-related bone disease such as osteoporosis by acting as a ROS scavenger, as evidenced by *in-vitro* and *in-vivo* experiments.

Identification and Validation of a Diagnostic Autoantibody Signature for Primary Cutaneous Melanoma

P. Zaenker[1], J. Lo[2], R. Thornton[3], R. Pearce[4], L. Cowell[5], M. Lee[6], C. Quirk[7], H. Law[8], E. Gray[1], M. Ziman[1, 9]

[1]School of Medical & Health Sciences & [2] School of Science, Edith Cowan University, [3] School of Medicine, UWA [4] Hollywood Specialist Centre, Hollywood Private Hospital,[5] Level 1 Melanoma, [6] St John of God Hospital, [7] Dermatology Specialist Group, Ardross, [8] Skin Check WA, Inglewood, [9]Department of Pathology & Laboratory Medicine, UWA

Introduction: Elevated levels of autoantibodies in the serum of cancer patients have been associated with the presence of various cancer types and may serve as biomarkers for cancer diagnosis. The aim of this study was to identify individual serologic autoantibody biomarkers as well as a combination of these to complement the routine diagnosis of cutaneous melanoma patients at early stages. **Procedures:** Peripheral blood and clinical data was collected from early stage melanoma patients (n=104) and healthy volunteers (n=105). Samples were screened against a functional protein microarray to measure IgG responses as potential melanoma autoantibody biomarkers. Extensive statistical analyses including machine learning based approaches were used to determine the reliability of individual and combinations of autoantibody biomarkers. A validation immunoassay was optimised and test samples were diluted 1:20, incubated with the protein-bound beads, washed and then incubated with a R-Phycoerythrin-conjugated goat anti-human IgG secondary antibody. Fluorescence was read using the BioPlex200 platform and IgG titres assigned using the in-house standard reference sera. ROC analysis helped to establish cutoffs and evaluate autoantibody biomarker sensitivity, specificity, AUC for melanoma diagnosis. **Results:** Out of 1627 recombinant proteins immobilised on the microarray, 139 proteins displayed increased seroreactivity in patients with melanoma compared to healthy volunteers, with sensitivity and specificity scores of individual autoantibody biomarkers of 18% and 100% respectively for melanoma diagnosis. To improve sensitivity, a panel of 10 autoantibodies was identified with a combined sensitivity of 79% at 84% specificity for primary melanoma. **Conclusion:** This is one of the first studies identifying autoantibodies in an extensive cohort of melanoma patients relative to healthy volunteers. Sensitivity levels of identified individual melanoma autoantibodies are comparable to results obtained in similar studies in other cancers, whereas the preliminary data of the validation immunoassay shows value as complementary diagnostic tool for the detection of early stage melanomas.

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Dr Torben Kimhofer

Bioinformatics Lecturer
Murdoch University

Metabolic Phenotyping: Technology and Applications

Biography
Dr Kimhofer is an expert in bioinformatics, specifically chemoinformatics, and leads the field in the phenotyping of microbiome interactions in autism spectrum disorders. His research supports other on-going research in Australia characterising population exposures to diet and environmental factors, personalised health care, clinical nutrition and research into cardiovascular, neuroscience and infectious diseases.

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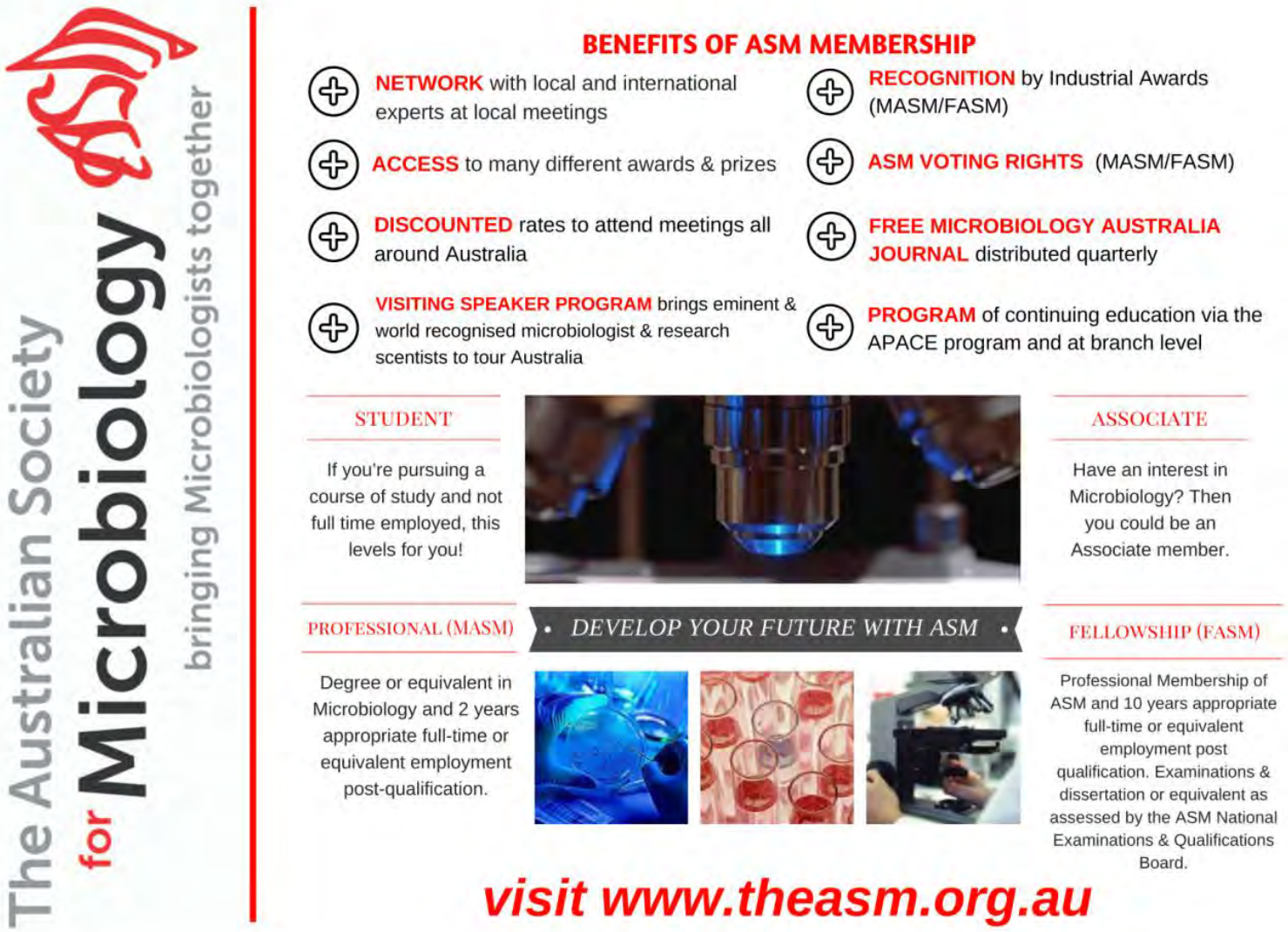

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Keynote Presentation		
Professor Helen Marshall	*Impact of MenB vaccine on meningococcal disease and carriage of Neisseria meningitidis in South Australian adolescents*	1:30
Student Presentations		
Ms Amy Davis	*Re-Evaluating the Role of Restriction Modification Systems as a Barrier to Horizontal Antimicrobial-Resistance Gene Transfer in Staphylococcus aureus*	2:10
Ms Patrice Maher	*The Nuclear Receptors at the Interface of the Gut Microbiota-Host Communication*	2:25
Ms Emily Kibble	*Macrophage Infectivity Potentiator-like Proteins Affect Virulence of Neisseria meningitidis*	2:40
Ms Aleesha Davis	*Is a PITP a novel regulator of encystation in Giardia duodenalis?*	2:55
	Tea Break	3:10
Invited Presentations		
Professor Jeffrey Keelan	*Development of a Microbiological Test for Infection-Associated Preterm Birth*	3:30
Assoc Professor Asha Bowen	*Healthy skin for Aboriginal kids: from bush to bench to bedside*	4:00

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Professor Helen Marshall

Adelaide Medical School
University of Adelaide

Impact of MenB vaccine on meningococcal disease and carriage of Neisseria meningitidis in South Australian adolescents

Abstract

Neiseria meningitidis is the cause of invasive meningococcal disease, primarily affecting young children and adolescents. Exposure to N. meningitidis is common in the general population, leading to asymptomatic pharyngeal carriage with highest carriage prevalence in adolescents. 4CMenB (Bexsero®) is a recombinant protein-based vaccine licensed to protect against invasive group B meningococcal disease, but its role in preventing transmission by impacting on carriage and inducing population protection is uncertain. In a cluster randomized controlled trial, 237 schools in South Australia were randomised to 4CMenB vaccination at baseline (intervention) or 12 months (control), for year 10-12 students (aged 15-18 years). Primary outcome was oropharyngeal carriage of disease-causing N. meningitidis (groups A,B,C,W,X,Y) in year 10/11 students, identified by both porA and genogroup PCR assays. Risk factors for carriage were assessed at baseline. During April-June 2017, 24,269 year 10/11 students and 10,220 year 12 students were enrolled in the study. At 12 months, there was no difference in carriage prevalence of disease-causing N. meningitidis between vaccinated (2.55%; 326/12746) and control (2.52%; 291/11523) students (aOR =1.02; 95%CI 0.80, 1.31; p=0.85). At baseline, significant risk factors for carriage of disease-causing N. meningitidis included year of schooling, current upper respiratory tract infection, smoking cigarettes, smoking a water-pipe, attending pubs/clubs and recent intimate kissing. Carriage prevalence of all N. meningitidis in Aboriginal adolescents was twice that of non-Aboriginal adolescents (6.8% vs 3.4%). In an exploratory post-hoc analysis, a 29% reduction in non-typeable N. meningitidis was identified in the vaccinated group compared to control group (1.65% vs 2.23%; aOR=0.71; p=0.008). Acquisition of invasive genogroups was the same in vaccinated (2.1%) and unvaccinated (2.1%) groups. Vaccine impact against all meningococcal disease in adolescents 17-19 years of age was 71.4% (95%CI 29.0%, 96.3%). 4CMenB protected against meningococcal disease but did not impact on carriage of disease causing N. meningitidis. Therefore 4CMenB immunisation strategies should focus on direct (individual) protection against invasive group B meningococcal disease. A MenB vaccine program has been introduced in South Australia based on direct protection in age groups with highest rates of disease.

Biography

Professor Marshall is a medical researcher and NHMRC Practitioner Fellow. She is Professor in Vaccinology in the Adelaide Medical School and is the Deputy Director, Clinical and Translational Research of the Robinson Research Institute at the University of Adelaide, Senior Medical and Public Health Practitioner and Director of the Vaccinology and Immunology Research Trials Unit at the Women's and Children's Hospital. She has been a member of the Australian Technical Advisory Group on Immunisation and has a research program in vaccine preventable diseases and prevention through immunisation. In recognition of her research leadership and translation she was awarded NHMRC "10 of the Best" in 2016, the South Australia Science Award for Excellence in Research for the Public Good in 2011 and Excellence in Research Collaboration in 2019.

See https://researchers.adelaide.edu.au/profile/helen.marshall

Re-Evaluating the Role of Restriction Modification Systems as a Barrier to Horizontal Antimicrobial-Resistance Gene Transfer in *Staphylococcus aureus*

Amy Davis[1], Karina Yui Eto[1], Geoffrey Coombs[2], Neville Firth[3] and Joshua Ramsay[1]

[1]Curtin Health Innovation Research Institute, Faculty of Health Sciences, Curtin University. [2]School of Veterinary and Life Sciences, Murdoch University. [3]School of Life and Environmental Sciences, University of Sydney

Staphylococcus aureus is a highly clonal species and can be divided into lineages – or clonal complexes – based on point mutations in the core genome. Most lineages carry one or more distinct variant of a Type I restriction-modification (RM) system (HsdR) and these are generally viewed as a major barrier to horizontal gene transfer (HGT) between lineages. Indeed, deletion of *hsdR* genes greatly increases the efficiency of DNA electroporation in laboratory experiments. To evaluate the extent that these RM systems disrupt natural HGT mechanisms, conjugation experiments and bacteriophage plaque assays were carried out using RM proficient and deficient strains. Representatives of three distinct conjugative plasmid families were transferred to wildtype and RM-deficient mutants of the three community-associated methicillin-resistant *S. aureus* (CA-MRSA) strains MW2, MRSA252 and JKD6159. pSK41 and pWBG749-family conjugative plasmids exhibited high rates of conjugative transfer (10^{-2}-10^{-3}) to all three CA-MRSA strain. Less than a 10-fold increase was observed for conjugation to the RM-deficient strains, which in most cases was not statistically significant. Only the pWBG4-family conjugative plasmid exhibited a 100-fold increase in transfer to the RM-deficient derivative of JKD6159, which carries deletions in two RM systems. Bacteriophage plaque assays with Phage K revealed the RM systems inhibited plaque formation around 100-1000-fold, but did not abolish phage infection. These observations conflict with the generally accepted role of RM systems as a major barrier to gene transfer in *S. aureus*. We propose that RM systems do not present a complete barrier to gene transfer in *S. aureus* to and that they are particularly ineffective in blocking the most common conjugative plasmids responsible for dissemination of antimicrobial resistance.

The Nuclear Receptors at the Interface of the Gut Microbiota-Host Communication

Patrice Maher[1,2], Jose A. Caparros-Martin[1,2], Fergal O'Gara[1,2,3]

[1]Human Microbiome Programme, School of Pharmacy and Biomedical Sciences, Curtin University, Australia, [2]Curtin Health Innovation Research Institute (CHIRI), Curtin University, Australia, [3]BIOMERIT Research Centre, School of Microbiology, University College Cork Ireland.

Communication is essential in living organisms. This fact is critical in the case of ecological communities, in which communication allows adaptation in response to environmental changes. The human body is home of complex communities of microorganisms, which are referred as to the human microbiota. In the recent years, evidence has continued to emerge on the role of these communities in the maintenance of human health and the development of metabolic disorders such as diabetes and cardiovascular disease. A necessity has emerged in favour of identifying the molecular pathways and mechanisms regulating the crosstalk host-microbiota in homeostatic conditions, in order to develop targeted interventions to reverse the microbiota-mediated disease-related phenotypes. Our recent research has highlighted the importance of a group of proteins called nuclear receptors, in regulating both human and microbiota metabolism. In this project we have gained insight into how this regulation occurs. Using mice models (wild type and double knock-out in each treatment group), we have investigated how activation of this group of proteins in the liver affects host-microbiota homeostasis. Processing the genetic material from our models through gut microbiota profiling and analysis as well as conducting a functionality profile enabled a broader understanding of the possible effects of activation of these proteins. Our preliminary results indicate that there is a correlation between activation of nuclear receptors and physiological changes in the host. This systemic response also can be associated with both changes in the functionality of the gut microbiota and alterations of the metabolism in the host. Our data suggests that modulation of the activity of these nuclear receptors in the liver could promote specific gut microbiota functional profiles to reinforce their effect on host metabolism.

Macrophage Infectivity Potentiator-like Proteins Affect Virulence of *Neisseria meningitidis*

Emily Kibble[1,2], Geoffrey Coombs[1], Charlene Kahler[2], Mitali Sarkar-Tyson[2]

[1] Antimicrobial Resistance and Infectious Diseases Research Laboratory, Murdoch University, [2] The Marshall Centre for Infectious Diseases Research and Training, School of Biomedical Science, University of Western Australia

Neisseria meningitidis is the bacterial causative agent of invasive meningococcal disease (IMD). The rate of IMD in Australia is increasing, with 2017 showing the highest infection rates since 2007. Macrophage infectivity potentiator (Mip) proteins exhibit peptidyl-prolyl *cis/trans* isomerase activity and are found in a wide range of pathogens. *N. meningitidis* encodes for two putative Mip-like proteins, which is uncommon in comparison to other pathogens which encode for a single Mip protein. Previous work has shown presence of one Mip protein to be important for survival of *N. meningitidis* in whole human blood. It is hypothesised both Mip-like proteins encoded by *N. meningitidis* are important novel anti-virulence targets.Three insertional deletion mutants have been created in the *N. meningitidis* strain NMB; two single mutants, each lacking one of the two putative Mip proteins (NMBΔ*mip1* and NMBΔ*mip2*), and one double mutant (NMBΔ*mip1*Δ*mip2*), lacking both putative Mip proteins. All three mutant strains have been assessed for growth at sub-optimal temperatures and survival within a range of host cells. Deletion of the putative Mip proteins has resulted in decreased survival of *N. meningitidis* at high temperatures. Adhesion of mutant strains to host epithelial cells is also impaired, with attachment rates of NMBΔ*mip1*, NMBΔ*mip2* and NMBΔ*mip1*Δ*mip2* decreased by 50%, 30% and 53% respectively, when compared to the control strain NMB. Reduced survival of mutant strains is observed in macrophages, with survival of NMBΔ*mip1*, NMBΔ*mip2* and NMBΔ*mip1*Δ*mip2* decreased by 69%, 79% and 91% respectively. Both recombinant Mip1 and Mip2 proteins are correctly folded and enzymatically active. This indicates both Mip-like proteins are important in *N. meningitidis* to resist macrophage killing, for epithelial cell attachment and survival, as well as growth at sub-optimal temperatures. Mip-like proteins represent important anti-virulence targets in *N. meningitidis*.

Is a PITP a Novel Regulator of Encystation in *Giardia duodenalis*?

Aleesha Davis[1,2], Dr. Carl Mousley[1,2], Dr. Rob Steuart[2]

[1]School of Pharmacy and Biomedical Sciences, [2]Curtin Health Innovation Research Institute, Curtin University, Perth, WA

Giardia duodenalis is an intestinal parasite with an estimated 280 million symptomatic cases annually worldwide. *Giardia* has a simple two step life-cycle; the trophozoite and the environmentally resistant cyst. The cyst is produced through the process of encystation. This process involves the production, synthesis and secretion of cyst wall proteins (CWPs) forming an extracellular matrix, conferring environmental resistance to the cyst. The trafficking of these CWPs via encystation specific vesicles (ESVs) is the only known regulated export pathway in *G. duodenalis*. Whilst *G. duodenalis* has no known Golgi apparatus, ESVs are considered to perform a similar function. Whole cell proteomic analysis has identified the expression of a putative phosphatidylinositol transfer protein (PITP) to be elevated during encystation, however not much is known about PITP function in *G. duodenalis*, through studies in other eukaryotes indicated that this expression profile is not coincidental. It is hypothesized that *Gd* PITP dependent PtdIns-4-P production is essential to promote trafficking of CWPs to the cell periphery during the developmental progression from the trophozoite to the environmentally resistant cyst. Preliminary data from our lab determined the increased gene expression of *Gd* PITP during encystation. Additionally an expression profile has been generated using a heterologous expression system for PITP and *Gd* PITP is found to be toxic to numerous mutants defective in PtdInd-4-P synthesis and the phenotypes are reminiscent of that determined for a unique PITP, sfh3. Furthermore a system has been generated to determine the lipid binding activity of *Gd* PITP to further investigate the function of this protein. As little is known about the molecular mechanisms that control encystation in G. duodenalis, understanding these mechanisms may act as a potential target for pharmaceutical intervention, diminishing the ability of *G. duodenalis* to successfully complete encystation, therefore ablating environmental resistance.

Invited Presentation

Professor Jeffrey Keelan

Division of Obstetrics & Gynaecology,
The University of Western Australia

Development of a Microbiological Test for Infection-Associated Preterm Birth

Abstract

Jeffrey A Keelan[1,2,3,] Matthew S Payne[1,3,] Dorota A Doherty[1,3] and John P Newnham[1,3]

[1]Division of Obstetrics & Gynaecology and [2]School of Biomedical Sciences, University of Western Australia; [3]Women and Infants Research Foundation

Introduction. Preterm birth is a multifactorial syndrome; 20-40% of all early deliveries are caused by ascending intrauterine infection (IUI), particularly those born more than 2 months preterm. A number of microorganisms have been identified in the vaginal mucosa that can cause IUI, yet translating this knowledge into improved diagnostics for identifying women at risk of preterm birth has remained elusive. **Problem Statement.** We set out to identify a vaginal microbial signature that identifies women at high risk of spontaneous preterm birth (sPTB) in early-/mid-pregnancy. **Procedures**. A thousand asymptomatic pregnant women (16-22 weeks' gestation, singleton pregnancies) were recruited from antenatal clinics at KEMH; vaginal swabs were collected, DNA extracted and vaginal microbial profiles generated at the species, serovar and clade level using targeted PCR assays and custom PCR arrays (n=25 targets). Obstetric outcomes were then used to identify bacterial signatures that were predictive of preterm birth. **Results**. No individual microbial target was indicative of increased PTB risk. Women with high levels of protective lactobacilli (*L. crispatus, L. gasseri or L. jensenii*) were at very low risk of PTB. However, in women deficient in these lactobacilli, a combination of *G. vaginalis* (clade 4), L. iners and *U. parvum* (serovars 3 or 6), which we termed 'GLU positive', was indicative of significantly increased risk of sPTB. Risk prediction was improved if *F. nucleatum* detection was included in the GLU test algorithm. The final GLU test algorithm predicted 39% of sPTBs and 48% of early sPTBs <34 weeks, with odds ratios of 3.1 and 4.6, respectively. **Conclusions**. GLU-positive singleton women in mid-pregnancy are at increased risk of sPTB and may benefit from targeted antimicrobial therapy. A randomised clinical trial is now underway to determine the clinical efficacy of screening and treating women based on their GLU test status.

Associate Professor
Asha Bowen

Telethon Kids Institute

Healthy skin for Aboriginal kids: from bush to bench to bedside

Biography

Associate Professor Asha Bowen is a clinician scientist working at Perth Children's Hospital as a paediatric infectious disease specialist and Head of Skin Health at the Telethon Kids Institute. Her clinical role informs her research. She was awarded her PhD at the Menzies School of Health Research in Darwin in 2015, a large randomized controlled trial on the treatment of impetigo in remote Indigenous children to find a better treatment. Dr Bowen is the lead investigator for the SToP trial to "see, treat and prevent" skin infections, a large cluster randomised trial with a stepped wedge design in the Kimberley, WA. This trial will commence following several years of community engagement to ensure the study is guided by the Aboriginal co-investigators and community members. Asha and her team recently launched the inaugural National Healthy Skin Guideline to guide clinicians in the recognition and evidence-based treatment of skin infection. Asha is the Deputy Chair of the Australian and New Zealand Paediatric Infectious Diseases committee and a member of the Australasian Society of Infectious Diseases clinical research network.

https://www.telethonkids.org.au/contact-us/our-people/b/asha-bowen/

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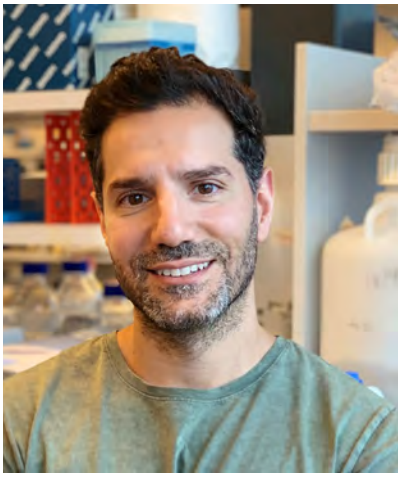
Keynote Presentation		
Dr Michael Lazarou	*Sending bad mitochondria to the garbage disposal: Insights into PINK1/ Parkin mitophagy*	1:30
Student Presentations		
Ms Janya Grainok	*Antisense Oligomer-mediated Functional Disruption of CNOT3 to Treat PRPF31-associated Retinitis Pigmentosa 11*	2:10
Mr Samuel Montgomery	*IL-1 Is Associated with Epithelial Necrosis in Cystic Fibrosis Airways Following Rhinovirus Infection*	2:25
Ms Emma Panting	*Endogenous glucocorticoids are an important regulator of mouse placental development and hemodynamic function in late gestation*	2:40
Mr Heng Qiu	*RANKL M199 residue is a structural determinant critical for osteoclast differentiation and signalling activation.*	2:55
	Tea Break	3:10
Invited Presentations		
Dr Sébastien Malinge	*Developmental and lineage specificity in Down syndrome leukaemia: a key role for the kinase DYRK1A*	3:30
Dr Yu Yu	*Translating Phosphoproteomics Data for New Therapy in Recurrent High-Grade Serous Ovarian Carcinoma*	4:00

Notes



Dr Michael Lazarou

Monash University

Sending bad mitochondria to the garbage disposal: Insights into PINK1/Parkin mitophagy

Abstract

Mitochondrial dysfunction is a major contributor to the pathogenesis of Parkinson's disease. PINK1 and Parkin are proteins mutated in familial Parkinson's disease, which function to maintain a healthy population of mitochondria. PINK1 and Parkin do this by removing damaged mitochondria through a selective form of garbage disposal termed mitophagy. Removal of damaged mitochondria maintains cellular and organismal health by maintaining mitochondrial energy supply, preventing oxidative stress, and by preventing the release of mitochondrial factors that cause cell death and inflammation. PINK1 and Parkin drive mitophagy by selectively tagging damaged mitochondria to trigger their encapsulation by a double membrane structure called an autophagosome. Autophagosomes deliver damaged mitochondria to lysosomes where they are degraded. Despite the importance of PINK1/Parkin mitophagy, little is known about how autophagosomes are built around damaged mitochondria and how autophagy-related (Atg) proteins function during mitophagy. The mechanisms behind how damaged mitochondria are targeted for mitophagy by PINK1 and Parkin will be discussed, with a focus on the molecular steps that govern autophagosome formation.

Biography

Michael's research interests are focused on autophagy pathways and various aspects of mitochondrial biology in health and disease. Michael was awarded his PhD in 2008 from La Trobe University studying the assembly of membrane protein complexes in mitochondria and their defects in energy generation disorders. In 2010, Michael conducted his post-doctoral research studies at the National Institutes of Health (USA) where he worked on mitochondrial dysfunction in Parkinson's disease. His research focused on the Parkinson's disease proteins PINK1 and Parkin and their role in maintaining mitochondrial health via selective autophagy. The work led to Michael receiving the 2013 ASBMB Boomerang Award, and in 2014 he returned to Australia to join the Department of Biochemistry and Molecular Biology at Monash University where he is currently an ARC Future Fellow.

Antisense Oligomer-mediated Functional Disruption of CNOT3 to Treat *PRPF31*-associated Retinitis Pigmentosa 11

Janya Grainok[1,2], Ianthe L Pitout[1], Steve D Wilton[1], May T Aung-Htut[1], Chalermchai Mitrpant[2], Fred K Chen[3] and Sue Fletcher[1]

[1]Centre for Molecular Medicine and Innovative Therapeutics, Murdoch University & Perron Institute, [2]Faculty of Medicine Siriraj Hospital, Mahidol University, Thailand, [3]Centre for Ophthalmology and Visual Science (Incorporating Lions Eye Institute)

Introduction. Heterozygous mutations in pre-mRNA processing factor 31 (*PRPF31*) cause retinitis pigmentosa 11 (RP11) and lead to retinal cell death and blindness. RP11 features incomplete penetrance within affected families, due to variable expression levels of functional *PRPF31* from the remaining healthy allele. CCR4-NOT transcription complex subunit 3 (CNOT3) is a negative transcriptional regulator of *PRPF31*, and is found at higher levels in RP11 patients, compared to asymptomatic family members carrying the same *PRPF31* mutations. **Problem Statement**. It is hypothesized that lowering CNOT3 activity will enhance transcription of the remaining healthy *PRPF31* allele and improve pre-mRNA splicing function to prevent or delay RP11 disease progression. In this study, antisense oligomer mediated modulation of CNOT3 expression and function in order to upregulate functional PRPF31 was evaluated. **Results.** Antisense oligomers were designed to target selected *CNOT3* exon(s) for exclusion during pre-mRNA processing in order to induce a frame shift and downregulate CNOT3 expression. As a consequence of *CNOT3* knockdown, PRPF31 expression was upregulated 1.8-fold and 1.2-fold at the mRNA and protein levels, respectively, in induced pluripotent stem cell-derived retinal pigment epithelium from an RP11 patient. Alternatively, targeting in-frame exon(s) encoding essential functional domains resulted in production of truncated CNOT3 isoforms. Consequently, PRPF31 mRNA and protein expression was increased up to 2.2-fold and 1.6-fold, respectively, which is above the predicted therapeutic threshold. Primary cilia that are crucial for complete retinal pigment epithelium maturation were assessed to evaluate PRPF31 function. Treated cells showed a significant increase in cilia incidence and length, compared to untreated RP11 patient cells. **Conclusions.** This study provides antisense oligomer sequences to downregulate or inhibit function of CNOT3, allowing PRPF31 transcription to increase to a level expected to provide functional benefit. Transcriptome analysis will be used to assess pre-mRNA splicing function resulting from increased PRPF31 expression in RP11 patient retinal cells.

IL-1 Is Associated with Epithelial Necrosis in Cystic Fibrosis Airways Following Rhinovirus Infection

Samuel T. Montgomery[1], AS Dittrich[2,3], LW Garratt[4], DL Frey[2,3], SM Stick[1,4,5], MA Mall[2,7,8], A Kicic[1,4,5,6] & AREST CF[1,5,9,10]

[1]University of Western Australia [2]University of Heidelberg, & [3]University Hospital Heidelberg, Germany [4]Telethon Kids Institute [5]Perth Children's Hospital [6]Curtin University [7]Charité Institute, Berlin, Germany [8]Berlin Institute of Health, Germany [9]Murdoch Children's Research Institute [10]University of Melbourne

Introduction: Necrosis of airway epithelial cells (AEC) resulting in airway inflammation driven by interleukin (IL)-1 is a characteristic finding in cystic fibrosis (CF), driven by mucus obstruction of the airway and a response to human rhinovirus (RV) infection. As little is known about IL-1 and pathogenesis of CF lung disease and RV is a common early life infection, this study aimed to assess cellular and inflammatory responses of CF and non-CF AEC infected with RV, with the hypothesis that rhinovirus infection drives epithelial necrosis and inflammation in the CF airway. **Procedures/Data/Observations**: CF and non-CF AEC (n=9) were infected with RV (MOI 3) for 24 hours and viable, necrotic and apoptotic events assessed via flow cytometry (% total events). IL-1α, IL-1β, IL-1Ra and IL-8 were measured in cell culture supernatants (pg/mL). Friedman and Mann-Whitney tests were used to test for significant differences and Spearman for correlations ($p<0.05$). **Results**: RV infection resulted in lower viable events in non-CF AEC ($p<0.05$), increased necrotic events in non-CF and CF AEC ($p<0.05$) and increased apoptotic events in non-CF AEC ($p<0.05$). RV infection also increased IL-1α and IL-1β protein in non-CF ($p<0.05$) and CF AEC ($p<0.05$) supernatant. IL-1α and IL-1β in supernatant positively correlated with necrosis ($r=0.80$ & $r=0.77$ respectively; $p<0.0001$) in CF but not non-CF AEC after RV infection. RV infection increased IL-1Ra protein in non-CF ($p<0.05$) and CF AEC ($p<0.05$) supernatant, although IL-1Ra was higher in non-CF AEC ($p<0.05$). RV infection increased IL-8 protein in non-CF and CF AEC ($p<0.05$) which correlated with IL-1α in non-CF and CF AEC ($r=0.63$ & $r=0.74$ respectively; $p<0.0001$). **Conclusions**: RV infection of CF and non-CF AEC increased necrotic events and in CF AEC was associated with IL-1, suggesting this pathway as a novel anti-inflammatory target for early CF disease.

Endogenous Glucocorticoids are an Important Regulator of Mouse Placental Development and Hemodynamic Function in Late Gestation

Emma N Panting[1,2], Jessica R Ivy[2], Adrian Thomson[2], Carmel M Moran[2], Karen E Chapman[1,2], Caitlin S Wyrwoll[1]

[1]School of Human Sciences, The University of Western Australia; [2]Centre for Cardiovascular Science, The Queen's Medical Research Institute, The University of Edinburgh

Maturation of fetal tissues requires glucocorticoid binding to the glucocorticoid receptor (GR). GR is also expressed in the placenta, though a functional role in placenta is not established. We hypothesised that glucocorticoids act via GR to modify placental function to meet fetal demand, particularly in late gestation. Here, we tested the effect of GR deletion upon placental development and hemodynamic function. Heterozygous GR mice, with C57BL/6J background, were time-mated to produce litters of WT, Het and GRKO fetuses. The morning of vaginal plug was designated E0.5. *In vivo* pulsed-wave Doppler ultrasound scanning was conducted at E14.5 or E17.5 to measure umbilical artery (UA) blood flow and fetal heart function. Immediately after scanning, tissues were collected. Data were analysed by two-way ANOVA with post-hoc Bonferroni's test. Global GR ablation did not alter fetal wet weight at E14.5 or E17.5, although placental wet weight was increased at E17.5 ($p=0.003$). At E14.5, UA blood flow was largely unchanged. Conversely, at E17.5, GRKO exhibited reduced UA systolic/diastolic ratio ($p=0.002$) and diastolic flow was detectable in only 38% of GRKO compared to 83% of WT. UA resistance index was increased in GRKO ($p=0.0004$). Moreover, whilst reversed end-diastolic flow (REDF) was undetectable in WT by E17.5, it persisted in GRKO. Minimal effects were seen on fetal heart function. GR ablation increased placental weight in late gestation, when endogenous glucocorticoid levels peak, perhaps reflecting the removal of normal growth inhibitory effects of glucocorticoids. Further, the compromised UA blood flow associated with persistence of REDF and high resistance index in GRKO indicates a functionally immature placenta at E17.5. Interestingly, despite impaired placental blood flow, fetal wet weight was maintained in GRKO. This is consistent with our previous report of fetal oedema in GRKO. Investigation is underway to determine how absent GR signalling affects placental morphology and fetal-placental interactions.

RANKL M199 Residue is a Structural Determinant Critical for Osteoclast Differentiation and Signaling Activation.

Heng Qiu[1#], An Qin[2#], Jiake Xu[1*]

[1]School of Biomedical Sciences, University of Western Australia, [2]Shanghai Key Laboratory of Orthopaedic Implant, Shanghai Jiao Tong University School of Medicine, Ninth People's Hospital, China;

Introduction: Bone undergoes constant remodelling maintained by osteoclasts and osteoblasts, while receptor activator of NF-κB ligand (RANKL) is indispensable for osteoclastogenesis. A naturally occurring mutation of human RANKL at residue 199 was described in patients with osteoclast-poor autosomal recessive osteopetrosis (ARO). However, how this mutation affects RANKL function has not been characterized. **Problem Statement**: We hypothesized that M199 residue is a structural determinant for osteoclast formation. **Procedures**: Site-directed mutagenesis was employed to create three rat RANKL mutants, replacing the M200 (rat M199 equivalent residue) with either lysine (M200K), alanine (M200A) or glutamic acid (M200E). MTS was used to measure the cellular toxicity. Cell culture, luciferase reporter assay, RT-PCR, confocal microscopy, western blot, calcium oscillation detection and computational methods were also used to investigate the biological effect of rRANKL mutants. Differential scanning fluorimetry, western blot and protein binding affinity experiments were later carried out for structural analyses. **Results**: M200s showed reduced ability to induce osteoclast formation, osteoclastic polarization and bone resorption. Additionally, M200s did not competitively inhibit the osteoclastogenic effect of rRANKL. M200s are incapable of activating osteoclast markers with a diminished induction of IκB degradation, inactivated signaling of NF-κB, NFATc1, AP-1 and ARE and impaired calcium oscillation. Analysis of the crystal structure of RANKL revealed that M199 is located within the hydrophobic core of the protein thus likely to be critical for protein folding and stability. Further, Western blot analyses showed an impaired RANKL trimerization after mutation. Differential Scanning Fluorimetry of M200s suggested that the proteins were significantly less stable than rRANKL. Protein binding affinity assays revealed that M200s display interrupted interaction to its intrinsic receptors. **Conclusion**: Collectively, our data showed that M199 in human is a structurally-sensitive residue for RANKL's function, and may represent a therapeutic motif for new anti-resorptive drugs.

Dr Sébastien Malinge

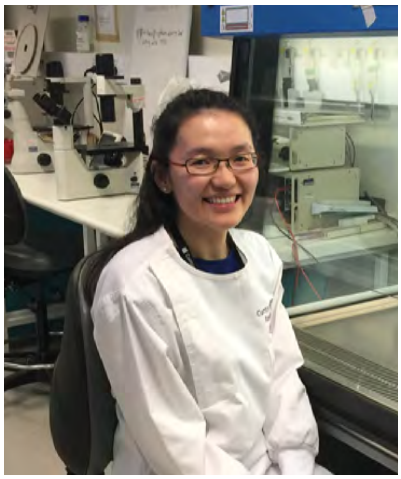
Telethon Kids Institute

Development and lineage specificity in Down syndrome leukaemia: a key role for the kinase DYRK1A

Biography

Dr Sébastien Malinge is a Senior Research Fellow highly qualified in the field of paediatric leukaemia, with a strong background in the mechanisms of leukaemia predisposition and development in children with Down syndrome. He obtained his PhD in 2006 in France, at Paris children's hospital (Necker-Enfants Malades Hospital), and subsequently joint Northwestern University (Chicago, USA) as a research fellow awarded by the Leukaemia and Lymphoma Society. In 2012, he became an independent investigator at the French National Institute for Health and Medical Research (INSERM), located at Gustave Roussy, one the first Cancer Centre in Europe. In 2017, Dr Malinge relocated to the Telethon Kids Institute - Cancer Centre, as the Children`s Leukaemia and Cancer Research Foundation (CLCRF) – U. Kees fellow, and became co-head of the Leukaemia and Cancer Genetics team in 2018.

Based on a solid background in fundamental research, next generation sequencing and the development of preclinical Patient Derived Xenograft (PDX) models, Dr Malinge is now developing cutting-edge approaches to unravel new vulnerabilities in leukaemia cells, with the view of identifying alternative therapeutic approaches and new effective treatments, to make a tangible difference to the way we treat paediatric leukaemia.



Dr Yu Yu

Curtin University

Translating Phosphoproteomics Data for New Therapy in Recurrent High-Grade Serous Ovarian Carcinoma

Biography

Dr Yu Yu is a Senior Research Fellow at Curtin University. Her research interest is cancer therapeutics with specific goals to pursue advances in the fields of experimental agents and translational oncology for the treatment of recurrent cancers. Dr Yu completed PhD from the University of Sydney studying the use of iron-chelating agents as anti-cancer therapeutics. She then pursued postdoctoral training at the TeLinde Gynecologic Pathology Laboratory at Johns Hopkins University School of Medicine, USA with a focus on understanding the mechanisms of recurrent ovarian tumours and therapeutic strategy to overcome the disease. Her most recent research findings illuminate how tumours developed paclitaxel resistance. The exciting results provided critical evidence for the use of SYK (Spleen Tyrosine Kinase) inhibitor and led to a recent phase I clinical trial combining SYK inhibitor with the dose-dense paclitaxel regimen to treat recurrent ovarian cancers.

Notes

Seminar Room 1

CHAIR: Dr Sarah Rea

Keynote Presentation		
Dr Sam Abraham	*Seagulls and Superbugs: Role of genomics in understanding the spread of antimicrobial resistance*	1:30
Student Presentation		
Ms Jessica Cale	*Rescue of Fibrillin-1 Microfibril Formation in Fibroblasts from Individuals with Marfan Syndrome*	2:10
Ms Adriana Foster	*ALS-associated TBK1 variant p.G175S is unable to phosphorylate p62 Serine residue 403 and fails to promote NF-κB signalling*	2:25
Ms Laetitia Hughes	*Misregulation of Mitochondrial Protein Synthesis Leads to Cardiomyopathy*	2:40
Ms Jessica Cheng	*Regulatory architecture of the RCA gene cluster captures a novel intragenic boundary and enhancer elements in B cells*	2:55
	Tea Break	3:10
Invited Presentation		
Dr Christian Pflüger	*Generating designer cells by epigenome editing*	3:30
Ms Sarah Beecroft	*Targeted gene panels in 2,200 neurology patients: the Australasian referral centre experience*	4:00

Notes

Dr Sam Abraham

Antimicrobial Resistance and Infectious Diseases Laboratory, Murdoch University

Seagulls and Superbugs: Role of genomics in understanding the spread of antimicrobial resistance

Abstract

Antimicrobial resistance is regarded as one of the greatest threats to human and animal health. Resistance to critically important antimicrobials (CIAs) or drugs of last resort such as extended-spectrum cephalosporins (ESCs), fluoroquinolones (FQs), colistin and carbapenems amongst Enterobacteriaceae and other Gram-negative bacteria is a major public health concern. This is attributed to co-associated resistance to other classes of antimicrobials and limited therapeutic options to treat infections with CIA-resistant bacteria in both humans and animals. Globally, emergence and dissemination of CIA-resistance in livestock, wild birds and companion animals are a major concern due to the potential for direct or indirect transfer of such resistant bacteria to humans

Our recent study [1] investigated the ecology, epidemiology and origins of CIA-resistant Escherichia coli carried by Australian Silver Gulls (Chroicocephalus novaehollandiae), a gregarious avian wildlife species that is a common inhabitant of coastal areas with high levels of human contact. Sampling locations were widely dispersed around the perimeter of Australian continent, with sites separated by up to 3,500 km. Whole-genome sequencing was used to study the diversity and molecular characteristics of resistant isolates to ascertain their epidemiological origin.

Investigation of 562 fecal samples revealed wide spread occurrence of extended-spectrum cephalosporin (21.7%) and fluoroquinolone (23.8%) resistant E. coli. Genome sequencing revealed CIA-resistant E. coli isolates (n=284) from gulls predominantly belonged to human-associated extra-intestinal pathogenic E. coli (ExPEC) clones responsible for causing urinary tract infections and sepsis in humans. This included globally disseminated clones such as ST131 (17%), ST10 (8%), ST1193 (6%), ST69 (5%), ST38 (4%). Comparative genomic analysis revealed that pathogenic and resistant E. coli clones isolated from gulls overlapped extensively with human clinical isolates from Australia and overseas. One isolate from Victoria was resistant to carbapenems and another isolate from Western Australia was resistant to colistin. Carbapenems and colistin are last resort antimicrobials used to treat multidrug-resistant bacterial infections.

This study uniquely establishes that Australian Silver Gulls are carriers of virulent and CIA-resistant human-associated pathogenic E. coli clones. The carriage of diverse CIA-resistant E. coli clones that strongly resemble pathogenic clones from humans, suggests that seagulls can act as ecological sponges indiscriminately accumulating and disseminating CIA-resistant bacteria over vast distances. The study illustrates the broader community risk entailed in transmission of CIA-resistant pathogenic E. coli in a cycle that encompasses humans, seagulls and the environment.

1. Mukerji, S., et al., Resistance to critically important antimicrobials in Australian silver gulls (Chroicocephalus novaehollandiae) and evidence of anthropogenic origins. Journal of Antimicrobial Chemotherapy, 2019. doi:10.1093/jac/dkz242

Biography

Sam Abraham is a Senior Lecturer at Murdoch university. His research focuses on the movement of antimicrobial resistance between humans and animals. He utilises next generation robotics and genomics in clarifying the origin and dispersal of antimicrobial resistant bacteria.

Rescue of Fibrillin-1 Microfibril Formation in Fibroblasts from individuals with Marfan Syndrome

Jessica M Cale[1,2], Sue Fletcher[1,2], Steve D Wilton[1,2]

[1]Centre for Molecular Medicine and Innovative Therapeutics, Murdoch University, [2]Perron Institute for Neurological and Translational Sciences

Introduction: Marfan syndrome (MFS) is one of the most common dominantly inherited connective tissue disorders, affecting 2-3 in 10,000 individuals, and is caused by any one of over 1800 unique fibrillin-1 (*FBN1*) mutations. Such mutations result in production of two different fibrillin-1 monomers that are unable to form functional microfibrils, resulting in destabilisation of the extracellular matrix. Disease management requires invasive surgical intervention and lifelong use of medications aimed at slowing disease progression, thus the need for new therapeutics. We aim to use short, synthetic nucleic acid sequences called antisense oligonucleotides (AOs) to manipulate fibrillin-1 pre-mRNA splicing. *FBN1* exon 59 harbours over 20 unique mutations, encodes one of 43 repeated motifs and its removal does not alter the reading frame. We hypothesise that removing exon 59 will allow production of identical monomers capable of forming functional microfibrils. **Procedures:** AO sequences were optimised using 2′-O-Methyl modified bases on a phosphorothioate backbone (2′OMe-PS), transfected into healthy control and patient fibroblasts. The most effective sequence was synthesised as a phosphorodiamidate morpholino oligomer (PMO), a chemistry shown to be safe and effective clinically. Transfected cells were assessed for fibrillin-1 expression and morphology by immunofluorescent staining. **Results:** Exon 59 was skipped in ~45% of the *FBN1* transcripts in healthy cells transfected with the 2′OMe-PS-AOs, whereas PMO-59 induced exon 59 skipping in healthy (50%), c.7205-2A>G (90%) and p.Arg2414X (80%) patient cells after 10 days in culture. Immunofluorescent staining revealed a corresponding increase in fibrillin-1 microfibrils when target exon skipping was greater than 75%. **Conclusions:** Exon 59 can be efficiently removed from *FBN1* pre-mRNA in fibroblasts from two different MFS patients, resulting in increased microfibril formation. We show proof-of-concept that removal of mutation harbouring exons from *FBN1* pre-mRNA allows the production of internally truncated but identical monomers capable of forming microfibrils, potentially reducing disease severity.

ALS-associated TBK1 Variant p.G175S is Unable to Phosphorylate p62 at Serine Residue 403 and Fails to Promote Autophagy or NF-κB Signalling

Foster, A[1, 2], Rea SL[1, 2]

[1]Harry Perkins Institute of Medical Research, University of Western Australia, [2]Department of Endocrinology and Diabetes, Sir Charles Gairdner Hospital

Introduction. Mutations in *SQSTM1*, coding for p62, and TANK-binding Kinase 1 (*TBK1*) have been implicated in amyotrophic lateral sclerosis (ALS) and frontotemporal degeneration (FTD). TBK1 is a serine-threonine kinase that regulates p62 autophagic activity via phosphorylation of serine residue 403. Loss of TBK1 function can lead to dysregulated autophagy and alterations in mitophagy via decreased phosphorylation of optineurin or p62. TBK1 also induces NF-κB pro-inflammatory signalling. The mechanisms underlying ALS and FTD pathogenesis due to *TBK1* mutations is currently unknown. **Problem Statement**. To determine the effect of ALS-associated variant p.G175S on autophagy and NF-κB signalling. **Methods**. p62-TBK1 interaction was assessed by co-immunoprecipitations, and alterations in Ser-403 and Ser-349 phosphorylation were determined by western blot analysis. p62 association with mitochondria was determined by western blot of isolated mitochondria. Effects on cell signalling were assessed by dual luciferase reporter assays. **Results**. We observed that p.G175S TBK1 retained the ability to bind to p62, however was unable to phosphorylate p62 at Ser-403. We observed that TBK1 wild type promoted downstream phosphorylation of p62 at Ser-349, while p.G175S-expressing cells did not. Consistent with these findings, expression of p.G175S led to decreased autophagy induction compared to wild type TBK1 expression, as well as decreased association of p62 with mitochondria. Expression of TBK1 wild type increased NF-κB signalling ~300 fold in comparison to empty vector cells, whereas p.G175S TBK1 failed to promote NF-κB signalling. **Conclusions**. Here, we demonstrate that the ALS-associated TBK1 variant p.G175S reduces phosphorylation of p62 Ser-403 and the downstream Ser-349. This TBK1 variant does not promote autophagy, and reduces recruitment of p62 to mitochondria. We also demonstrated that p.G175S fails to promote NF-κB signalling, whilst wild type TBK1 increased signalling markedly. Our data suggests that TBK1 mutations impair p62-mediated autophagy and mitophagy and decrease cell signalling, all of which may reduce neuronal survival.

Misregulation of Mitochondrial Protein Synthesis Leads to Cardiomyopathy

Laetitia A. Hughes[1], Danielle L. Rudler[1], Kara L. Perks[1], Tara Richman[1], Irina Kuznetsova[1], Judith A. Ermer[1], Laila N. Abudulai[2], Ann-Marie J. Shearwood[1], Helena M. Viola[3], Livia C. Hool[3], Stephan J. Siira[1], Oliver Rackham[1,4] and Aleksandra Filipovska[1,5]

[1]Harry Perkins Institute of Medical Research; [2]Centre for Microscopy, Characterisation and Analysis, UWA; [3]School of Human Sciences, UWA; [4]School of Pharmacy and Biomedical Sciences, Curtin University; [5]School of Molecular Sciences, UWA.

Mitochondria produce more than 90% of the energy required by our bodies and thereby have a fundamental role in cell and energy metabolism. Mitochondria are composed of proteins encoded by both the nuclear and mitochondrial genomes and the coordinated expression of both genomes is essential for energy production. Impaired energy production leads to mitochondrial dysfunction that causes or contributes significantly to a variety of diseases including metabolic disorders and cardiovascular diseases. Mutations in genes encoding components of the mitochondrial translation machinery are among the most common causes of mitochondrial disease, but little is known about the mechanisms involved in the regulation of mitochondrial translation. Furthermore, how mitochondrial dysfunction leads to heart and metabolic diseases is poorly understood, making it difficult to develop effective treatments. We have created a new model of cardiovascular disease caused by loss of an essential nuclear encoded protein, involved in the regulation of mitochondrial protein synthesis. Loss of this protein is embryonic lethal, so to characterise its molecular role we developed and investigated a homozygous heart- and skeletal-muscle-specific knockout (KO) mouse model. At 25 weeks KO mice weighed approximately 25% less than wild-type (wt) counterparts ($n=6$, $p<0.001$). Echocardiography revealed that KO mice developed dilated cardiomyopathy by 25 weeks, with a significant decrease in fractional shortening, posterior wall and intraventricular septum thickness versus wt mice ($n=4$, $p<0.05$). Experiments to characterise the molecular function, indicate that this protein is an essential regulator of translation rate and fidelity, assembly and function of the electron transport chain, and ultimately heart function. We used transcriptomics and proteomics to provide new insights into the mechanisms controlling translation in mitochondria, and how mitochondrial dysfunction leads to disease.

Regulatory Architecture of the Regulators of Complement Activation (RCA) Gene Cluster Captures a Novel Intragenic Boundary and Enhancer Elements in B cells

Jessica Cheng[1], Joshua S. Clayton[2], José Luis Gómez-Skarmeta[3], Rhonda L. Taylor[2], John B. Harley[4], Elizabeth Quail[1], Daniela Ulgiati[1]

[1]School of Biomedical Sciences, The University of Western Australia, [2]Harry Perkins Institute of Medical Research, [3]Centro Andaluz de Biología del Desarrollo, Universidad Pablo de Olavide, [4]Centre for Autoimmune Genomics and Etiology, Cincinnati Children's Hospital Medical Centre.

Introduction. The Regulators of Complement Activation (RCA) gene cluster comprises of six tandemly-arranged genes on human chr1q32.2 which have close evolutionary relationship and share key functions in the immune system. Recently, an intronic variant (rs1876453) within RCA member, *CR2*, was identified to be associated with an autoimmune disease called Systemic Lupus Erythematosus (SLE). It was shown that rs1876453 altered the expression of the downstream RCA gene, *CR1*, in B cells without influencing *CR2*. Further studies revealed that rs1876453 also altered the binding of a transcription factor called CTCF; a master regulator of chromatin looping. This indicated that these RCA members may be co-regulated by chromatin looping, but this is not yet known. **Hypothesis**. We hypothesised that the RCA gene cluster is transcriptionally co-regulated by chromatin looping in B cell lineage, and chromatin looping regulates the expression of RCA genes by mediating enhancer-gene interactions. **Methods and Results**. Using 4C-seq, we mapped chromatin looping across the RCA gene cluster and between several distal RCA genes in B cells. Importantly, chromatin looping was organised into two domains by a boundary located within RCA gene, *CR1*. We also identified several enhancer-gene interactions across the region and uncovered a novel intergenic enhancer (BEN). Using reporter gene assays and CRISPR deletion, we functionally demonstrated that BEN regulates the transcription of its two adjacent RCA genes in B cells. **Conclusion**. We have identified novel regulatory loci which co-regulate a critical cluster of immune genes through long-range interactions. The effect of disease-associated variants across the RCA may be amplified through these co-regulatory mechanisms and alter expression of multiple genes in parallel. This work illustrate novel mechanisms by autoimmune disease may be exacerbated and emphasises the important contribution of chromatin architecture to complex genetic disease.

Dr Christian Pflüger

Harry Perkins Institute of Medical Research

Generating designer cells by epigenome editing

Abstract

Chemical tags on histones and DNA, known as epigenetic modifications, are vital in regulating transcription and cell identity. Recently, first generation targeted epigenome editing systems based on nuclease-dead Cas9 (dCas9) have been developed to investigate defined changes in the epigenome and their impact on transcription. However, the induced epigenome changes are predominantly transient, and frequently ineffective likely due to differing chromatin states. Consequently, we are implementing a high throughput single cell resolution combinatorial epigenome modifier screen to discover the most effective combinations of transcriptional activators and repressors. Furthermore, we have developed a novel dCas9-based recruitment system that will allow precise recruitment of multiple independent epigenome modifiers to desired genomic regions to confer stable impacts on transcription. This will work will fundamentally advance our understanding of transcriptional regulation and will transform our ability to control the epigenome regulatory layers of the genome, with immense benefits for cell identity manipulation and design.

Ms Sarah Beecroft

The University of Western Australia

Targeted gene panels in 2,200 neurology patients: the Australasian referral centre experience

Abstract

Sarah Beecroft,* Kyle S. Yau, Richard J.N. Allcock, Kym Mina, Rebecca Gooding, Fathimath Faiz, Vanessa J Atkinson, Cheryl Wise, Padma Sivadorai, Daniel Trajanoski, Nina Kresoje, Royston Ong, Rachael M. Duff, Macarena Cabrera-Serrano, Kristen Nowak, Nicholas Pachter, Gianina Ravenscroft, Phillipa J. Lamont, Mark R. Davis, Nigel G. Laing

Neuromuscular diseases are a broad group of mostly heritable disorders that impair voluntary movement. The considerable clinical and genetic heterogeneity of these diseases makes genetic diagnosis challenging. Next-generation DNA sequencing has revolutionised genetic diagnosis, with both targeted gene panels and whole-exome sequencing now commonplace approaches. We report the results from the first two iterations of a targeted panel we designed to provide comprehensive testing of myogenic and neurogenic neuromuscular disorders. The first iteration of the panel included 336 genes, which increased to 464 genes on Version 2. We received >2200 samples from Australia and New Zealand, allowing us to describe and characterise the genetic landscape of neuromuscular disorders in Australasia. Overall, the diagnostic success rate was 1/3, varying by clinical category. Diagnostic success decreased with patient age, suggesting late-onset disease may be less likely to have a monogenic aetiology. Pathogenic variants in 11 genes explained 40% of the disease burden in the cohort. The percentage of autosomal dominant (AD) pathogenic variants in our cohort (50%) is similar to a previously reported outbred Chinese population. Additionally, more than 60% of reported pathogenic variants were non-recurrent, reflecting the diverse population architecture of Australia. Use of a comprehensive gene panel was beneficial for discovering phenotypic expansions. A large number of patients without a diagnosis following analysis using the gene panels were triaged for research exome sequencing, facilitating the discovery of six new disease genes. Overall, the comprehensive panels balanced cost, throughput, and diagnostic yield.

Notes

Seminar Room 2

CHAIR: Dr Charlotte Oskam
Dr Amanda Barbosa

Keynote Presentation		
Dr Nathalie Butt	*Climate change and biodiversity: Migrate, adapt or die*	1:30
Student Presentations		
Alice Michie	*Genome-scale Phylogeny and Evolutionary Analysis of Ross River virus Reveals Periodic Sweeps of Lineage Dominance in Western Australia, 1977 – 2014.*	2:10
Kamil Braima	*Retrospective analysis of Cryptosporidium species in Western Australian human patients (2015-2018), and emergence of the C. hominis IfA12G1R5 subtype*	2:25
Siobhon Egan	*Insights into the ecology of the ornate kangaroo tick, Amblyomma triguttatum*	2:40
Kenny Choo	*Characterising aroma profile and investigating bacterial communities on Australian and Spanish black Périgord truffle (Tuber melanosporum)*	2:55
	Tea Break	3:10
Invited Presentation		
Dr Jennifer Kelley	*How 'Nemo' got his stripes*	3:30
Dr Alison Ritchie	*The Use of Innovative Seed Enhancement Technologies to Overcome Barriers to Restoration Success In A Biodiversity Hotspot*	4:00

BacPath 15

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Timothy Atkins, DSTL | Paal Skytt Andersen, Staten Serum Institute
Richard Titball, University of Exeter

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Committee Members
Charlene Kahler, Geoff Coombs, Mitali Sarkar-Tyson, Josh Ramsay, Dan Knight, Nicole Bzdyl

Keynote Presentation

CBSM CHAIR: Dr Charlotte Oskam

Dr Nathalie Butt

University of Queensland

Climate change and biodiversity: Migrate, adapt or die

Abstract

Biodiversity faces many threats, of which climate change is one. But how will it respond to climate change, and what can we do about it? Species' ecophysiology determines how they interact with and respond to their environments, and I will explore these interactions, describe what climate change looks like, in terms of changes in both climatic means and extremes, and discuss how we can predict impacts on biodiversity. Given that species exist where conditions are climatically suitable for them, what happens when the climate changes? Species are vulnerable to climate change in terms of both the climate exposure and their own characteristics. Vulnerability is determined by three factors, exposure, sensitivity, and adaptive capacity. Of these, exposure is extrinsic and fixed, while sensitivity and adaptive capacity are intrinsic and governed by species' traits. Together they determine a species' response capacity to climate change, and knowledge of trait-based vulnerability can help us predict or describe impacts, and inform more effective conservation management.

Biography

https://researchers.uq.edu.au/researcher/2743

Genome-scale Phylogeny and Evolutionary Analysis of Ross River virus Reveals Periodic Sweeps of Lineage Dominance in WA, 1977 – 2014

Alice Michie[1], Vijaykrishna Dhanasekaran[2], John S. Mackenzie[3], David W. Smith[3], Allison Imrie[1,3]

[1]School of Biomedical Sciences, University of Western Australia, Western Australia, [2]Biomedicine Discovery Institute, Monash University, Victoria, [3]PathWest Laboratory Medicine Western Australia

Ross River virus (RRV) is the most medically significant mosquito-borne virus of Australia, with an average 5000 clinical cases reported per annum. In Western Australia (WA), surveillance of RRV is based on the isolation and identification of viruses from the homogenate of pooled and trapped mosquitoes. Routine and opportunistic mosquito trapping for surveillance purposes has been conducted throughout WA since the mid-1980s. Isolates have been classically identified using monoclonal antibodies, and more recently, RT-PCR. Past RRV phylogenetic and evolutionary analyses have been based on a small collection of partial genomes only. Three geographically distinct RRV lineages: the Eastern, the Western and the supposedly extinct North-Eastern lineage have been classified. We sought to expand on past phylogenies through robust genome-scale analysis to better understand RRV genetic diversity and evolutionary dynamics. We analysed 106 RRV whole genome sequences which included 12 publicly available sequences and 94 novel sequences derived for this study, sampled throughout WA (1977 – 2014) and during the substantial Pacific Islands RRV epidemic (1979 – 1980). Our final dataset comprised isolates sampled over a 59 year period (1959 – 2018), from a range of locations. Four distinct genotypes (G1-4) were defined, with the newly described G4 found to be the contemporary lineage in circulation. The prior geographical classification of RRV lineages was not supported by our findings, with evidence of geographical and temporal co-circulation. Bayesian Markov Chain Monte Carlo (MCMC) analysis revealed that RRV lineages diverged from a common ancestor approximately 94 years ago, with distinct lineages emerging roughly every 10 years over the past 50 years in periodic bursts of genetic diversity. Our study has enabled a more robust analysis of RRV evolutionary history, and resolved greater genetic diversity than previously defined by partial genome analysis.

Retrospective Analysis of *Cryptosporidium* Species in Western Australian Human Patients (2015-2018), and Emergence of the *C. Hominis* Ifa12g1r5 Subtype

Kamil Braima[1], Alireza Zahedi[1], Charlotte Oskam[1], Simon Reid[2], Nevada Pingault[3], Lihua Xiao[4], Una Ryan[1]*.

[1]Vector and Waterborne Pathogens Research Group, Murdoch University, Perth, Australia [2]The University of Queensland, Australia [3]Department of Health, Perth, Western Australia, Australia [4]South China Agricultural University, Guangzhou, China

Cryptosporidium species are a major cause of diarrhoea worldwide. In the present study, a retrospective analysis of 109 microscopically *Cryptosporidium*-positive faecal specimens from Western Australian patients with gastrointestinal symptoms, collected between 2015 and 2018 was conducted. Sequence analysis of the 18S rRNA and the 60 kDa glycoprotein (*gp60*) gene loci identified four *Cryptosporidium* species; *C. hominis* (86.2%, 94/109), *C. parvum* (11.0%, 12/109), *C. meleagridis* (1.8%, 2/109) and *C. viatorum* (0.9%, 1/109). Subtyping at the *gp60* locus identified the emergence of the previously rare *C. hominis* IfA12G1R5 subtype in 2017 as the dominant subtype (51.2%, 21/41). This subtype has also recently emerged as the dominant subtype in the United States but the reasons for its emergence are unknown. This is also the first report of *C. viatorum* in humans in Australia and a novel subtype variant (XVaA3g) was identified in the one positive patient.

Insights into the Ecology of the Ornate Kangaroo Tick, *Amblyomma triguttatum*

Siobhon Egan[1], Joe Fontaine[2], Willa Veber[2], Katinka Ruthof[2], Una Ryan[1], Peter Irwin[1], Charlotte Oskam[1].

[1] Vector and Waterborne Pathogens Research Group, Murdoch University, Perth, Western Australia, [2] Terrestrial Ecology Research Group, Murdoch University, Perth, Western Australia

Amblyomma triguttatum (Koch 1844), commonly known as the ornate kangaroo tick, is one of the most common ticks encountered in Western Australia. This widespread tick has been implicated in the epidemiology of Q fever (*Coxiella burnetii*) and possibly spotted fever (*Rickettsia gravesii*). Despite its distribution throughout much of mainland Australia relatively little is known about the ecology and diversity of *A. triguttatum*. Habitat preferences of *A. triguttatum* were investigated in a pilot study of 11 sites from Perth's northern suburbs. Spearman rank correlation (non-parametric test) was performed on eight habitat characteristics relating to ground cover, vegetation height and potential host abundance (via scat detection). Analysis revealed that scat mass had the strongest positive relationship with tick abundance. While overall litter depth was also associated with an increase in ticks, there was no clear relationship with specific types of habitat composition. These results suggest, unsurprisingly, that estimates on host abundance provide the most reliable measurement of *A. triguttatum* abundance in a landscape. This presentation will discuss data collected in the current study and explore what this might mean for the distribution of the ornate kangaroo tick, including the influence of fire and host dynamics within localities. Lastly, this pilot study highlights the need for improved taxonomic descriptions of the *A. triguttatum* species-group in order to understand the ecological dynamics of this widespread group of ticks in different regions of Australia.

Characterising Aroma Profile and Investigating Bacterial Communities on Australian and Spanish Black Périgord Truffle (*Tuber Melanosporum*)

Kenny S.O. Choo [1], Maike Bollen [2], Joshua Ravensdale [1], Gary A. Dykes [1], and Ranil Coorey [3]

[1]School of Public Health, Curtin University, [2]Biological and Molecular Mass Spectrometry Facility, Centre for Microscopy, [3]School of Molecular Life Sciences, Curtin University

Black Périgord truffle (*Tuber melanosporum*) is a high-valued fungi traditionally grown in the northern hemisphere. In the past two decades, the Australian truffle industry has grown rapidly and become one of the leading truffle-producing countries in the world. There is little understanding of the volatile profile and ecology of truffle bacterial microbiome. The aim of this study was to investigate the volatile profile and population structure of the bacterial community of Spanish and Australian black Périgord truffle using metagenomics and gas chromatography-mass spectrometry (GC-MS). Black Périgord truffle samples from Spain and Western Australia were assessed during the respective truffle season. The GC-MS results suggested that there is a high resemblance of volatile profile of Spanish and Australian truffle. The major difference between them being that Australian truffles contained 2,4-dithiapentane and dimethyl sulfoxide compounds which have previously only been reported in European white truffle. The V3-V4 region of 16s rRNA DNA was sequenced using the Illumina MiSeq Platform using paired end reads. Alpha-diversity of both Spanish and Australian truffles were low, with Alphaproteobacteria dominating the populations. Operational taxonomic units in the Rhizobiaceae and Bradyrhizobiaceae families where the most abundant sequences in the Spanish samples. The Xanthobacteraceae and Rhizobiaceae families were the most abundant sequences in the Australian samples. Previous studies found the presence of Bradyrhizobiaceae in most truffle species from various regions, but none were detected in Australian truffle. Previous studies have also found Rhizobiales dominate across truffle species and geographic regions including Western Australian truffles. The results in this study adds further evidence to the possibility that truffles may form a mutualistic relationship with Rhizobiales. These bacteria may be able to fix atmospheric nitrogen in the truffle fruiting bodies. The comparison of data from the two regions expands our understanding on how geographical region affect the bacteria communities and potentially the aroma profile of truffles from different regions.

Dr Jennifer Kelley

The University of Western Australia

How ‘Nemo’ got his stripes

Abstract
Coral reef fishes are renowned for their spectacular colouration, but surprisingly, the function of these patterns is often unknown. The clownfishes (which include the charismatic movie character ‘Nemo’) are particularly intriguing because of their characteristic bold white stripes on an orange background, and their unique symbiotic relationship with toxic sea anemones. Interestingly, not all clownfishes are orange, and not all have white stripes, which raises the question of why this iconic colouration evolved. In this presentation, I will explore whether the clownfishes’ colouration evolved to serve a social function (species recognition) or a defensive function (e.g. camouflage or warning colouration). I present evidence that the evolution of stripes is associated with the defensive properties of the host anemone. This raises the intriguing possibility that the clownfishes’ iconic colouration might serve as a warning signal that advertises the toxicity of the host anemone.

Dr Alison Ritchie

The University of Western Australia

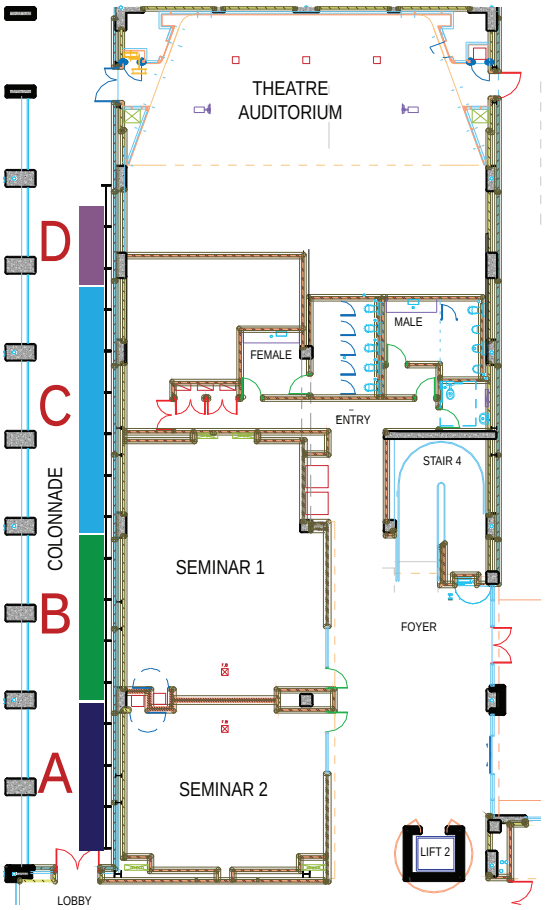
The Use of Innovative Seed Enhancement Technologies to Overcome Barriers to Restoration Success In A Biodiversity Hotspot

Abstract

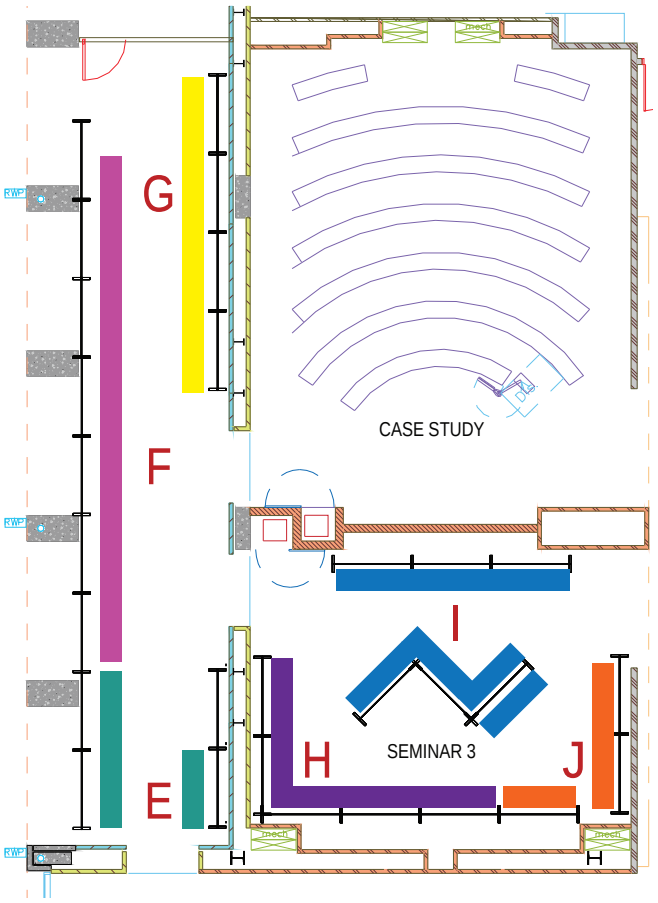
New technologies are needed to overcome the diverse array of barriers to native plant establishment for achieving restoration success. Large parts of southern Australia have already passed beyond “safe limits” of environmental degradation. For low-moderate reinstatement of species diversity, 10-15kg/ha of seed is required, and with an average price of $2,800/kg (current commercial prices) it is therefore imperative that seed resources are used efficiently. Water repellent soils are a major barrier to plant establishment globally, affecting >10M ha of arable sandy soils in southern Australia alone. Soil water repellency leads to decreased water infiltration and moisture retention in the seed zone with resulting poor germination and seedling survival. Weeds are another major barrier to native species in the southwest of WA and are known to change the nutrient status of soils and outcompete native species. Recent developments that include ‘extruded’ seed pellets (seeds embedded in a soil matrix) with soil surfactants (wetting agents) and with activated carbon (herbicide protection) have shown promise in restoring native species in the Southwest of Western Australia. Lambertia inermis seedling emergence was 20% greater from pellets in comparison to bare seeds. For Banksia menziesii, pellets with a surfactant significantly improved the average survival of seedlings after a severe drought by 2.6 days. Trials with activated carbon pellets have shown to protect Jacksonia furcellata from harmful herbicides, targeting the removal of weeds. Using novel methods of distributed temperature sensing (DTS) to aid in the quantification and capture of niche-level processes (e.g. fine-scale hydrological processes), we are now able to quantify the affects to which these pellets formulations influence water infiltration rates. These proof-of-concept investigations into new technologies demonstrate the possibility of creating favourable microsite conditions for seedling establishment and improving the deployment of seeds, provide the necessary steps in the advancement of restoration programs across Australia.

Poster Locations

POSTERS: GROUND FLOOR
LOWER COLONNADES



POSTERS: FIRST FLOOR
UPPER COLONNADES & SEMINAR ROOM 3



SECTION	TOPIC	POSTER AWARD SPONSOR
A	Biochemistry & Molecular Biology	ASBMB
B	Health Sciences	CBSM
C	Senior Research Presentations	John Morris Scientific
D	Plant Science	CBSM
E	Cell & Developmental Biology	ANZSCDB
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the big
picture
factory

Combined Biological Sciences Meeting 2019

Time	Session	Speaker	Title	Chair
8-8:40	Early Career Researcher Development Session (Case Study Room)	Dr Joshua Lewis	Writing a successful fellowship application, lessons learned the hard way!	Dr Raelene Endersby Ms Hannah Greig
8-8:30	**Registration and Breakfast**			
8:45	Welcome	Dr Clayton Fragall	Opening address	
9:00	Plenary (Theatre Auditorium)	Professor Sarah Bekessy	The critical role of 'everyday nature' for the future of cities	Dr Charlotte Oskam
9:40	Dept of Heath WA New Investigator Session (Theatre Auditorium)	Ms Shelley Waters Ms Jennifer Currenti Dr Lucy Furfaro Dr Rachael Zemek	Digging deep to reveal novel CMV variants and associations with patient outcome Deep sequence analysis of HIV adaptation following vertical transmission reveals the impact of immune pressure on the evolution of HIV Group B Streptococcus colonisation of Western Australian pregnant women Sensitisation to immune checkpoint blockade through activation of a STAT1/NK axis in the tumour microenvironment	Dr May Aung-Htut Dr Jessica Buck
	ECU New Investigator Session (Case Study Room)	Mrs Julie Sartori Dr Kritu Panta Dr Kai Chen Dr Pauline Zaenker	The Placenta Project: Characteristics in Assisted & Non-Assisted Pregnancy for a Western Australian Cohort Dengue antibody and Zika Pseurotin A Prevents Estrogen Deficiency-induced Bone Loss by Inhibiting Osteoclastogenesis Identification and Validation of a Diagnostic Autoantibody Signature for Primary Cutaneous Melanoma	Dr Megan Lloyd
10:40	**Morning Tea (sponsored by Murdoch University)**			
11:00	Upper and Lower Colonnade	**Poster Session**		
11:30	Plenary (Theatre Auditorium)	Professor Mark Nicol	Early life microbial exposures and health outcomes in young African children	Assoc Prof Charlene Kahler
12:10	Enabling Technologies Session (Theatre Auditorium)	Dr Torben Kimhofer	The Australian National Phenome Centre	Mr Lawrence Liew
12:30	**Lunch (sponsored by Telethon KIDS Institute)**			

		Australian Society for Microbiology (Theatre Auditorium)	**Cell and Developmental Biology** (Case Study Room)	**Frontiers in Genetics** (Seminar Room 1)	**Biodiversity** (Seminar Room 2)
1:30	Keynote Speakers	Professor Helen Marshall Impact of MenB vaccine on meningococcal disease and carriage of N. meningitidis in S. Aust. Adolescents	Dr Michael Lazarou Sending bad mitochondria to the garbage disposal: Insights into PINK1/Parkin mitophagy	Dr Sam Abraham Seagulls and Superbugs: Role of genomics in understanding the spread of antimicrobial resistance	Dr Nathalie Butt Tough choices: assessing threats and prioritising conservation investment
2:10	Student Speakers	Amy Davis Patrice Maher Emily Kibble Aleesha Davis	Janya Grainok Samuel Montgomery Emma Panting Heng Qiu	Jessica Cale Adriana Foster Laetitia Hughes Jessica Cheng	Alice Michie Kamil Braima Siobhon Egan Kenny Choo
3:10	**Afternoon Tea**				
3:30	Invited Speakers	Professor Jeffrey Keelan Assoc Professor Asha Bowen	Dr Sébastien Malinge Dr Yu Yu	Dr Christian Pflüger Ms Sarah Beecroft	Dr Jennifer Kelley Dr Alison Ritchie

4:30	Plenary (Theatre Auditorium)	Professor Alpha Yap	Tissue forces and epithelial homeostasis	Assoc Prof Nathan Pavlos
5:10	Scientific Awards	Dr Clayton Fragall	Scientific award ceremony and closing address	
5:30 - 7:30	**Sundowner (sponsored by WRAYS)**			

New Investigator Symposia

Specialist Symposia