

Programs & Abstracts

**ASP ANNUAL
CONFERENCE**

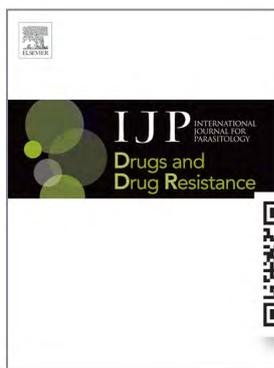
MELBOURNE 2025
30th June - 3rd July



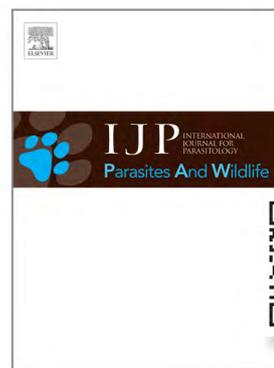
Publish your next article in an Elsevier Parasitology Journal



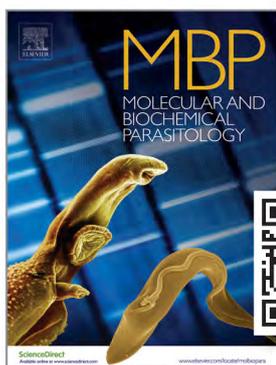
International Journal for Parasitology



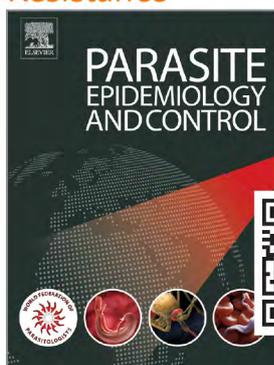
International Journal for Parasitology: Drugs and Drug Resistance



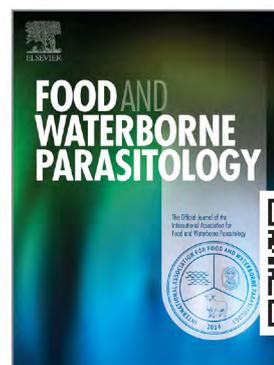
International Journal for Parasitology: Parasites and Wildlife



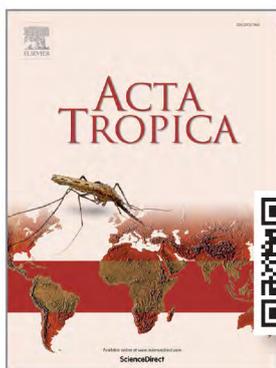
Molecular and Biochemical Parasitology



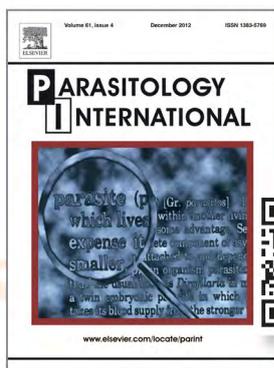
Parasite Epidemiology and Control



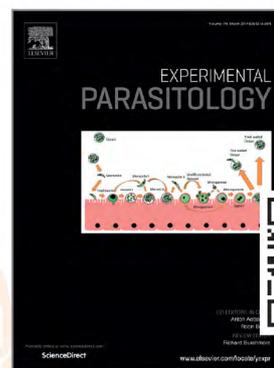
Food and Waterborne Parasitology



Acta Tropica



Parasitology International



Experimental Parasitology



Visit elsevier.com for more information, including how to publish your paper as open access.

2025 Annual Conference of the Australian Society for Parasitology Inc.

30 June – 3 July, 2025 Melbourne Connect, Melbourne, Vic, Australia

Table of Contents

Contents

Table of Contents.....	1
Welcome from the ASP President.....	3
Program Overview.....	4
Presentations	9
P1: Elsevier Plenary Lecture Series International Journal for Parasitology (IJP) Invited Lecturer	9
S1: Pet Parasites Symposium sponsored by Vetoquinol	10
CP1: Drugs & Drug Resistance 15 min talks	10
CP2: Helminth Biology 15 min talks	12
CP3: Pet Parasites sponsored by Vetoquinol.....	14
CP1.1: Drugs & Drug Resistance 5 min talks	15
CP2.1: Helminth Biology 5 min talks	16
CP4: Cell & Molecular Biology 15 min talks.....	17
CP5: Epidemiology 15 min talks	19
CP6: Livestock Parasites 15 min talks sponsored by Elanco	20
CP4.1: Cell & Molecular Biology 5 min talks.....	22
CP5.1: Epidemiology 5 min talks	24
CP6.1: Livestock Parasites 5 min talks sponsored by Elanco	26
CP7: Omics 15 min talks	27
CP8: Arthropod Parasites & Vectors 15 min talks	29
CP9: Fish Parasites 15 min talks	31
CP7.1: Omics 5 min talks	33
CP8.1: Arthropod Parasites & Vectors 5 min talks	34
P2: Plenary Lecture: Don McManus Tropical Health Research Centre and QIMR Berghofer Prize	36
CP10: Drugs & Drug Resistance 15 min talks	36
CP11: Immunology & Vaccination 15 min talks.....	38
CP12: One Health 15 min talks.....	40
CP10.1: Drugs & Drug Resistance 5 min talks	42

CP11.1: Immunology & Vaccination 5 min talks.....	43
CP13: Cell & Molecular Biology 15 min talks.....	44
CP14: Helminth Biology 15 min talks	45
CP15: Pet Parasites 15 min talks	47
CP13.1: Cell & Molecular Biology 5 min talks.....	49
CP14.1: Helminth Biology 5 min talks	51
P3: Elsevier Plenary Lecture Series International Journal for Parasitology: Drugs and Drug Resistance (IJP:DDR) Invited Lecturer.....	52
P4: Elsevier Plenary Lecture Series International Journal for Parasitology: Parasites and Wildlife (IJP:PAW) Invited Lecturer	53
CP16: Drugs & Drug Resistance 15 min talks	53
CP17: Immunology & Vaccination 15 min talks.....	55
CP18: Wildlife Parasites 15 min talks.....	57
CP16.1: Drugs & Drug Resistance 5 min talks	58
CP17.1: Immunology & Vaccination 5 min talks.....	59
CP18.1: Wildlife Parasites 5 min talks.....	60
CP19: Cell & Molecular Biology 15 min talks.....	61
CP20: Diagnostics 15 min talks sponsored Abacus dx.....	63
CP21: Livestock Parasites 15 min talks	65
CP19.1: Cell & Molecular Biology 5 min talks.....	67
CP20.1: Diagnostics 5 min talks sponsored Abacus dx.....	69
CP22: Omics 15 min talks	70
CP23: One Health 15 min talks.....	71
CP24: Fasciola 15 min talks	73
CP22.1: Omics 5 min talks	75
CP23.1: One Health 5 min talks.....	77
CP24.1: Fasciola 5 min talks	78
Delegates	80
Author List.....	87
Conference Organisation	133
Amazing Conference Volunteers	133

2025 Annual Conference of the Australian Society for Parasitology Inc.

30 June – 3 July, 2025 Melbourne Connect, Melbourne, Vic, Australia

Welcome from the ASP President



Dear Colleague,

On behalf of the Australian Society for Parasitology (ASP) Council and the 2025 Conference Organising Committee, we extend a warm welcome to the 2025 ASP Annual Conference. The Conference, at Melbourne Connect, Melbourne, Victoria, will begin, with an evening Welcome Reception, on Monday 30 June 2025, and culminate, with our Conference Dinner, at State Library Victoria on Thursday 3 July 2025.

Discuss the latest research and state-of-the-art technologies in parasitology. The scientific program will cover all parasitology themes from Veterinary Parasitology to Human Parasitology, with Malaria, Strongyloides, Bioinformatics, Microscopy, Livestock, Wildlife Parasitology, Fish Parasitology, Companion Animals, One Health and more. The program covers all aspects of parasitology research and that includes basic research in all areas of life science.

This year's program includes Veterinary Parasitology Day 2025ASP, an outreach program for Veterinarians who want to find out the latest research in pet and livestock parasitology from researchers on Tuesday 1 July, 2025, followed by a discussion with the researchers. As part of the Conference we will run a Women in Parasitology mentorship program with coach, mentor and educator Melissa Rosenthal who will help develop communication skills in negotiation, and a careers panel discussion with Leann Tilley, UoM; Karena Waller, UoM; Shookofeh Shamsi, SCU; Tania De-koning Ward, Deakin Uni; Simona John von Freyend, PTNG Scientific and Lisa Jones, ASP.

We would like to acknowledge the generous support of our 2025 ASP Conference sponsors, thanks to **Elsevier and the International Journal for Parasitology (IJP), IJP DDR and IJP PAW, Elanco, Vetoquinol, Abacus dx, Thermo Fisher Scientific, New England Biolabs, Southern Cross Diagnostics, Promega, Burnet Institute, Global Health TPA at Monash University, VectorBuilder, The Peter Doherty Institute for Infection and Immunity and the Don McManus Tropical Health Research Centre and QIMR Berghofer.**

The International Journal for Parasitology (IJP) and The University of Melbourne are proud sponsors of the Women in Parasitology Mentorship program.

We also would like to thank you, the ASP Membership, for supporting our Society and this Conference so enthusiastically.

A/Prof Danny Wilson
President, ASP

2025 Annual Conference of the Australian Society for Parasitology Inc.

30 June – 3 July, 2025 Melbourne Connect, Melbourne, Vic, Australia

Program Overview

Date: Monday, 30/June/2025

11:30am - 4:30pm	2025 WiP: ASP Women in Parasitology program Session Chair: Ghizal Siddiqui , Monash University Session Chair: Hayley Bullen , Burnet Institute As part of the 2025 ASP Annual Conference the ASP will run a mentorship day program for women in parasitology. (limit of 30 can attend, application by EOI.)
2:00pm - 5:00pm Superfloor	Registration Location: Superfloor
6:00pm - 6:15pm Superfloor	WelcometoCountry: Welcome to Country Location: Superfloor
6:00pm - 8:00pm Superfloor	Welcome: Welcome Reception Location: Superfloor The Welcome Reception will take place at Melbourne Connect on Monday June 30, from 6pm

Date: Tuesday, 01/July/2025

7:30am - 9:00am Superfloor	ECRBreakfast: ECR and Student Breakfast Event Location: Superfloor Session Chair: Elizabeth Aitken , Peter Doherty Institute, University of Melbourne On the first full day of the 2025 ASP Annual Conference we start with a networking breakfast event for our research students and early career researchers. Our amazing mentors are: Rhea Longley, WEHI, Ala Tabor, UQ, Claire Sayers, UNSW, Adele Lehane, ANU and Herbert Opi, KEMRI-Wellcome Trust (Kenya)/ University of Edinburgh.
9:00am - 9:15am Conference plenary room	Intro: Welcome to the Conference Location: Conference plenary room Session Chair: Hayley Bullen , Burnet Institute Session Chair: Aaron Jex , WEHI
9:15am - 10:30am Conference plenary room	P1: Elsevier Plenary Lecture Series International Journal for Parasitology (IJP) Invited Lecturer Location: Conference plenary room Session Chair: Brian M Cooke , James Cook University
10:30am - 11:00am	Morning Tea Break Tuesday
11:00am - 11:30am Conference room 3	S1: Pet Parasites Symposium sponsored by Vetoquinol Location: Conference room 3 Session Chair: Don Strazzeri , Vetoquinol Australia Pty Ltd
11:00am - 12:15pm Conference room 1	CP1: Drugs & Drug Resistance 15 min talks Location: Conference room 1 Session Chair: Hayley Bullen , Burnet Institute Session Chair: Brad Sleeb s, Walter and Eliza Hall Institute
11:00am - 12:15pm Conference room 2	CP2: Helminth Biology 15 min talks Location: Conference room 2 Session Chair: Nichola Eliza Davies Calvani , The University of Sydney Session Chair: Charles Gauci , University of Melbourne
11:30am - 12:30pm Conference room 3	CP3: Pet Parasites sponsored by Vetoquinol Location: Conference room 3 Session Chair: Don Strazzeri , Vetoquinol Australia Pty Ltd
12:15pm - 12:30pm Conference room 1	CP1.1: Drugs & Drug Resistance 5 min talks Location: Conference room 1 Session Chair: Hayley Bullen , Burnet Institute Session Chair: Brad Sleeb s, Walter and Eliza Hall Institute
12:15pm - 12:30pm	

Conference room 2	CP2.1: Helminth Biology 5 min talks Location: Conference room 2 Session Chair: Nichola Eliza Davies Calvani , The University of Sydney Session Chair: Charles Gauci , University of Melbourne
12:30pm - 1:30pm	Lunch Tuesday
1:00pm - 1:30pm	Bite1: Lunch Bites Tuesday Location: The Launch Pad Session Chair: Sarah Preston , Federation University Australia with Graham Mitchell , Foresight
1:30pm - 2:30pm	CP4: Cell & Molecular Biology 15 min talks Location: Conference room 1 Session Chair: Jill Chmielewski , WEHI Session Chair: Giel G van Dooren , Australian National University
1:30pm - 2:30pm	CP5: Epidemiology 15 min talks Location: Conference room 2 Session Chair: Shannon M Hedtke , La Trobe University Session Chair: Kirsty M Mccann , Deakin University
1:30pm - 2:45pm	CP6: Livestock Parasites 15 min talks sponsored by Elanco Location: Conference room 3 sponsored by Elanco and chaired by Liisa Ahlstrom & Monica Commons Elanco
2:30pm - 3:00pm	CP4.1: Cell & Molecular Biology 5 min talks Location: Conference room 1 Session Chair: Jill Chmielewski , WEHI Session Chair: Giel G van Dooren , Australian National University
2:30pm - 3:00pm	CP5.1: Epidemiology 5 min talks Location: Conference room 2 Session Chair: Shannon M Hedtke , La Trobe University Session Chair: Kirsty M Mccann , Deakin University
2:45pm - 3:00pm	CP6.1: Livestock Parasites 5 min talks sponsored by Elanco Location: Conference room 3 sponsored by Elanco and chaired by Liisa Ahlstrom & Monica Commons Elanco
3:00pm - 3:30pm	Afternoon Tea Break Tuesday
3:30pm - 4:45pm	CP7: Omics 15 min talks Location: Conference room 1 Session Chair: Ghizal Siddiqui , Monash University Session Chair: Balu Balan , Walter and Eliza Hall Institute
3:30pm - 4:45pm	CP8: Arthropod Parasites & Vectors 15 min talks Location: Conference room 2 Session Chair: Charlotte Oskam , Murdoch University Session Chair: Vern Bowles , The University of Melbourne
3:30pm - 5:00pm	CP9: Fish Parasites 15 min talks Location: Conference room 3 Session Chair: Cecilia Power , RMIT University Session Chair: Storm Martin , Murdoch University
4:45pm - 5:00pm	CP7.1: Omics 5 min talks Location: Conference room 1 Session Chair: Ghizal Siddiqui , Monash University Session Chair: Balu Balan , Walter and Eliza Hall Institute
4:45pm - 5:00pm	CP8.1: Arthropod Parasites & Vectors 5 min talks Location: Conference room 2 Session Chair: Charlotte Oskam , Murdoch University Session Chair: Vern Bowles , The University of Melbourne
Date: Wednesday, 02/July/2025	
9:00am - 9:45am	BOM: The 2025 Bridget Ogilvie Medal oration Location: Conference plenary room Session Chair: Danny Wilson , The University of Adelaide
9:45am - 10:30am	P2: Plenary Lecture: Don McManus Tropical Health Research Centre and QIMR Berghofer Prize Location: Conference plenary room Session Chair: Darren Gray , QIMR Berghofer
10:30am - 11:00am	Morning Tea Break Wednesday sponsored by Thermo Fisher Scientific

11:00am - 12:15pm	CP10: Drugs & Drug Resistance 15 min talks
Conference room 1	Location: Conference room 1 Session Chair: Jacinta Macdonald , Griffith University Session Chair: Darren Creek , Monash University
11:00am - 12:15pm	CP11: Immunology & Vaccination 15 min talks
Conference room 2	Location: Conference room 2 Session Chair: Li Jin Chan , The Walter and Eliza Hall Institute Session Chair: Michael Duffy , University of Melbourne
11:00am - 12:30pm	CP12: One Health 15 min talks
Conference room 3	Location: Conference room 3 Session Chair: Amanda Ash , Murdoch University Session Chair: Lucas Huggins , University Of Melbourne
12:15pm - 12:30pm	CP10.1: Drugs & Drug Resistance 5 min talks
Conference room 1	Location: Conference room 1 Session Chair: Jacinta Macdonald , Griffith University Session Chair: Darren Creek , Monash University
12:15pm - 12:30pm	CP11.1: Immunology & Vaccination 5 min talks
Conference room 2	Location: Conference room 2 Session Chair: Li Jin Chan , The Walter and Eliza Hall Institute Session Chair: Michael Duffy , University of Melbourne
12:30pm - 1:30pm	Lunch Wednesday
1:00pm - 1:30pm	Bite2: Lunch Bites Wednesday
The Launch Pad	Location: The Launch Pad Session Chair: Hayley Bullen , Burnet Institute Session Chair: Alex Loukas , James Cook University
1:30pm - 2:30pm	CP13: Cell & Molecular Biology 15 min talks
Conference room 1	Location: Conference room 1 Session Chair: Fleur Sernee , The University of Melbourne Session Chair: Paul Gilson , Burnet Institute
1:30pm - 2:45pm	CP14: Helminth Biology 15 min talks
Conference room 2	Location: Conference room 2 Session Chair: Sarah Preston , Federation University Australia Session Chair: Anson Koehler , University of Melbourne
1:30pm - 3:00pm	CP15: Pet Parasites 15 min talks
Conference room 3	Location: Conference room 3 Session Chair: Ryan O'Handley , Adelaide University Session Chair: Bonny Cumming , AMRRIC
2:30pm - 3:00pm	CP13.1: Cell & Molecular Biology 5 min talks
Conference room 1	Location: Conference room 1 Session Chair: Fleur Sernee , The University of Melbourne Session Chair: Paul Gilson , Burnet Institute
2:45pm - 3:00pm	CP14.1: Helminth Biology 5 min talks
Conference room 2	Location: Conference room 2 Session Chair: Sarah Preston , Federation University Australia Session Chair: Anson Koehler , University of Melbourne
3:00pm - 3:30pm	Afternoon Tea Break Wednesday sponsored by Thermo Fisher Scientific
3:30pm - 4:00pm	President: ASP President Address
Conference plenary room	Location: Conference plenary room
4:00pm - 6:00pm	AGM: 2025 ASP AGM
Conference plenary room	Location: Conference plenary room Session Chair: Danny Wilson , The University of Adelaide
6:30pm - 9:00pm	Student social event: ECR Student Social evening
	ECR Student evening social event at The Clyde Hotel. Attendees must pay for their own drinks, there will be a limited amount of bar food provided by the ASP, games, quiz night, and if you need more food you can purchase extra there.
Date: Thursday, 03/July/2025	
9:00am - 9:45am	P3: Elsevier Plenary Lecture Series International Journal for Parasitology: Drugs and Drug Resistance (IJP:DDR) Invited Lecturer
Conference plenary room	Location: Conference plenary room Session Chair: Kevin Saliba , Australian National University
9:45am - 10:30am	

Conference plenary room	P4: Elsevier Plenary Lecture Series International Journal for Parasitology: Parasites and Wildlife (IJP:PAW) Invited Lecturer Location: Conference plenary room Session Chair: Ian Beveridge , University of Melbourne
10:30am - 11:00am	Morning Tea Break Thursday
11:00am - 12:15pm	CP16: Drugs & Drug Resistance 15 min talks Location: Conference room 1 Session Chair: Kathy Andrews , Griffith University Session Chair: Adele Lehane , Australian National University
Conference room 1	
11:00am - 12:15pm	CP17: Immunology & Vaccination 15 min talks Location: Conference room 2 Session Chair: Denise Doolan , University of Queensland Session Chair: Lee M. Yeoh , Burnet Institute
Conference room 2	
11:00am - 12:15pm	CP18: Wildlife Parasites 15 min talks Location: Conference room 3 Session Chair: Kate Hutson , Cawthron Institute Session Chair: Abdul Jabbar , University of Melbourne
Conference room 3	
12:15pm - 12:30pm	CP16.1: Drugs & Drug Resistance 5 min talks Location: Conference room 1 Session Chair: Kathy Andrews , Griffith University Session Chair: Adele Lehane , Australian National University
Conference room 1	
12:15pm - 12:30pm	CP17.1: Immunology & Vaccination 5 min talks Location: Conference room 2 Session Chair: Denise Doolan , University of Queensland Session Chair: Lee M. Yeoh , Burnet Institute
Conference room 2	
12:15pm - 12:30pm	CP18.1: Wildlife Parasites 5 min talks Location: Conference room 3 Session Chair: Kate Hutson , Cawthron Institute Session Chair: Abdul Jabbar , University of Melbourne
Conference room 3	
12:30pm - 1:30pm	Lunch Thursday
1:00pm - 1:30pm	Bite3: Lunch Bites Thursday Location: The Launch Pad Session Chair: Clare A. Anstead , University of Melbourne Session Chair: Vern Bowles , The University of Melbourne Session Chair: Alex Loukas , James Cook University
The Launch Pad	
1:30pm - 2:30pm	CP19: Cell & Molecular Biology 15 min talks Location: Conference room 1 Session Chair: Claire Sayers , University of New South Wales Session Chair: Colin Sutherland , LSHTM London
Conference room 1	
1:30pm - 2:45pm	CP20: Diagnostics 15 min talks sponsored Abacus dx Location: Conference room 2 Session Chair: Kelly Ly , Abacusdx
Conference room 2	
1:30pm - 3:00pm	CP21: Livestock Parasites 15 min talks Location: Conference room 3 Session Chair: Emily Francis , The University of Sydney Session Chair: Johann Schroder , Gemini R&D Services
Conference room 3	
2:30pm - 3:00pm	CP19.1: Cell & Molecular Biology 5 min talks Location: Conference room 1 Session Chair: Claire Sayers , University of New South Wales Session Chair: Colin Sutherland , LSHTM London
Conference room 1	
2:45pm - 3:00pm	CP20.1: Diagnostics 5 min talks sponsored Abacus dx Location: Conference room 2 Session Chair: Kelly Ly , Abacusdx
Conference room 2	
3:00pm - 3:30pm	Afternoon Tea Break Thursday
3:30pm - 4:30pm	CP22: Omics 15 min talks Location: Conference room 1 Session Chair: Shilpa Kapoor , The University of Melbourne Session Chair: Aaron Jex , WEHI
Conference room 1	
3:30pm - 4:45pm	CP23: One Health 15 min talks Location: Conference room 2 Session Chair: Breanna Knight , Murdoch University Session Chair: Harsha Sheorey , St Vincent's Hospital, Melbourne
Conference room 2	
3:30pm - 4:45pm	

Conference room 3	CP24: Fasciola 15 min talks Location: Conference room 3 Session Chair: Tanapan Sukee , The University of Melbourne Session Chair: Neil Young , The University Of Melbourne
4:30pm - 5:00pm	CP22.1: Omics 5 min talks Location: Conference room 1 Session Chair: Shilpa Kapoor , The University of Melbourne Session Chair: Aaron Jex , WEHI
Conference room 1	
4:45pm - 5:00pm	CP23.1: One Health 5 min talks Location: Conference room 2 Session Chair: Breanna Knight , Murdoch University Session Chair: Harsha Sheorey , St Vincent's Hospital, Melbourne
Conference room 2	
4:45pm - 5:00pm	CP24.1: Fasciola 5 min talks Location: Conference room 3 Session Chair: Tanapan Sukee , The University of Melbourne Session Chair: Neil Young , The University of Melbourne
Conference room 3	
7:00pm - 11:00pm	Dinner: Dinner and award of student prizes

2025 Annual Conference of the Australian Society for Parasitology Inc.

30 June – 3 July, 2025 Melbourne Connect, Melbourne, Vic, Australia

Presentations

P1: Elsevier Plenary Lecture Series International Journal for Parasitology (IJP) Invited Lecturer

Time: Tuesday, 01/July/2025: 9:15am - 10:30am · *Location:* Conference plenary room

Session Chair: Brian M Cooke, James Cook University

ID: 294 / P1: 1

Invited speaker abstract

Drug resistant malaria: the shared blame on host and parasite.

Nancy Quashie

Noguchi Memorial Institute for Medical Research, University of Ghana, Ghana

The persistent challenge of emergence of drug-resistant malaria necessitates a more comprehensive and integrative understanding of the biological and biochemical interactions between the human host and the malaria parasite. While significant progress has been made in elucidating parasite-driven mechanisms of resistance, there remains a critical need to expand the focus to host-related factors. This perspective is essential to mitigate the global burden of malaria, characterized by high morbidity and mortality rates. Historically, resistance to antimalarial drugs has originated from Southeast Asia, as observed with chloroquine and recently, artemisinin. Genetic mutations in the parasite often arise spontaneously during sexual reproduction in the mosquito vector through gene recombination. The resistant progenies spread by selection due to drug pressure and by human population movement. Other host-related factors include variability in drug metabolism and transport, non-compliance, and substandard or counterfeit drugs. For over two decades, the Malaria Research group at the Noguchi Memorial Institute for Medical Research (NMIMR), University of Ghana, in collaboration with the National Malaria Elimination Programme, has implemented robust surveillance systems to monitor antimalarial drug efficacy and resistance. These efforts include clinical, in vitro, molecular, and pharmacokinetic methodologies. This holistic strategy helped the early detection of evolution and spread of drug-resistant malaria. Data generated from these studies were instrumental in informing the 2005 national treatment policy change from the use of chloroquine to artemisinin-based combination therapies (ACTs). Recently, a growing concern in malaria diagnostics is the reliability of rapid diagnostic tests (RDTs), targeting the *Plasmodium falciparum* histidine-rich protein 2 (PFHRP2). Deletions in the genes in parasite strains result in false-negative tests, potentially enabling the undetected spread of resistant parasites with the deletion. Continuous surveillance has been maintained to monitor infection rates, treatment outcomes and diagnostics incorporating both clinical assessments and genetic analyses of host and parasite. To effectively combat malaria, it is imperative to adopt a multidisciplinary approach that integrates hosts and parasite biology, pharmacology, diagnostics, and public health strategies. Thus, this is what the Malaria group at NMIMR under the parasitological umbrella has done over the years with NMEP and has helped in the control of the disease in Ghana.

S1: Pet Parasites Symposium sponsored by Vetoquinol

Time: Tuesday, 01/July/2025: 11:00am - 11:30am · Location: Conference room 3

Session Chair: Don Strazzeri, Vetoquinol Australia Pty Ltd

ID: 292 / S1: 1

Invited speaker abstract

Prevalence of *Dirofilaria immitis*, *Dipylidium caninum* and *Taenia* spp. in populations of cats from shelters and research colonies, in two endemic regions of Eastern Australia.

Florian Roeber¹, Riannon Apicella¹, Michael Chambers¹, Don Strazzeri², Norbert Mencke³, Katrin Blazejak³

¹Invetus Pty Ltd., Wongaburra Research Centre, Casino NSW 2470, Australia; ²Vetoquinol Australia PTY LTD, 485 Kingsford Smith Drive, Hamilton QLD 4007 Australia; ³Vetoquinol SA, 37 rue de la Victoire, 75009 Paris, France

There is a paucity of available prevalence data for key parasite species and genera infecting cats in Australia. The present study aimed to determine the prevalence of key parasites infecting cats in Australia, namely the feline heartworm (*Dirofilaria immitis*), *Dipylidium caninum* and *Taenia* spp. Prevalence of these parasites was assessed in cat populations in five separate locations of eastern Australia and within two geographic regions – humid subtropical and oceanic. A total of 141 cats were enrolled in this study. Out of these cats, 91 were tested for *D. immitis* by feline heartworm Antigen/Antibody testing, 93 samples were tested for *Di. caninum* and for *Taenia* spp. by faecal floatation and a subset of 48 cat samples were tested for *Di. caninum* and *Taenia* spp. by PCR performed on peri-anal swabs. Test results were negative for the two species with a single positive result observed for *Taenia* spp. Point estimates of prevalence were therefore 0% for feline heartworm and *Di. caninum* and 1.1% for *Taenia* spp. Associated 95% confidence intervals around the point estimate were 0 - 4.1% for feline heartworm, 0 – 3.9% for *Di. caninum* and 0 – 5.8 % for *Taenia* spp. It should be noted that estimates were calculated using nominal values for test sensitivity and specificity, however the overall prevalence of all three parasite species in these geographic regions is likely to be minimal based on data from this study.

CP1: Drugs & Drug Resistance 15 min talks

Time: Tuesday, 01/July/2025: 11:00am - 12:15pm · Location: Conference room 1

Session Chair: Hayley Bullen, Burnet Institute

Session Chair: Brad Sleebs, Walter and Eliza Hall Institute

ID: 171 / CP1: 1

Contributed abstract

Conference Topics: Drugs, Malaria

Keywords: Antimalarial Drug Discovery

Optimization and Characterization of N-Acetamide Indoles as Antimalarials Targeting PfATP4

Kyle Awalt^{1,2}, Madeline Dans^{1,2}, Brad Sleebs^{1,2}

¹The Walter and Eliza Hall Institute of Medical Research, Parkville 3052, Australia; ²Department of Medical Biology, The University of Melbourne, Parkville 3010, Australia

Malaria is a disease caused by infection from species of the parasite *Plasmodium*. Its effects were responsible for over 600,020 deaths in 2022 with children and pregnant woman most impacted. The effectiveness of clinically used antimalarials, including the frontline artemisinin combination therapies, is under threat from emerging drug resistance, putting an emphasis on the discovery of new antimalarial chemotypes targeting novel mechanisms of action and multiple stages of the parasitic lifecycle.

Our pursuit of new and effective antimalarials has included a screen of the Janssen Jumpstarter library against asexual stage parasites and has led to the discovery of an N-acetamide indole hit class. Structure-activity relationships were investigated and the series optimized to produce the frontrunner compound **WJM664**, featuring potent activity against asexual stage parasites, high metabolic stability and moderate plasma exposure in mice. Application of forward genetics and subsequent validation revealed the series to target the cation ATPase PfATP4, a protein essential for parasite development due to its role in maintaining cytosolic Na⁺ homeostasis. In addition to asexual stage parasites these compounds were shown to inhibit male and female gamete development and block transmission from infected blood to mosquito.

ID: 141 / CP1: 2

Contributed abstract

Conference Topics: Cell Biology, Drugs, Genomics, Malaria, Microscopy, Molecular Biology

Keywords: Malaria, drug development, high throughput screen, mechanism of action studies

Uncovering the mechanism of action of novel antimalarials from the Janssen Jumpstarter library

Madeline Dans^{1,2}, William Nguyen^{1,2}, Coralie Boulet³, Jon Kyle Awalt^{1,2}, Wenyin Su^{1,2}, Anna Ngo^{1,2}, Jocelyn Penington^{1,2}, Cindy Evelyn^{1,2}, Niall Geoghegan^{1,2}, Kate Jarman^{1,2}, Alexander Maier⁴, Giel van Dooren⁴, Tony Papenfuss^{1,2}, Adele Lehane⁴, David Fidock^{5,6}, Sergio Wittlin^{7,8}, Stephen Brand⁹, Kym Lowes^{1,2}, Kelly Rogers^{1,2}, Paul Gilson³, Paul Jackson¹⁰, Alan Cowman^{1,2}, Brad Sleebs^{1,2}

¹The Walter and Eliza Hall Institute of Medical Research, Parkville 3052, Australia.; ²Department of Medical Biology, The University of Melbourne, Parkville 3010, Australia.; ³Burnet Institute, Melbourne, VIC, 3004, Australia.; ⁴Research School of Biology, The Australian National University, Canberra, 2600, Australia.; ⁵Department of Microbiology & Immunology, Columbia University, Irving Medical Center, New York, New York 10032, United States.; ⁶Center for Malaria Therapeutics and Antimicrobial Resistance, Division of Infectious Diseases, Department of Medicine, Columbia University, Irving Medical Center, New York, New York 10032, United States.; ⁷Swiss Tropical and Public Health Institute, Kreuzstrasse 2, 4123 Allschwil, Switzerland.; ⁸University of Basel, 4003 Basel, Switzerland.; ⁹Medicines for Malaria Venture, ICC, Route de Pré-Bois 20, 1215 Geneva, Switzerland.; ¹⁰Global Public Health, Janssen R&D LLC, La Jolla, California 92121, United States.

New classes of molecules to target *Plasmodium falciparum* are required to combat clinical resistance to current antimalarials. To discover new chemotypes for development, we screened the Janssen Jumpstarter library of 80,000 drug-like compounds against asexual stage parasites using lactate dehydrogenase assays. Hit compounds were confirmed in a dose-response assay and counterscreen and cytotoxic assays were carried out to further triage compounds. This culminated in a total of 85 hits from 8 novel compound classes.

We have conducted in-depth mechanism of action studies to deconvolve the parasite targets of these novel classes. We employed forward genetics to select resistant parasites against our hit compounds. By pairing this approach with whole genome sequencing, we identified genetic variants in our resistant parasites, revealing targets that include *P. falciparum* ATPase 4 (*PfATP4*), a StAR-related lipid transfer 1 protein (*PfSTART1*) and cytochrome bc1 complex (*PfCytB*). To validate these targets, we have conducted phenotypic assays to assess Na⁺ efflux (*PfATP4*), live cell imaging (*PfSTART1*) and mitochondrion consumption (*PfCytB*). Additionally, we have carried out profiling of our hits against clinically resistant parasite lines, demonstrating they remain susceptible to our chemotypes. These compounds show promise for antimalarial development and serve as valuable tools for studying essential parasite processes.

ID: 276 / CP1: 3

Contributed abstract

Conference Topics: Drugs, Malaria

Keywords: Malaria, artemisinin, artemisinin resistance, Sulforaphane

PfK13-associated artemisinin resistance slows drug activation and enhances antioxidant defence, which can be overcome with Sulforaphane.

Ghizal Siddiqui¹, Carlo Giannangelo¹, Natalie A Counihan², Yunyang Zhou¹, Annie Ugobobuaku-Roys¹, Amanda De Paoli¹, Bethany M Anderson³, Laura Edgington-Mitchell³, Tania F de Koning-Ward², Darren J Creek¹

¹Drug Delivery, Disposition and Dynamics, Monash Institute of Pharmaceutical Sciences, Monash University, Parkville Campus, Parkville, Victoria, Australia.; ²School of Medicine and Institute for Mental and Physical Health and Clinical Translation, Deakin University, Waurn Ponds, Victoria, Australia.; ³Department of Biochemistry and Pharmacology, The University of Melbourne, VIC 3000, Australia.

Artemisinin resistance is increasingly prevalent in Africa, raising concerns and highlighting the need to better understand the cellular mechanisms behind this resistance. In *Plasmodium falciparum*, artemisinin resistance is primarily attributed to mutations in the PfK13 gene. In this study, we performed proteomics analysis on a range of artemisinin-resistant (both laboratory-generated and field isolates) and sensitive *P. falciparum* parasites at 3-6h ring- and 22-24h trophozoite-stage, revealing dysregulation of only PfK13 protein abundance. Reduced PfK13 levels were linked to impaired hemoglobin digestion, decreased free heme levels, and consequently, decreased artemisinin activation. Resistant parasites exhibited elevated thiol levels, indicating a more reduced state. Targeting the parasite redox capacity with sulforaphane potentiated artemisinin activity *in vitro* and *in vivo*, offering a potential strategy to overcome resistance. Our findings provide critical insights into the molecular mechanisms of artemisinin resistance and suggest novel therapeutic interventions to restore drug sensitivity.

ID: 156 / CP1: 4

Contributed abstract

Conference Topics: Apicomplexa Biology, Drugs, Malaria

Keywords: Plasmodium, Transmission, Electron transport chain, Antimalarial drugs, Sugar baits

Feeding antimalarials to mosquitoes infected with *Plasmodium* blocks parasite transmission.

Sarah N. Farrell¹, Anton Cozijnsen¹, Vanessa Mollard¹, Papireddy Kancharla², Rozalia A. Dodean³, Jane X. Kelly^{2,3}, Geoffrey I. McFadden¹, Christopher D. Goodman¹

¹School of Biosciences, The University of Melbourne, Parkville, VIC, 3010, Australia; ²Department of Chemistry, Portland State University, Portland, Oregon 97201, United States.; ³Department of Veterans Affairs Medical Center, Portland, Oregon 97239, United States.

A decade-long decline in malaria cases has plateaued, primarily due to parasite drug resistance and mosquito resistance to insecticides. Here, we explore an innovative control strategy that targets *Plasmodium* with antimalarials during the mosquito stages. This strategy could reduce the risk of resistance emerging because a small population of parasites within the mosquito is subject to selection. We screened a range of parasitocidal compounds by feeding them to mosquitoes already infected with mouse malaria (*P. berghei*). T111, a next generation compound targeting the parasite electron transport chain, reduced sporozoite numbers in *P. berghei* at equivalent concentrations to the gold standard electron transport chain inhibitor, atovaquone. T111 also prevented sporozoite production in mosquitoes infected with human malaria, *P. falciparum*, even after very short exposure times. Encouragingly, T111 remained efficacious after being freeze-dried onto a substrate and later reconstituted with water, suggesting this compound would be effective in easy-to-distribute-and-deploy transmission control devices. Our findings suggest that targeting mosquito-stage parasites via sugar baits should limit malaria transmission. Importantly, this control strategy vastly increases the range of potentially useful parasitocidal compounds to include those failing to meet the exacting standards required for human antimalarial drugs, potentially improving malaria control for minimal cost.

ID: 116 / CP1: 5

Contributed abstract

Conference Topics: Apicomplexa Biology, Drugs, Malaria, Molecular Biology

Keywords: antimalarial, protein, malaria, plasmodium

Characterisation of protein disulphide isomerases and their inhibitors to prevent erythrocyte invasion and egress by malaria parasites

Senna Steen¹, Molly Schneider¹, Claudia Barnes¹, Trent Ashton², Will Nguyen², Maria Gancheva³, Emma Yuxin Mao³, Amelia Ford⁴, Ghizal Siddiqui⁵, Darren Creek⁵, Andrew Blagborough⁴, Danny Wilson³, Brad Sleebbs², Paul Gilson¹, Fiona Angrisano^{*1}, Hayley Bullen^{*1}

¹Burnet Institute, Melbourne, Australia; ²WEHI, Melbourne, Australia; ³University of Adelaide, Adelaide, Australia; ⁴University of Cambridge, UK; ⁵MIPS, Melbourne, Australia

Resistance to antimalarials is a looming barrier to malaria control, necessitating therapeutics with novel targets. Plasmodium parasites are obligate intracellular pathogens, their survival and pathogenicity being contingent on egress of daughter parasites from host erythrocytes and subsequent invasion of new erythrocytes. Marketable inhibitors of egress/invasion are highly sought after but remain elusive.

Protein disulphide isomerases (PDIs) are established eukaryotic protein folding chaperones and disulphide bonding mediators. *P. falciparum* possesses four highly conserved PDIs (8, Trans, 11, and 14) which were suspected to help shape essential egress/invasion proteins.

We have shown that mammalian PDI inhibitor analogues impede *P. falciparum* egress/invasion, causing misfolding of essential proteins involved in these parasite-specific processes. We have generated a compound that prevents *P. falciparum* invasion, egress, and growth in the erythrocyte, likely by inhibiting PDI activity. This inhibitor is also active against another clinically relevant Plasmodium species, suggesting that the compound could have pan-species activity.

We also have data that implicates PDI-Trans as a mediator of merozoite surface protein 1 folding, which enables egress. With an established relationship between PDIs and the integral processes of egress/invasion as well as transmission, repurposing and refining existing PDI inhibitors may offer a novel means of eliminating malaria parasites.

CP2: Helminth Biology 15 min talks

Time: Tuesday, 01/July/2025: 11:00am - 12:15pm · *Location:* Conference room 2

Session Chair: Nichola Eliza Davies Calvani, The University of Sydney

Session Chair: Charles Gauci, University of Melbourne

ID: 268 / CP2: 1

Contributed abstract

Conference Topics: Genomics, Helminthology, Strongyloides, Veterinary Parasitology, Other

Keywords: Haemonchus contortus, Molecular parasitology, Genetic diversity, Host-parasite interactions, Systems biology

Systems biology of *Haemonchus contortus*: from molecular insights to applied outcomes

Yuanting Zheng¹, Neil Young¹, Tao Wang¹, Bill Chang¹, Jiangning Song², Robin Gasser¹

¹Faculty of Science, Melbourne Veterinary School, The University of Melbourne, Parkville, Victoria, Australia; ²Department of Data Science and AI, Faculty of IT, Monash University, Victoria, Australia

Parasitic nematodes, particularly *Haemonchus contortus*, continue to present major challenges to animal health and agricultural sustainability worldwide. As a model organism for strongyloid nematodes, *H. contortus* provides a powerful system for investigating parasite molecular biology, host-parasite interactions and mechanisms underlying drug resistance. This review-based presentation summarises progress made during my PhD project and highlights how integrative 'omic approaches and informatics are advancing our understanding of *H. contortus* at the molecular level. A major focus of my research was the reconstruction of molecular pathways, such as the ubiquitination pathway. In parallel, genome-wide analyses of the degradome and secretome revealed expansive repertoires of proteases and secreted proteins, many with predicted roles in blood-feeding and immune modulation. To support these efforts, I developed an informatic workflow that integrates protein structure prediction with machine learning-enhanced annotation tools, substantially improving the functional annotation of parasite proteins. My work also produced a chromosome-level reference genome for the Australian Haecon-5 strain of *H. contortus*, enabling comparative genomic analyses and supporting the prediction of essential genes. Collectively, these efforts illustrate the potential of systems biology to accelerate molecular discovery in parasitic nematodes and to inform the development of innovative strategies to address the growing problem of anthelmintic resistance.

ID: 269 / CP2: 2

Contributed abstract

Conference Topics: Bioinformatics, Genomics, Helminthology

Keywords: Informatics, Protein annotation, Haemonchus contortus, Excretory/secretory proteins

A bioinformatic workflow for the enhanced annotation of excretory/secretory proteins

Yuanting Zheng¹, Neil Young¹, Jiangning Song², Bill Chang¹, Robin Gasser¹

¹Faculty of Science, Melbourne Veterinary School, The University of Melbourne, Parkville, Victoria, Australia; ²Department of Data Science and AI, Faculty of IT, Monash University, Victoria, Australia

The rapid advancement of genomic and computational technologies has underscored the need for robust bioinformatic workflows capable of accurately annotating genes and their products through comparative and functional analyses. However, annotating protein-coding genes in evolutionarily divergent organisms – such as parasitic nematodes – remains challenging, despite the availability of reference datasets from model species like *Caenorhabditis elegans* and *Drosophila melanogaster*. To address this gap, we developed a dedicated and evolving informatic pipeline for the functional annotation of excretory/secretory (ES) proteins – collectively termed the “secretome” – in *Haemonchus contortus*, a parasitic nematode of major veterinary importance. This integrated workflow combines five complementary annotation strategies, each refined and benchmarked for accuracy, to predict molecular functions, biological processes, and metabolic pathways. The pipeline has been iteratively enhanced to incorporate advanced tools, including structure-informed deep learning methods such as AlphaFold3. Using this optimised workflow, we functionally annotated 2,591 of 3,353 predicted ES proteins (77.3%) in *H. contortus*, representing a 10-25% improvement over previous approaches relying on individual tools and default parameters. These results demonstrate the utility, flexibility, and scalability of our pipeline for the functional annotation of genes and gene products across diverse eukaryotic organisms.

ID: 267 / CP2: 3

Contributed abstract

Conference Topics: Genomics, Host-parasite interactions

Keywords: lymphatic filariasis, *Wuchereria bancrofti*, population genomics, Pacific history, host-parasite, migration

Genetic differences between islands in Samoa of *Wuchereria bancrofti*, the filarial nematode parasite that causes lymphatic filariasis, is strikingly congruent with differences in human genomes

Shannon M Hedtke^{1,2}, Warwick N Grant¹, Helen J Mayfield^{3,4}, Neha Sirwani¹, Robert Tomsen⁵, Millicent Opoku¹, Constantin Constantinoiu⁶, Colleen L Lau^{3,4}, Patricia M Graves⁶

¹Department of Environment and Genetics, La Trobe University, Australia; ²La Trobe Institute for Molecular Science, Bundoora, VIC, Australia; ³University of Queensland Centre for Clinical Research, Faculty of Health, Medicine, and Behavioural Sciences, The University of Queensland, Brisbane, Queensland, Australia; ⁴School of Public Health, Faculty of Health, Medicine, and Behavioural Sciences, The University of Queensland, Brisbane, Queensland, Australia; ⁵Samoa Ministry of Health, Apia, Samoa; ⁶College of Public Health, Medical and Veterinary Sciences, James Cook University, QLD, Australia

In the South Pacific, prevalence of *Wuchereria bancrofti* is persistently high, with intense transmission putting >14.7 million at risk of infection. Infection causes lymphatic filariasis, whose chronic manifestations include irreversible and painful swelling of the limbs and scrotum. We identified 515,485 autosomal variants from whole genomes of 74 *W. bancrofti* from the islands of Upolu and Savai'i in Samoa and five from Papua New Guinea. Consistent with published work on 3,316 human genomes, Samoan *W. bancrofti* are genetically distinct from PNG parasites. We hypothesize that Samoan parasites arrived with the original wave of Austronesian settlers and that later immigration of Papuan people to Samoa did not introduce genetically distinct *W. bancrofti*. Genetic structure is strikingly congruent between parasites and people: the lack of parasite differentiation near urban areas on Upolu mimics the impact of urbanization on human genetic differentiation. Incongruously, parasites from Le'auva'a were genetically distinct, despite its closeness to urban Apia. Post-hoc examination found an explanation for this discrepancy: Le'auva'a was founded by people relocated from Savai'i during the 1905-1911 eruption of Mt Matavanu. The overall congruence between genetic differentiation of parasites and migration of people is strong support for our hypothesis that parasite genomics can illuminate human history.

ID: 205 / CP2: 4

Contributed abstract

Conference Topics: Bioinformatics, Helminthology, Livestock Parasites, Veterinary Parasitology

Keywords: Chemosensation, HPM, Transcriptomics, Microfluidics

Ascaris chemosensation regulates hepatopulmonary migration in the early phase of the infection of Ascariasis

Pradip Roy^{1,2}, Joy Liu^{2,3}, Peter Thurgood⁴, Amrita Vijay^{2,3}, Louise Baker^{2,3}, Balu Balan^{2,3}, Khashayar Khoshmanesh⁴, Aaron Jex^{1,2,3}

¹Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, University of Melbourne, Parkville, VIC 3010, Australia; ²Walter and Eliza Hall Institute of Medical Research, Parkville, VIC 3052, Australia; ³Department of Medical Biology, University of Melbourne, Parkville, VIC 3052, Australia; ⁴School of Engineering, RMIT University, Melbourne, Victoria, Australia

Ascariasis is a major helminthiasis that affects ~800 million people world-wide. New treatments are urgently needed, particularly to prevent reinfection. Hepatopulmonary migration (HPM) is an essential phase of *Ascaris* infection involving transit through the host liver and then lung before maturation in the small intestine. We investigated whether, contrary to current dogma, *Ascaris* HPM is an active process regulated by chemotaxis. Hence, we explored the role of chemosensation in guiding larval *Ascaris suum* during infection. The chemosensory genes and pathways of *A. suum* were curated through comparative analysis with orthologous genes in *Caenorhabditis elegans*. We conducted tissue-specific transcriptional studies, identifying a chemosensory pathway specifically present in the head and amphidial tissues of *Ascaris*. We showed that freshly hatched *A. suum* larvae migrate toward pig liver homogenates and engage in locomotory behaviours consistent with foraging and targeted movement under attractive chemotaxis using microfluidics. Notably, this appears tissue-specific, with similar exposures to lung preparations having no comparable effect. Transcriptomic analysis confirmed tissue-specific up-regulation of chemosensory signalling, cilium production and metabolic pathways in liver-stimulated larvae providing molecular support for attractive chemotaxis in HPM, consistent with observations of larvae *in vivo*. Future studies will aim to identify target-receptor interactions to potentially block chemotaxis and disrupt HPM.

ID: 226 / CP2: 5

Contributed abstract

Conference Topics: Bioinformatics, Genomics, Molecular Biology

Keywords: MicroRNAs, isomiRs, bioinformatic tool, small RNA sequencing

E.M.M.A.: Enhanced Multispecies IsomiR Analyser Tool: A bioinformatic pipeline for the assessment of IsomiR patterns across parasitic development

Dayna Sais¹, Phuong Thao Nguyen², Sumaiya Chowdhury³, Sheila Donnelly^{3,4}, Nham Tran¹

¹School of Biomedical Engineering, Faculty of Engineering and Information Technology, University of Technology Sydney, Sydney, Australia; ²Transdisciplinary School, University of Technology Sydney, Sydney, Australia; ³School of Life Sciences, Faculty of Science, University of Technology Sydney, Sydney, Australia; ⁴School of biological and chemical sciences, University of Galway, Galway, Ireland

MicroRNAs (miRNAs) and their isoforms (isomiRs) are increasingly recognised as key regulatory molecules in parasite biology. IsomiRs arise through sequence variations of miRNAs and can modulate target specificity, alter miRNA stability, and expand regulatory potential, making their accurate annotation essential for understanding fine-scale gene regulation.

Existing tools for isomiR analysis are often restricted to model species, lack customisation, and provide limited insights into biologically meaningful variants. To address these gaps, we developed **E.M.M.A.** (Enhanced Multispecies isomiR Analysis and Visualisation Tool), a bioinformatic pipeline designed for flexible, in-depth isomiR analysis across diverse species.

E.M.M.A. identifies, classifies, and quantifies isomiRs, detects SNPs, and evaluates 5' and 3' end modifications, distinguishing between templated and non-templated extensions using genomic coordinates. It assigns isomiRs to 12 distinct classes and

produces intuitive visualisations for downstream biological interpretation. We tested E.M.M.A. on small RNA sequencing datasets from *Fasciola hepatica* and *Schistosoma mansoni*, capturing stage-specific isomiR signatures, highlighting its relevance to parasitology research.

E.M.M.A. enables more comprehensive interrogation of isomiR biology, offering new insights into the adaptive RNA-based regulation employed by parasitic species. Our tool offers a novel route to uncover functional isomiR dynamics in parasites, providing insight into host-parasite interactions and RNA-based regulation

CP3: Pet Parasites sponsored by Vetoquinol

Time: Tuesday, 01/July/2025: 11:30am - 12:30pm · Location: Conference room 3

Session Chair: Don Strazzeri, Vetoquinol Australia Pty Ltd

ID: 124 / CP3: 1

Contributed abstract

Conference Topics: Climate Change, Ecology, Epidemiology, Parasites of companion animals, Veterinary Parasitology

Keywords: *Dirofilaria immitis*, heartworm, risk-based prevention, evidence-based medicine

Temperature-dependent disruption to canine heartworm transmission: how current and future climate could impact clinical decision making

Peter Atkinson¹, Mark Stevenson², Ryan O'Handley¹, Torben Nielsen¹, Charles Caraguel³

¹University of Adelaide, Australia; ²University of Melbourne, Australia; ³University of Sydney, Australia

Canine heartworm disease is caused by chronic infection with *Dirofilaria immitis*, with the progression of disease relying on the build-up of high worm burdens requiring repeated infection events. This nematode has a mosquito vector, with the necessary maturation of larval stages within the mosquito only possible when the average ambient temperature is sustained above 14°C. Therefore, like other filarioses, it is considered a hot weather condition with higher occurrence reported in areas experiencing continuous warm weather for most of the year.

Across Australia's territory, we used temperature records to identify areas where recent temperature conditions would disrupt transmission, then classified locations experiencing disrupted transmission either year-round, seasonally or never. Overall, temperature conditions disrupted transmission of *D. immitis* for most of the populated areas in Australia. We collated our findings into an online, open access dashboard called "Transmission Tracker – *Dirofilaria*", available at <https://heartworm-mapping.adelaide.edu.au/shiny/>. This presentation will demonstrate the utility of our dashboard in veterinary clinical decision making and suggest alternative heartworm management strategies to the currently recommended blanket and year-round preventative use, as well as explore the impact of past, present and future climate on transmissibility.

ID: 155 / CP3: 2

Contributed abstract

Conference Topics: Ectoparasites, Epidemiology, Parasites of companion animals, Parasites of dogs, Veterinary Parasitology, Wildlife parasitology

Keywords: Tick-borne diseases, *Babesia* sp., *Ehrlichia canis*, *Dirofilaria immitis*, *Candidatus Rickettsia jingxinensis*

Prevalence of ticks and vector-borne pathogens in wild canids in southeast Queensland, Australia

Grace Reeves¹, Justine Gibson¹, Rowland Cobbold², Swaid Abdullah¹

¹University of Queensland, Australia; ²Southern Cross University

Tick-borne diseases (TBD) are an increasing concern within Australia and worldwide, presenting significant challenges due to complex interactions among various pathogens and their hosts. In south-east Queensland (SEQ), limited research on tick-borne pathogens makes defining the status of TBDs difficult. Recent findings, such as the emergence of *Ehrlichia canis* in free-roaming dogs in remote areas, highlight the need to reassess its epidemiology, especially with the encroachment of wild dogs and foxes into urban areas, which may serve as pathogen reservoirs. This study investigated 125 wild canid cadavers, collecting blood, spleen, and tick samples for molecular testing of *E. canis*, *Rickettsia* spp., and *Babesia* species. Neither *E. canis* nor *Babesia* spp. were detected. However, *Candidatus Rickettsia jingxinensis* was found in a tick, and *Hepatozoon canis* was identified in a fox. Additionally, four foxes tested positive for heartworm (*Dirofilaria immitis*), with one fox having a concurrent infection of *H. canis*, *D. immitis*, and its endosymbiont *Wolbachia*. Our findings indicate that wild canid populations in SEQ may harbour a wide variety of vector-borne pathogens. The study highlights the need for increased sampling coverage for a more comprehensive assessment of the status of these pathogens and their transmission in wild canid populations in Australia.

ID: 273 / CP3: 3

Contributed abstract

Conference Topics: Epidemiology, Parasites of companion animals, Parasites of dogs

Keywords: canine, vector-borne, Australia, KAP, veterinarians

A nationwide cross-sectional study on canine vector-borne pathogens in pet dogs and associated awareness and preventative practices of veterinarians and dog owners in Australia.

Ushani Atapattu¹, Anke Wiethoelter¹, Luca Massetti¹, Louise Rae², Rebecca Traub^{1,3}, Phillip McDonagh², Vito Colella¹

¹Melbourne Veterinary School, University of Melbourne, VIC 3010, Australia; ²Boehringer Ingelheim Animal Health Australia, North Ryde, New South Wales 2113, Australia; ³Department of Infectious Diseases and Public Health, City University of Hong Kong, Hong Kong

Canine vector-borne pathogens (CVBP) have been reported in dogs in Indigenous communities in Australia, but information on pet dogs from urban areas is limited. A cross-sectional survey of 946 blood samples from pet dogs in urban and peri-urban areas was conducted to assess common CVBP prevalent in Australia. Two questionnaires targeting Australian veterinarians and dog owners were administered to assess their awareness and preventative practices regarding CVBP. Overall, the

apparent prevalence of CVBP in pet dogs was 3.3% (95% CI 2.2–4.6%), with *A. platys*, haemotropic mycoplasma, and filarial worms having apparent prevalences of 0.1% (95% CI 0–0.6%), 2.5% (95% CI 1.6–3.8%), and 0.5% (95% CI 0.2–1.2%), respectively. Higher infection levels were associated with non-temperate climatic zones (OR 5, 95% CI 2.2–12.6, $p < 0.001$) and regions with lower socioeconomic status (OR 2.2, 95% CI 1.1–4.6, $p = 0.029$). One dog from Woree, Queensland, was found molecularly positive for *H. canis*. Despite low awareness of CVBP among veterinarians and dog owners, effective prophylaxis recommendations and administration practices were identified. To sustain consistently low CVBP prevalence, we recommend educating dog owners, particularly in regions with higher infection rates, and implementing targeted, risk-based control measures across Australia.

ID: 214 / CP3: 4

Contributed abstract

Conference Topics: One Health, Parasites of cats, Parasites of companion animals, Veterinary Parasitology, Zoonoses

Keywords: Toxoplasma, cat, raw meat, zoonosis

Toxoplasma gondii oocyst excretion in a suburban house cat fed raw pet food

Ryan O'Handley¹, Emily Dean²

¹Adelaide University, Australia; ²Wayville Animal Hospital, Wayville SA

The practice of feeding raw meat to pet cats and dogs is increasing. Raw pet food is now commonly available online and at grocery stores. The feeding of raw meat to dogs and cats is concerning from a parasite transmission standpoint as many raw meat products are minced and contain a mixture of meat types. In this case report, assistance in managing a *Giardia* infection in 2 house cats, 1-year of age, from suburban Adelaide was requested. A faecal sample from one of the cats was sent to the Roseworthy Veterinary Hospital Diagnostic lab to examine for the presence of *Giardia* cysts by faecal flotation. In addition to *Giardia* cysts, numerous *Toxoplasma gondii* oocysts were observed. The owner confirmed that the cats were being fed a diet consisting of raw beef but may have also been fed raw duck and kangaroo meat. In addition, the owner of the cats was pregnant at the time of the *T. gondii* diagnosis. These oocysts were infectious to laboratory mice, producing tissue cysts and has now been established in cell culture as the "Wayville Isolate". This case demonstrates the potential risk to pet owners and the public when raw meat is fed to pets.

CP1.1: Drugs & Drug Resistance 5 min talks

Time: Tuesday, 01/July/2025: 12:15pm - 12:30pm · *Location:* Conference room 1

Session Chair: Hayley Bullen, Burnet Institute

Session Chair: Brad Sleebs, Walter and Eliza Hall Institute

ID: 245 / CP1.1: 1

Contributed abstract

Conference Topics: Apicomplexa Biology, Drugs, Malaria

Keywords: Plasmodium falciparum Drug Resistance | Molecular Parasitology | Infectious Diseases Research

New insights on the mode of action of the malaria drug proguanil

Patrick Tumwebaze¹, Gillian M Fisher¹, Joshua Morrow³, Carlo Giannangelo³, John H. Ryan², Andrew G Riches², Jacinta R Macdonald¹, Darren Creek³, Ghizal Siddiqui³, Tina S Skinner-Adams¹, Katherine T. Andrews¹

¹Griffith University, Nathan Campus, QLD, Australia; ²Commonwealth Scientific and Industrial Research Organization;

³Monash University, Victoria, Australia

The drug combination atovaquone-proguanil (initially sold as Malarone[®]) has been used for >50y for prevention and treatment of malaria. Atovaquone is a cytochrome *bc₁* inhibitor and proguanil (PG) is metabolised to the dihydrofolate reductase (DHFR) inhibitor cycloguanil and synergises atovaquone activity. While PG was thought to lack potent intrinsic activity, we have shown that proguanil and a metabolism-blocked analogue (fBuPG) have potent intrinsic anti-plasmodial activity, that occurs via a slow action mechanism[1] The novel slow action activity of proguanil is different to the action of its metabolite cycloguanil and to other slow-acting antimalarial agents like delayed death inhibitors (e.g., clindamycin), but remains un characterised. In this presentation, we will provide an update on the use of solvent proteome profiling with *Plasmodium falciparum* protein lysates and proguanil to investigate the protein target/s of this drug.

Reefrence

1. Skinner-Adams, T.S., et al., *Cyclization-blocked proguanil as a strategy to improve the antimalarial activity of atovaquone*. *Commun Biol*, 2019. **2**: p. 166.

ID: 106 / CP1.1: 2

Contributed abstract

Conference Topics: Protozoa

Keywords: Blastocystis, 5-Fluorouracil, Colorectal cancer, Chemotherapy, Nrf2

Impact of Blastocystis sp. on the Efficacy of 5-Fluorouracil in Colorectal Cancer Treatment: An In Vitro Investigation

Vinoth Kumarasamy¹, Suresh Kumar Govind²

¹National University of Malaysia; ²University of Malaya

Blastocystis sp. (*Blastocystis*) is an enteric protozoan parasite with extensive genetic diversity and unclear pathogenicity, commonly associated with gastrointestinal symptoms in immunocompromised individuals. This study examines the *in vitro* effects of *Blastocystis* on the efficacy of 5-fluorouracil (5-FU), a widely used chemotherapeutic agent for colorectal cancer (CRC). HCT116 human colon cells and CCD-18Co normal colon fibroblast cells were treated with solubilized *Blastocystis* antigen in the presence of 5-FU. The inhibitory potency of 5-FU at 8 μ M and 10 μ M significantly decreased from 57.7% to 31.6% ($p < 0.001$) and 69.0% to 36.7% ($p < 0.001$), respectively, when co-incubated with *Blastocystis* antigen for 24 hours.

However, the inhibitory effect of 5-FU on CCD-18Co cells was not significantly affected in the presence of Blastocystis antigen. The presence of Blastocystis antigen was associated with the upregulation of type 2 cytokines, transforming growth factor-beta (TGF- β), and nuclear factor E2-related factor 2 (Nrf2) gene expression in HCT116. These findings suggest that Blastocystis infection may interfere with CRC chemotherapy by modulating key molecular pathways, potentially reducing 5-FU efficacy. Understanding the role of Blastocystis in CRC treatment outcomes is crucial for optimizing therapeutic strategies.

ID: 107 / CP1.1: 3

Contributed abstract

Conference Topics: Protozoa

Keywords: Blastocystis, 5-Fluorouracil, Colorectal Cancer, In Vivo Model, Chemotherapy Resistance, Aberrant Crypt Foci, Inflammation, Oxidative Stress

Blastocystis sp. Infection Compromises 5-Fluorouracil Efficacy in Colorectal Cancer Progression: Evidence from an In Vivo Model

Vinoth Kumarasamy¹, Suresh Kumar Govind²

¹National University of Malaysia; ²University of Malaya

This study investigates the effect of *Blastocystis* sp. (Blastocystis) infection on the efficacy of 5-fluorouracil (5-FU) in colorectal cancer (CRC) progression. Thirty male Wistar rats were divided into six groups: Control, A (azoxymethane [AOM]), A-30FU (AOM + 30 mg/kg 5-FU), B-A-30FU (Blastocystis + AOM + 30 mg/kg 5-FU), A-60FU (AOM + 60 mg/kg 5-FU), and B-A-60FU (Blastocystis + AOM + 60 mg/kg 5-FU). All AOM-treated rats developed aberrant crypt foci (ACF), but Blastocystis-infected groups exhibited significantly higher ACF numbers, indicating enhanced carcinogenic progression despite 5-FU treatment. The reduction in ACF formation was more pronounced at a higher 5-FU dose, but Blastocystis infection still limited its effectiveness. Histopathological analysis revealed severe intestinal damage, increased epithelial sloughing, and inflammatory changes in infected rats, particularly in the B-A-30FU group. Additionally, Blastocystis-infected rats displayed slower weight gain and loose stools. These findings suggest that Blastocystis may interfere with 5-FU activity, potentially through inflammation and oxidative stress, leading to reduced chemotherapy efficacy. Further studies are necessary to understand the underlying mechanisms and the clinical relevance of Blastocystis infection in CRC treatment.

CP2.1: Helminth Biology 5 min talks

Time: Tuesday, 01/July/2025: 12:15pm - 12:30pm · *Location:* Conference room 2

Session Chair: Nichola Eliza Davies Calvani, The University of Sydney

Session Chair: Charles Gauci, University of Melbourne

ID: 168 / CP2.1: 1

Contributed abstract

Conference Topics: Strongyloides

Keywords: Strongyloides, Strongyloides papillosus, Molecular characterisation

Molecular Characterisation of Strongyloides spp. from Australian Dairy Goats

Jingjing Zhang¹, Endris Aman Ali¹, Huan Zhao², Charles Gauci², Elysia Ling¹, Abdul Jabbar¹

¹University of Melbourne, Australia; ²James Cook University

Members of the genus *Strongyloides* (Nematoda; Rhabditida) infect a wide range of animals, including humans, and can cause significant economic losses in livestock industries. *Strongyloides papillosus* is known to infect ruminants (e.g., cattle, goats, and sheep) globally and can cause intestinal damage, malabsorption, and growth retardation. Although our understanding of the important internal parasites of Australian goats is increasing, there is still limited knowledge about the prevalence and genetic features of *Strongyloides* in goats. This study aims to genetically characterise *Strongyloides* species in faecal samples of dairy goats across Australia. Between November 2023 and July 2024, 1,028 faecal samples collected from 68 dairy goat farms were analysed using the Modified McMaster technique for faecal egg counts. DNA was extracted from individual faecal samples. *Strongyloides* spp. will be characterised using PCR amplification of the 18S rRNA hyper-variable regions (HVR-I and HVR-IV), and mt *cox1* genes. Bioinformatic analyses will then be used to assess the genetic profiles *Strongyloides* spp. detected in Australian goats. The expected outcomes of this study include identifying *Strongyloides* species present in Australian dairy goats and characterising its genetic features.

ID: 225 / CP2.1: 2

Contributed abstract

Conference Topics: Bioinformatics, Fasciolosis/Liver fluke

Keywords: isomiRs, Developmental RNAs, Fasciola, genomics

Temporal Shifts in Small ncRNA Variants During Fasciola Hepatica Maturation

Nham Tran¹, Dayna Sais¹, Sumaiya Chowdury¹, Phuong Thao Nguyen², Krystina Cwilinski³, Trung Duc Nguyen⁴, Tuan Anh Nguyen⁴, John Dalton⁵, Sheila Donnelly⁶

¹School of Biomedical Engineering, Faculty of Engineering and Information Technology, University of Technology Sydney, Sydney, Australia; ²Transdisciplinary School, University of Technology Sydney, Sydney, Australia; ³Institute of Infection, Veterinary and Ecological Sciences, University of Liverpool, UK; ⁴Division of Life Sciences, the Hong Kong University of Science and Technology, Hong Kong, China; ⁵Centre for One Health School of Natural Sciences, Ryan Institute, University of Galway, Galway, Ireland; ⁶School of biological and chemical sciences, University of Galway, Galway, Ireland

MicroRNAs (miRNAs) have been established as critical regulators of gene expression in helminths, influencing parasite development and host-parasite interactions. While most studies focus on canonical miRNA sequences, we investigated miRNA isoforms (isomiRs) expressed across the developmental stages of *Fasciola hepatica*. IsomiRs are sequence variants of canonical miRNAs, and display differences in stability, target recognition, and efficacy of gene regulation. Our analysis revealed a significant shift in isomiR distribution across parasite maturation, with newly excysted juveniles (NEJ) exhibiting the

highest abundance and diversity of isomiRs, many of which were the dominant form. Among these, 3' isomiRs were most prevalent, typically featuring non-templated uridine additions, followed by adenosine. These modifications can enhance miRNA stability or alter their interaction with RNA-binding proteins. Additionally, over 10% of isomiRs in NEJs were 5' variants, known to shift seed sequences and redirect target specificity, potentially reprogramming host or parasite gene expression. Additionally, cleavage site analysis revealed that *F. hepatica* isomiRs share features with mammalian miRNA cleavage, suggesting conservation of biogenesis mechanisms. Together, these findings suggest that isomiRs contribute to regulatory plasticity during *F. hepatica* development, potentially enhancing parasite adaptation to dynamic host environments and enabling stage-specific fine-tuning of gene expression.

ID: 223 / CP2.1: 3

Contributed abstract

Conference Topics: Aquaculture, Bioinformatics, Fish parasitology, Genomics

Keywords: Blood fluke, Mitochondrial genome, Aquatic animal health, Nanopore, Hi-C

Elucidating the genomes of the blood flukes *Cardicola forsteri* and *Cardicola orientalis* (Trematoda: Aporocotylidae)

Jemma Hudson¹, Sunita Sumanam², Bronwyn Campbell¹, Lachlan Coff³, Barbara Nowak⁴, Paul Ramsland^{1,5,6}, Neil Young², Nathan Bott¹

¹School of Science, RMIT University, Australia; ²Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Australia; ³Australian Centre for Disease Preparedness, CSIRO, Australia; ⁴Institute for Marine and Antarctic Studies, University of Tasmania, Australia; ⁵Department of Immunology, Monash University, Australia; ⁶Department of Surgery, Austin Health, The University of Melbourne, Australia

Parasitic diseases can be a significant constraint on aquaculture, an industry that continues to develop in response to the rise in global demand for sustainable protein sources. Aporocotylid blood flukes, *Cardicola forsteri* and *Cardicola orientalis*, are economically significant parasites of Southern bluefin tuna (Australia), Pacific bluefin tuna (Japan), and Atlantic bluefin tuna (Mediterranean) as they are responsible for blood vessel obstruction in the gills leading to branchitis and mortalities when untreated. Genomic information is limited for these species. Here, we have defined the mitochondrial genomes for these species and assembled the nuclear genome of *C. forsteri* to a chromosome level. Oxford nanopore-long read sequencing was used to sequence *C. orientalis* from a single individual. Hi-C sequencing for chromosome assembly was done for a pool of adult *C. forsteri*. Both *Cardicola* spp. mitogenomes contained the typical 36 genes, however there was significant nucleotide variation between the two species, despite their common locality and shared host. A long repetitive control region was identified for each species, however the region for *C. forsteri* was longer, and the overall pattern differed between the two species. The *C. forsteri* genome has 8 chromosomes. These are the first mitochondrial genomes, and chromosome assembly for any aporocotylid.

CP4: Cell & Molecular Biology 15 min talks

Time: Tuesday, 01/July/2025: 1:30pm - 2:30pm · *Location:* Conference room 1

Session Chair: Jill Chmielewski, WEHI

Session Chair: Giel G van Dooren, Australian National University

ID: 264 / CP4: 1

Contributed abstract

Conference Topics: Apicomplexa Biology, Biochemistry, Cell Biology, Host-parasite interactions, Molecular Biology, Protozoa

Keywords: Toxoplasma, CRISPR, Differentiation, ubiquitination, Metabolism

The requirements for differentiation of *Toxoplasma* into latent forms revealed by *in vitro* and *in vivo* gene disruption CRISPR screens

Alex Uboldi^{1,2}, Sachin Khurana^{1,2}, Amalie Jaywickrama^{1,2}, Amber Simonpietri^{1,2}, Sai Lekkala^{1,2}, Karan Singh^{1,2}, Nicholas Katris^{1,4}, Ushma Ruparel^{1,2}, Nicholas Scott³, Malcolm McConville⁴, David Komander^{1,2}, Simon Cobbold^{1,2}, Chris Tonkin^{1,2}

¹The Walter and Eliza Hall Institute of Medical Research, Australia; ²Department of Medical Biology, The University of Melbourne; ³Department of Microbiology and Immunology, Peter Doherty Institute, The University of Melbourne; ⁴Department of Biochemistry and Pharmacology, Bio21 Molecular Science and Biotechnology Institute, The University of Melbourne

Infection with *Toxoplasma* parasites is a risk factor for congenital toxoplasmosis, progressive retinal damage, and encephalitis in immune suppressed individuals. Following initial acute infection with *Toxoplasma*, the parasites differentiate from acute stage tachyzoites into encysted bradyzoites in the central nervous system and muscle. These latent forms are resistant to both immune clearance and all currently available drugs. How parasites sense these tissue types and trigger differentiation remains largely unknown. Therefore, understanding the factors that regulate differentiation may lead to new therapeutic interventions. Through a whole genome gene disruption CRISPR screen, as well as more focused *in vitro* and *in vivo* screens, we have identified 25 new genes required for differentiation. These include genes that encode nucleic acid binding proteins and enzymes involved in central carbon metabolism and ubiquitination. We bring to light that the metabolic environment of the infected host cell influences the efficiency of differentiation, and that an E3 ubiquitin ligase acts to regulate expression of BFD1, the major transcription factor controlling differentiation, thereby adding new layers to our understanding of this process. This study also lays bare the power of CRISPR screens to illuminate important biological processes.

ID: 159 / CP4: 2

Contributed abstract

Conference Topics: Biochemistry, Host-parasite interactions, Protozoa

Keywords: Leishmania, metabolism, infection

Systematic analysis of *Leishmania* central carbon metabolism.

Fleur Sernee, Eleanor Saunders, Vinzenz Hofferek, Julie Ralton, Lilith Flint, Malcolm McConville

The University of Melbourne Department of Biochemistry and Pharmacology, Bio21 Molecular Science and Biotechnology Institute, School of Biomedical Sciences/ Faculty of Medicine, Dentistry & Health Sciences, Parkville, Melbourne, Australia

The sandfly-transmitted protozoa parasite *Leishmania spp* proliferate within the mature lysosome of macrophages in the mammalian host. In order to understand the metabolic requirements of these intracellular stages we have generated a library of metabolic mutants, using CRISPR/Cas9, lacking key enzymes in central carbon metabolism. The biochemical and virulence phenotypes of these mutant lines have been defined using comprehensive metabolomic analysis and infection assays in macrophages and susceptible mice. Most of the targeted genes were successfully deleted and grew normally in rich media, reflecting the metabolic flexibility of these parasites, and extensive redundancy. However, a significant majority of mutants lacking all homologues of the same enzyme displayed reduced or severely attenuated virulence in macrophages and/or susceptible mice. Interestingly, some mutants exhibited highly attenuated virulence in inflammatory (M1) macrophages, but not in non-inflammatory (M2) macrophages revealing differences in nutrient levels and/or restrictive microbicidal processes in these different macrophage populations. These analyses have highlighted the importance of glycolysis for ATP synthesis, as well as distinct pathways of glutamate synthesis that are essential for intracellular parasite survival. Overall, this strategy has provided high confidence validation of essential metabolic pathways in these parasites that might be targets for new and more effective therapies.

ID: 295 / CP4: 3

Contributed abstract

Conference Topics: Malaria

Keywords: clindamycin resistance, methionine formylation pathway

A novel mechanism of clindamycin resistance in *Plasmodium*

Jessica Home¹, Hayley Buchanan¹, Lee Yeoh², James Beeson², Michael Duffy³, Geoffrey McFadden¹, Dean Goodman¹

¹School of BioSciences, The University of Melbourne, Melbourne, Australia; ²Burnet Institute, Melbourne, Victoria, Australia;

³Department of Microbiology and Immunology, The University of Melbourne, Melbourne, Australia

Clindamycin is a well-tolerated antibiotic that kills malaria parasites by targeting the apicoplast prokaryotic translational machinery, but mechanisms of resistance in *Plasmodium* remain largely unknown.

We selected for clindamycin resistance in *P. falciparum* and in *P. berghei*. Clindamycin resistant *P. falciparum* had point mutations in the peptidyl transferase site of apicoplast-encoded 23S ribosomal RNA—the same mechanism of clindamycin resistance in bacteria. These mutants exhibited impaired development in mosquitoes, suggesting a fitness cost associated with resistance that should restrict spread of such resistance. In contrast, two independently generated clindamycin resistant *P. berghei* lines had loss-of-function mutations in different nucleus-encoded genes responsible for formylating the initiator methionine used for apicoplast translation. Changes to this machinery have never been connected to clindamycin resistance in any organism.

To validate this novel resistance mechanism, we disrupted initiator methionine formylation genes in both *P. berghei* and *P. falciparum* and confirmed clindamycin resistance. Interestingly, methionine formylation mutants conferred lower resistance levels than 23S rRNA mutants but had no impairment of mosquito transmission, suggesting weaker resistance but more facile spread. Clindamycin resistance in *Plasmodium* is thus more complex than anticipated, and further investigation of the methionine formylation pathway is imperative to dissect this new resistance mechanism.

ID: 244 / CP4: 4

Contributed abstract

Conference Topics: Host-parasite interactions, Malaria

Keywords: Malaria, host-parasite interaction, clinical metabolomics

Parasite-Host Metabolic Cross-Talk to Detect Malaria

Teha Shumbej Gebi¹, Christopher MacRaid¹, Joshua Morrow Morrow¹, Endalew Zemen², Darren Creek¹

¹Monash University, Australia; ²Jimma University, Ethiopia

Current malaria diagnostic methods face practical and diagnostic limitations, often lacking the required sensitivity. Previous research in our lab has revealed a significant bystander effect in uRBCs metabolism when co-cultured with infected RBCs (iRBCs) in the same culture dish. Given the relatively low number of iRBCs in malaria patients, measuring the biochemistry of more abundant bystander uninfected RBCs (uRBCs) could provide a highly sensitive approach to malaria detection and offer a better insight into the disease's pathogenesis.

The in vitro metabolomic profile validation of DBS (dry blood spot) and blood samples highlights the efficacy of DBS samples in malaria metabolomics studies. Clinical DBS samples were collected from healthy controls and malaria patients with a range of infection intensity in a malaria-endemic region of Ethiopia. DBS metabolomics analysis was performed using High-resolution LC-MS.

Univariate and multivariate clinical DBS metabolomics analyses revealed a significant metabolic signature associated with malaria infection, including perturbation in glycolytic metabolites in both symptomatic and asymptomatic malaria cases, compared to non-malaria pyretic patients and healthy controls. Pathway enrichment analysis identified the most affected metabolic pathways in both symptomatic and asymptomatic malaria. Additionally, candidate host metabolites with potential as diagnostic markers for malaria were identified.

CP5: Epidemiology 15 min talks

Time: Tuesday, 01/July/2025: 1:30pm - 2:30pm · Location: Conference room 2

Session Chair: Shannon M Hedtke, La Trobe University

Session Chair: Kirsty M Mccann, Deakin University

ID: 215 / CP5: 1

Contributed abstract

Conference Topics: Epidemiology, Genomics, Helminthology

Keywords: Onchocerciasis, Blackfly, Genomics, Vector Migration, Cross-border Transmission

Persistence transmission of onchocerciasis in Southwest Ethiopia, 2001 to 2021

Sindew Mekasha Feleke, Shannon Hedtke, Emily Hendrickson, Millicent Opoku, Warwick Grant

La Trobe University, Australia

Onchocerciasis, a debilitating skin and eye disease transmitted by blackflies, affects over 240 million people globally, with 99% of cases in Africa, including 25 million in Ethiopia. Mass drug administration (MDAi) with ivermectin was introduced in Ethiopia in 2001, initially targeting hyper- and meso-endemic districts in the Keffa and Sheka zones. It was later expanded to 285 districts across the country. The MDAi program, expected to interrupt transmission after 12 annual rounds, was administered yearly until 2012, then biannually from 2013. However, by 2021, despite over 20 years of good coverage (above 80%), transmission interruption goals were not achieved. Serological and entomological data indicated persistent transmission. Genetic analysis of blackfly populations revealed substantial movement between endemic areas over a wide geographical range, with weak genetic isolation-by-distance. Dispersal distances of 80-100 km were observed for at least one subspecies of *Simulium damnosum*. This migration suggests that transmission zones may extend beyond the boundaries currently used for program implementation. As a result, vector movement could play a crucial role in the ongoing transmission observed in some areas. The findings support the use of vector genomics to refine transmission zone definitions and recommend targeting MDAi within these newly identified zones for improved program.

ID: 259 / CP5: 2

Contributed abstract

Conference Topics: Genomics, Malaria

Keywords: Population genomics, parasite relatedness, malaria genomics, Plasmodium falciparum

Population genetic signatures of Plasmodium falciparum transmission decline and rebound in a hyperendemic area of Papua New Guinea

Kirsty M Mccann^{1,2}, Zahra Razook^{1,2}, Dulcie Lautu-Gumal², Somya Mehra², Shazia Ruybal-Pesantez⁴, Jessy Vibin³, Elma Nate⁵, Maria Ome-Kaius⁵, Moses Laman⁵, Ivo Mueller⁶, Leanne Robinson², Alyssa E Barry^{1,2}

¹Centre for Innovation in Infectious Disease and Immunology Research (CIIDIR), Institute for Mental and Physical Health and Clinical Treatment (IMPACT), School of Medicine, Deakin University, Geelong, Victoria, AUSTRALIA; ²Life Sciences Discipline, Burnet Institute, Melbourne, Victoria, AUSTRALIA; ³Deakin University, Waurn Ponds, Victoria, AUSTRALIA; ⁴School of Public Health, Imperial College London, UK; ⁵Vector Borne Diseases Unit, Papua New Guinea Institute of Medical Research, Madang, Papua New Guinea; ⁶Population Health and Immunity Division, Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria, AUSTRALIA

Malaria genomic surveillance is key to understanding how control strategies drive elimination. Classifying transmission dynamics and zone boundaries helps assess control impact and guide targeted efforts. In Papua New Guinea, extensive measures since 2006 initially reduced parasite prevalence, but recent rebounds and artemisinin resistance have emerged. We used SNP barcoding to genotype 624 *P. falciparum* isolates collected from Madang and East Sepik Provinces at nine time points between 2005-2020. We conducted population genetic analyses to investigate genomic signatures of transmission decline and rebound, and features that may explain the resurgence. Increasing proportions of related parasites were observed during decline consistent with a population bottleneck and more focal transmission, followed by an increase in related pairs in East Sepik in 2016 when prevalence rebounded which were maintained in more recent timepoints, i.e., 2019. These patterns suggest dynamic shifts in transmission, with resurgence allowing for both persistence and diversification of parasite lineages. Relatedness networks reveal the presence of several clonal lineages after transmission reduced, with expansion and recombination of distinct lineages as transmission rebounded. This highlights the need for sustained control to reduce the parasite population and advance malaria elimination in PNG.

ID: 174 / CP5: 3

Contributed abstract

Conference Topics: Epidemiology, Immunology, Malaria

Keywords: Anopheles, Mosquitoes, Salivary Biomarkers, Vector surveillance, Malaria

Characterisation of human antibodies to mosquito salivary proteins following controlled mosquito biting exposure

Freya Fowkes^{1,2,3}, Victor Chaumeau⁴, Ellen Kearney^{1,2}, Sunisa Sawasdichai⁴, Praphan Kittiphanakul⁴, Paul Agius⁵, Katherine O'Flaherty^{1,2}, Julie Simpson¹, Francois Nosten⁴

¹University of Melbourne, Australia; ²Burnet Institute, Melbourne, Australia; ³Monash University, Melbourne, Australia; ⁴Shoklo Malaria Research Unit, Thailand; ⁵Deakin University, Australia

Measurement of human antibodies specific for mosquito salivary antigens has been proposed as an outcome measure to assess human exposure to vector bites. However, only a handful of antigens have been identified and the specificity and longitudinal dynamics of antibody responses are not well known. We conducted a world-first clinical trial of controlled exposure to mosquito bites to identify and validate biomarkers of exposure to malaria and dengue mosquito vectors in Southeast Asia. This trial was an exploratory randomized controlled trial of controlled exposure to mosquito bites with 10 arms corresponding to different species (*Aedes aegypti*, *Ae. albopictus*, *Anopheles dirus*, *An. maculatus* and *An. minimus*) and biting levels (35 or 305 bites total over 6 weeks) and its effect on species-specific antibody levels longitudinally (17 weekly measurements). Antibodies against species-specific candidate antigens were measured by high-throughput ELISA in sera collected from participants

(n=210) before, during and after mosquito challenges. We found that antibody levels against Anopheles and Aedes salivary peptides decayed slowly overall but with small boosts during and after mosquito biting challenges. This research generates important knowledge on species-specific antigens for vector sero-surveillance in Southeast Asia.

ID: 241 / CP5: 4

Contributed abstract

Conference Topics: Bioinformatics, Diagnostics, Epidemiology, Immunology, Malaria

Keywords: Plasmodium vivax, Machine learning, Elimination, Serology, Diagnostics

PvSeroTaT: Optimisation and standardisation of a machine learning method to identify individuals recently infected with *Plasmodium vivax*

Lauren Smith^{1,2}, Dionne Argyropoulos^{1,2}, Nick Walker¹, Janise Lin¹, Macie Lamont¹, Anju Abraham¹, Pailene Lim^{1,2}, Eamon Conway^{1,2}, Jetsummon Sattabongkot³, Marcus Lacerda^{4,5}, Ventis Vahi⁶, Michael White⁷, Ramin Mazhari^{1,2}, Ivo Mueller^{1,2}, Rhea Longley^{1,2,3}

¹Infection and Global Health Division, Walter and Eliza Hall Institute of Medical Research, Melbourne, Victoria, AU;

²Department of Medical Biology, University of Melbourne, Melbourne, Victoria, AU; ³Mahidol Vivax Research Unit, Faculty of Tropical Medicine, Mahidol University, Bangkok, Thailand; ⁴Fundação de Medicina, Tropical Dr. Heitor Vieira Dourado, Manaus, Brazil; ⁵Instituto Leônidas & Maria Deane, (Fiocruz), Manaus, Brazil; ⁶Ministry of Health and Medical Services, Honiara, Solomon Islands; ⁷Unité Malaria: Parasites et Hôtes, Département Parasites et Insectes Vecteurs, Institut Pasteur, Paris, France

The unprecedented persistence of *Plasmodium vivax* is largely driven by the hidden reservoir of hypnozoite liver-stage parasites, presenting a key obstacle to elimination. Here we present a machine learning algorithm that classifies recent *P. vivax* infections using serological biomarkers to identify likely hypnozoite carriers. Antibody levels persist even after infections are cleared, and thus can act as indicators of current and recent *P. vivax* infections. This algorithm underpins the *P. vivax* serological testing and treatment (PvSeroTaT) strategy, an alternative to mass drug administration. Serological assays were performed on samples from year-long observational cohort studies conducted from low-transmission settings in Thailand, Brazil and the Solomon Islands, as well as negative controls ($N=2,635$). We first balanced performance of the sero-diagnostic algorithm against practical feasibility by selecting optimal subsets of serological markers and then trained a random forest classification algorithm. The top-performing combination of eight antigens for classifying serostatus achieved an area under the receiver operated characteristic curve (AUC) of 0.874. An online R Shiny application (PvSeroApp) was developed to automate raw serological data processing, quality control and serostatus classification, and facilitated PvSeroTaT implementation in multiple countries. Ultimately, this sero-surveillance strategy enables targeted anti-hypnozoite therapy and strengthens the toolkit for *P. vivax* elimination.

CP6: Livestock Parasites 15 min talks sponsored by Elanco

Time: Tuesday, 01/July/2025: 1:30pm - 2:45pm · *Location:* Conference room 3

sponsored by Elanco and chaired by Liisa Ahlstrom & Monica Commons Elanco

ID: 221 / CP6: 1

Contributed abstract

Conference Topics: Ectoparasites, Immunology, Livestock Parasites, Vaccines, Veterinary Parasitology

Keywords: Cattle tick, vaccine, reverse vaccinology, tick

Vaccinating against ticks: A novel recombinant cattle tick vaccine

Hannah Siddle¹, Mikayla Crouch¹, Nimitha Ramachandran¹, Ala Tabor^{1,2}

¹The University of Queensland, Queensland Alliance for Agriculture & Food Innovation, Centre for Animal Science, St Lucia 4072, Queensland, Australia; ²The University of Queensland, School of Chemistry & Molecular Biosciences, St Lucia 4072, Queensland, Australia

Cattle ticks are a growing burden on global agriculture. Climate changes are increasing the geographic range of ticks, such as *Rhipicephalus microplus*, and increasing resistance to chemical control makes the need for alternative control methods urgent. A novel vaccine has been developed against *Rhipicephalus australis* (Australian cattle tick) using a reverse vaccinology pipeline, resulting in the identification of two candidate proteins. Two vaccine trials have been conducted to assess efficacy in *Bos taurus* cattle, Trial 1 (n=6/group) and Trial 2 (n=10/group). Both trial protocols involved vaccination followed by a 28-day boost and challenge with larval infestation at one month and 6 months post boost. The efficacy of the vaccines was determined using tick numbers, egg weight and larval emergence. Results of Trial 1 show efficacy of 86% ($p < 0.05$) and reduction of tick fecundity using a combination of the two candidate proteins. The preliminary results of Trial 2 indicate a reduction in tick numbers in vaccinated animals using a higher dose of the two candidate proteins (*Student's t-test*, $p = 0.05$). The results highlight the potential of vaccines as a tool for tick control in agriculture, with ongoing work interrogating the immune responses of cattle to the vaccine.

ID: 243 / CP6: 2

Contributed abstract

Conference Topics: Bioinformatics, Drugs, Epidemiology, Helminthology, Livestock Parasites, Molecular Biology

Keywords: Anthelmintic resistance, Gastrointestinal nematodes, Metabarcoding, Molecular surveillance, Data visualisation dashboard

From paddocks to pixels: A statewide look at worm resistance

Emily Francis¹, Analise McDonald¹, Crystal Elliot¹, Olivia Kelly¹, Mark Westman², Janina McKay-Demeler², Jan Slapeta¹

¹Sydney School of Veterinary Science, Faculty of Science, The University of Sydney, New South Wales 2006, Australia;

²Elizabeth Macarthur Agricultural Institute, New South Wales Department of Primary Industries, Menangle, New South Wales 2568, Australia

Australia's livestock producers are navigating an increasingly complex worm control landscape, with rising levels of anthelmintic resistance threatening the sustainability of grazing systems. While molecular diagnostics have transformed our ability to detect resistance, their value depends on delivering results in a form that is accessible, timely, and meaningful to end users.

We present the WormResistanceMonitor (WoRM) Dashboard: a first-of-its-kind, interactive platform that integrates four years of surveillance data from over 500 livestock properties across New South Wales. Using mixed amplicon metabarcoding and targeted SNP detection, the dashboard visualises gastrointestinal nematode species and resistance to levamisole and benzimidazole across sheep and cattle populations. Users can explore trends by postcode, host species, nematode, resistance marker, and frequency threshold.

This longitudinal dataset reveals the regional extent and persistence of resistance, offering practical insights to guide more targeted and sustainable parasite control.

By bridging the gap between laboratory diagnostics and farm decision-making, this open-access tool marks a step-change in applied parasitology and highlights the value of statewide, collaborative molecular surveillance.

ID: 250 / CP6: 3

Contributed abstract

Conference Topics: Apicomplexa Biology, Drugs, Livestock Parasites, Parasites of cats, Veterinary Parasitology

Keywords: Toxoplasma, Eimeria, livestock, drugs

Development of novel compounds for the treatment and prevention of *Toxoplasma gondii* and *Eimeria* spp in livestock.

Quinn Mackie^{1,2}, Kelly Young³, Maria Gancheva¹, Connor Bury^{1,2}, Emma Mao¹, Stephen Page⁴, Adam McCluskey³, Danny Wilson¹, Ryan O'Handley²

¹Research centre for infectious diseases, School of Biological Sciences, University of Adelaide, Adelaide 5005, South Australia; ²School of Animal and Veterinary Sciences, University of Adelaide, Roseworthy 5371, South Australia; ³Chemistry, School of Environmental and Life Sciences, University of Newcastle, Callaghan, New South Wales, Australia; ⁴Neoculi Pty Ltd., Burwood, Victoria, Australia

Toxoplasma gondii and *Eimeria* spp are related apicomplexan parasites responsible for significant production losses in sheep and poultry respectively. *T. gondii* causes reproductive losses in sheep and no effective prophylactics are available in Australia, whereas poultry producers rely on anticoccidials to varying extents worldwide. We screened a library of 338 novel derivatives of the anticoccidial robenidine. Nine compounds were found to inhibit *T. gondii* growth by >50% (IC₅₀) at concentrations below 320 nanomolar. Our two lead compounds had IC₅₀s of 74 and 125 nanomolar and were 25x and 998x more potent against *T. gondii* than mammalian cells respectively. Resistant parasites developed against both lead drugs share a growth defect, indicating that resistance comes with a fitness cost. We established a chicken-*Eimeria* model to test for the efficacy of our lead drugs against this parasite that causes significant losses in the poultry industry. Initial trials of our lead drugs resulted in a 10 to 30-fold reduction in oocyst shedding, supporting further development of these leads as potential prophylactics and treatments for these economically important livestock pathogens.

ID: 121 / CP6: 4

Contributed abstract

Conference Topics: Immunology, Livestock Parasites, Protozoa, Vaccines, Veterinary Parasitology

Keywords: Tritrichomonas foetus, Bovine trichomonosis, Vaccine, Cattle, Immunity

Immunogenicity and efficacy of an Australian whole-cell killed *T. foetus* vaccine in young bulls experimentally infected with *Tritrichomonas foetus*

Harvey Santos¹, Gry Boe-Hansen^{1,2}, Kieren McCosker¹, Michael McGowan², Hannah Siddle¹, Loan Nguyen¹, Ali Raza^{1,3}, Ala Tabor^{1,4}

¹The University of Queensland, Queensland Alliance for Agriculture & Food Innovation, Centre for Animal Science, St Lucia 4072, Queensland, Australia; ²The University of Queensland, School of Veterinary Science, Gatton 4343, Queensland, Australia; ³University of New England, School of Environmental and Rural Science, Armidale, New South Wales, 2351, Australia; ⁴The University of Queensland, School of Chemistry & Molecular Biosciences, St Lucia 4072, Queensland, Australia

Tritrichomonas foetus is a protozoan parasite that causes bovine trichomonosis, a venereal disease leading to early reproductive failure in cows while bulls remaining asymptomatic. Currently, there is no vaccine available in Australia. This study aimed to develop and evaluate the safety, efficacy and immunogenicity of a locally sourced *T. foetus* whole-cell killed vaccine in young bulls.

The TfOz5 Queensland *T. foetus* isolate was utilised as the vaccine strain, while the TfOz-N36 Northern Territory isolate was used as the challenge strain. The heat-inactivated vaccine, adjuvanted with Montanide ISA 61 VG, was administered subcutaneously to young *T. foetus*-naïve bulls (n=30) in two doses (5 x 10⁷ cells/dose) one month apart. Control bulls (n=30) received a mock adjuvant Phosphate-buffered saline. Bulls were experimentally infected intraprepuccially with live *T. foetus* cells at two and four weeks post-second vaccination.

The vaccine demonstrated safety, causing only mild local reactions, and no significant differences in weight and average daily gain between the two groups. Vaccinated bulls exhibited a significantly shorter average *T. foetus* infection duration compared to controls (7 vs. 16 days, *P* = 0.003). The vaccine stimulated high serum anti-*T. foetus* IgG antibodies which were boosted after each *T. foetus* challenge.

ID: 112 / CP6: 5

Contributed abstract

Conference Topics: Veterinary Parasitology

Keywords: Cyathostomin, equine, saliva, encysted larvae

Understanding the role of immune molecules in horse saliva during a small strongyle (cyathostomin) infection.

Tanya King, Leni Horner, Habtamu Derseh, Rob Bischof, David Piedrafita, Sarah Preston

Federation University, Australia

Small strongyles (cyathostomin) infection in horses can cause lethargy, weight loss, debilitation, and diarrhoea. The encysted stage of cyathostomin can enter a dormant state (hypobiosis) and are undetectable with standard faecal egg counts (FEC). While a commercial IgG(T) blood test can identify encysted larvae, its reliance on blood samples impedes its wide scale adoption. The aim of the current study was to investigate whether saliva can be used as an alternative to blood to detect antibodies against encysted and adult stage larvae.

Saliva, blood, and faeces were collected over 84 days from horses (n=27) in Victoria, Australia. Blood was processed for IgG(T) antibodies, FEC was measured using Mini-FLOTAC, and an IgA/IgG(T) ELISA was optimised for saliva. A significant correlation was found between saliva and blood IgG(T) against late encysted and adult larvae at Day 0 ($r=0.55$, $P=0.003$) and Day 70 ($r=0.47$, $P=0.029$). Saliva IgA correlated with blood IgG(T) for early encysted larvae at Day 70 ($r=0.51$, $P=0.014$). IgA levels increased with infection over time, though no correlation was observed with FEC.

Findings suggest antibodies detected in the commercial blood test are also present in saliva, highlighting its potential as a non-invasive alternative for diagnosing cyathostomin infections in horses.

CP4.1: Cell & Molecular Biology 5 min talks

Time: Tuesday, 01/July/2025: 2:30pm - 3:00pm · Location: Conference room 1

Session Chair: Jill Chmielewski, WEHI

Session Chair: Giel G van Dooren, Australian National University

ID: 153 / CP4.1: 1

Contributed abstract

Conference Topics: Apicomplexa Biology, Malaria, Molecular Biology

Keywords: Merozoite, Plasmodium knowlesi, CRISPR, Gene-editing, Blood-Stage

Investigating the importance of merozoite surface protein 3 in blood-stage growth of *Plasmodium knowlesi* parasites.

Keng Heng Lai^{1,2}, Isabelle Henshall^{1,2}, Jill Chmielewski^{1,2}, Danny Wilson^{1,2}

¹Research Centre for Infectious Diseases, School of Biological Sciences, The University of Adelaide, 5005, SA, Australia;

²Institute for Photonics and Advanced Sensing (IPAS), The University of Adelaide, 5005, SA, Australia

Nearly half of the world's population is at risk of malaria, with an estimated 263 million malaria cases in 2023 alone. The disease is caused by *Plasmodium* parasites, with *P. vivax* and *P. falciparum* being the major causes of morbidity and mortality. Our understanding of *P. vivax* vaccine candidate function and suitability falls significantly behind due to difficulties in culturing this parasite. The *in vitro* culturable *P. knowlesi*, which is closely related to *P. vivax*, provides an alternative model to characterise the function and antigenicity of vaccine candidates such as merozoite surface protein 3 (MSP3). Our studies on *P. knowlesi* MSP3 proteins found that all four *PkMSP3s* were individually dispensable, however attempts to remove all *PkMSP3s* in culture were unsuccessful, suggesting that at least one MSP3 is required for blood-stage growth. Using a transgenic line with three of the *PkMSP3s* (*PkMSP3I/G/B1*) knocked out, we were able to flank the remaining *PkMSP3B2* with LoxP sites, enabling inducible excision of the gene and protein functional knock-out which resulted in reduced parasite growth. These findings will help determine the function of *PkMSP3* genes in *P. knowlesi* and provide tools to investigate the suitability of MSP3s as vaccine candidates.

ID: 262 / CP4.1: 2

Contributed abstract

Conference Topics: Apicomplexa Biology, Biochemistry, Cell Biology

Keywords: Toxoplasma, mitochondria, glycolysis, metabolism

Interlinked metabolic pathways regulate central carbon metabolism in *Toxoplasma* parasites

Capella S. Maguire, F. Victor Makota, Giel G van Dooren

Research School of Biology, Australian National University, Canberra, ACT, Australia

Toxoplasma gondii is an apicomplexan parasite that causes severe disease in humans and livestock. *T. gondii* utilises carbon sources such as glucose and glutamine to generate energy and macromolecules required for its proliferation and survival. During an infection, *T. gondii* inhabits host organs with differing availabilities of carbon sources, but how the parasite adjusts its metabolism to account for these differences is poorly understood. The mitochondrial tricarboxylic acid (TCA) cycle is a key component of central carbon metabolism in *T. gondii*. We undertook CRISPR-based forward genetic screens to explore the importance of different metabolic pathways in parasites defective in the TCA cycle. Surprisingly, we found that fructose biphosphatase 2 (*TgFBP2*), an enzyme that is normally essential for parasite survival, became *dispensable* in parasites lacking the TCA cycle. *TgFBP2* has a key role in regulating the flux of metabolites through glycolysis, the pathway that catabolises glucose. Our data indicate that this regulation is no longer required when the TCA cycle is impaired. We hypothesise that TCA cycle impairment results in slowed flux of metabolites in glycolysis, overcoming the need for *TgFBP2* in this process. Our data highlight the sophisticated regulation of central carbon metabolism by interlinked metabolic pathways in these parasites.

ID: 126 / CP4.1: 3

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Malaria, Microscopy, Molecular Biology, Protozoa

Keywords: ExM, invasion, erythrocyte, blood, plasmodium

Ultrastructure expansion microscopy of the malaria parasite tight junction during blood-stage invasion

Dawson B Ling^{1,2}, **Cindy Evelyn**^{1,2}, **Aurelie T Dawson**^{1,2}, **Danushka S Marapana**^{1,2}, **Alan F Cowman**^{1,2}, **Niall D Geoghegan**^{1,2}, **Kelly L Rogers**^{1,2}

¹Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria, Australia; ²Department of Medical Biology, The University of Melbourne, Parkville, Victoria, Australia

Malaria caused 597,000 deaths in 2023, with 96% linked to the deadliest species, *Plasmodium falciparum*¹. Clinical malaria results from parasite invasion of erythrocytes, rendering it a promising candidate for therapeutic development. This process involves a series of complex and tightly coordinated events driven by ligands from the rhoptries and micronemes^{2,3}, the merozoite's apical secretory organelles. The merozoite uses these ligands to form a tight junction (TJ) with the erythrocyte, enabling stable attachment to remodel and invade the erythrocyte.

Despite recent advancements in our understanding of the TJ, much remains unclear, chiefly how the TJ interacts with the host erythrocyte membrane and cytoskeleton to facilitate invasion. This is owing to the intrinsically small nature of the merozoite (~1.5 µm in length)⁴, rhoptries (~600 nm in length)⁵, and the micronemes (~160 nm in length)⁶. Moreover, TJ formation is a fleeting process and relies on the RON complex being secreted from the parasite, which embeds on the erythrocyte surface as a receptor that binds AMA1 on the parasite surface². To overcome this dynamic and multi-component nature of TJ formation, we employed ultrastructure expansion microscopy of fluorescent parasites fixed at different phases of erythrocyte invasion, obtaining a high-resolution structural view of the TJ.

ID: 261 / CP4.1: 4

Contributed abstract

Conference Topics: Cell Biology, Malaria, Molecular Biology, Proteomics

Keywords: Plasmodium falciparum, gametocytes, RBPs, transmission, translational repression

Understanding the role of the RNA binding protein FD4 in malaria transmission

Qingqing Lin^{1,2}, **Sash Lopaticki**^{1,2}, **Mary-Lou Wilde**³, **Geoff McFadden**³, **James McCarthy**^{1,2}, **Mohini Shibu**^{1,2}, **Matthew Dixon**^{1,2}

¹Department of Infectious Diseases, Doherty Institute, University of Melbourne, Victoria.; ²Infection and Global Health Division, Walter and Eliza Hall Institute, Victoria.; ³School of BioSciences, University of Melbourne, Victoria.

The malaria-causing parasite *Plasmodium falciparum* relies on the formation of specialised sexual cells, known as gametocytes, for successful transmission. Male and female gametocytes develop in the human bloodstream and are ingested during a mosquito blood meal. Following ingestion, they rapidly differentiate into male microgametes and female macrogametes, which undergo fertilisation, initiating further development within the mosquito. This human-to-mosquito transmission represents a critical bottleneck in the parasite's lifecycle and is therefore an attractive target for therapeutic intervention. Previous studies have shown that translational repression operates during gametogenesis. This process involves the sequestration of mRNA transcripts by RNA-binding proteins (RBPs), preventing their translation into proteins until the precise time they are required. In this work, we will investigate the function of a putative RBP, Female Development 4 (FD4). Preliminary data indicate that deletion of FD4 leads to transmission failure and significant dysregulation of pathways essential for gamete differentiation and fertilisation. We aim to characterise the RNA-binding capacity of FD4 protein and define the biological programmes it regulates during this key developmental stage.

ID: 234 / CP4.1: 5

Contributed abstract

Conference Topics: Biochemistry, Malaria, Molecular Biology

Keywords: malaria, nanobody

Nanobodies to inhibit malaria parasite fertilization and development in mosquitoes

Chunyi Qian^{1,2}, **Li Lynn Tan**^{1,2}, **Melanie Dietrich**^{1,2}, **Wai-Hong Tham**^{1,2}

¹Walter and Eliza Hall Institute of Medical Research, Australia; ²Department of Medical Biology, The University of Melbourne, Parkville, Victoria, Australia

Malaria is a major disease in humans with over 600,000 deaths each year, with *Plasmodium falciparum*, being responsible for almost all deaths. *P. falciparum* has a complex life cycle involving the female *Anopheles* mosquito and a vertebrate host, the human. Targeting sexual stages of malaria parasites (gametocytes, gametes, ookinetes) is advantageous compared to other life cycle stages, as the development of sexual forms entails bottlenecks in terms of parasite numbers.

For malaria parasites, fertilisation occurs in the female *Anopheles* mosquito. Successful fertilisation results in the maturation of parasites within the mosquito, which are then transmitted to humans via the bite of an infected mosquito. By stopping parasite fertilisation and parasite development in the mosquito, we can stop the transmission of the malaria parasites from mosquito to human.

Pfs47 is expressed on female gametocytes, zygotes and ookinetes. Through its interaction with a specific mosquito midgut receptor protein, Pfs47 is involved in a lock and key model that drives host tropism between parasite and mosquito. PfsPSOP12 is expressed in the ookinete and gametocyte stages and important for success development of malaria parasites in the mosquito. We aim to investigate whether nanobodies against Pfs47 and PfsPSOP12 can block transmission of the malaria parasites.

CP5.1: Epidemiology 5 min talks

Time: Tuesday, 01/July/2025: 2:30pm - 3:00pm · Location: Conference room 2

Session Chair: Shannon M Hedtke, La Trobe University

Session Chair: Kirsty M McCann, Deakin University

ID: 111 / CP5.1: 1

Contributed abstract

Conference Topics: Genomics, Malaria

Keywords: Plasmodium falciparum, Plasmodium malariae, Plasmodium ovale spp, malaria, genome complexity, West Africa

Metagenomic analysis reveals extreme complexity of *Plasmodium* spp. in asymptomatic infections in high, seasonal transmission in Northern Sahelian Ghana

Mun Hua Tan¹, Oscar Bangre², Cecilia Rios-Teran¹, Kathryn Tiedje¹, Samantha Deed¹, Qi Zhan³, Fathia Rasyidi¹, Mercedes Pascual⁴, Patrick Ansa², Karen Day¹

¹Department of Microbiology and Immunology, Bio21 Institute and The Peter Doherty Institute for Infection and Immunity, The University of Melbourne, VIC, Australia; ²Navrongo Health Research Centre, Ghana Health Service, Navrongo, Ghana;

³Committee on Genetics, Genomics and Systems Biology, The University of Chicago, Chicago, Illinois, USA; ⁴Department of Biology, New York University, New York, NY, USA

Mixed-species and mixed-strain *Plasmodium* infections are well-documented in malaria-endemic regions, yet the full extent of their complexity has not been systematically explored, particularly in high-burden countries of sub-Saharan Africa. This is especially critical in the asymptomatic reservoir across all host age groups, which sustains transmission. Here, we take a metagenomic lens to investigate the diversity and complexity of infections in afebrile individuals living in Northern Sahelian Ghana, where malaria transmission is seasonal but intense. By analysing infection complexity obtained by sampling variable blood volumes, we reveal a significantly higher prevalence of *Plasmodium* spp. and increased intra- and inter-species complexity in larger blood volumes. Our findings demonstrate a high degree of metagenomic complexity, with infections comprising single, double, and triple *Plasmodium* species, including *P. falciparum*, *P. malariae*, *P. ovale curtisi*, and *P. ovale wallikeri*, with varying levels of complexity. Notably, we identified a subset of individuals with highly-complex infections that cannot be explained by age or geographic location. The implications of these findings to malaria epidemiology and control are illustrated by a geographic scaling exercise to district and region levels in Ghana.

ID: 247 / CP5.1: 2

Contributed abstract

Conference Topics: Bioinformatics, Epidemiology, Genomics, Host-parasite interactions

Keywords: Malaria, Genomic epidemiology, Transmission, Immunity

Identifying sources of *Plasmodium falciparum* infection resurgence in low transmission

Sonakshi Madan¹, Kirsty McCann^{1,2}, Zahra Razook^{1,2}, Dulcie Lautu-Gumal^{1,2,3,4}, Benson Kiniboro⁵, Peter M. Siba⁵, Stephan Karl⁶, Maria Ome-Kaius⁵, Moses Laman⁵, James W. Kazura⁷, Shazia Ruybal-Pesántez^{8,9}, Ivo Mueller^{3,4}, Leanne J. Robinson^{2,3,5}, Alyssa E. Barry^{1,2,3}

¹Centre for Innovation in Infectious Disease and Immunology Research, Institute for Mental and Physical Health and Clinical Treatment (IMPACT), School of Medicine, Deakin University, Geelong, Victoria, AUSTRALIA; ²Life Sciences Discipline, Burnet Institute, Melbourne, Victoria, AUSTRALIA; ³Population Health and Immunity Division, Walter and Eliza Hall Institute, Parkville, Victoria, AUSTRALIA; ⁴Department of Medical Biology, University of Melbourne, Parkville, Victoria, AUSTRALIA; ⁵Vector Borne Diseases Unit, Papua New Guinea Institute of Medical Research, Madang, PAPUA NEW GUINEA; ⁶Australian Institute of Health and Tropical Medicine, James Cook University, Cairns, Queensland, AUSTRALIA; ⁷Center for Global Health and Diseases, Case Western Reserve University, Cleveland, Ohio, USA; ⁸MRC Centre for Global Infectious Disease Analysis, School of Public Health, Imperial College London, UK; ⁹Instituto de Microbiología, Universidad San Francisco de Quito, Ecuador

Despite major progress in reducing malaria transmission, elimination remains challenged by hidden asymptomatic infections and risk of resurgence via imported cases. With declining transmission and exposure to fewer, perhaps only local strains; host immunity decreases, potentially increasing susceptibility to symptomatic infection by introduced strains. However, low-transmission regions are exhibiting more asymptomatic infections which act as undetected reservoirs for ongoing transmission. The relationship between declining transmission, host immunity, and parasite diversity in shaping infection outcome remains unclear. Papua New Guinea presents an opportunity to analyse population structure and immunity during declining transmission (2012) followed by malaria resurgence (2016). Through cross-sectional surveys, we are characterising genetic diversity of *Plasmodium falciparum* strains to determine whether genetically distinct, potentially imported strains correlate with symptomatic malaria, contributing to resurgence. Using SNP barcoding and immune evasion markers, *P. falciparum* infections were genotyped to analyse parasite relatedness and population structure. Pairwise relatedness, clustering, phylogenetic trees, and nucleotide diversity are being used to identify infection origin, associations between strain diversity and symptoms, and how they vary with changing transmission levels. Addressing this critical knowledge gap will pave the way for better outbreak predictions, targeted interventions and more effective case management for malaria elimination in endemic regions.

ID: 110 / CP5.1: 3

Contributed abstract

Conference Topics: Epidemiology, Immunology, Malaria

Keywords: Bayesian statistics, malaria, immunology, antibodies

Estimating time of infection based on serological data to identify *Plasmodium vivax* hypnozoite carriers

Felicia Bongiovanni^{1,3}, Eamon Conway^{1,3}, Jodie McVernon^{2,3}, Ivo Mueller^{1,3}

¹The Walter and Eliza Hall Institute of Medical Research, Australia; ²The Peter Doherty Institute for Infection and Immunity, Australia; ³The University of Melbourne, Australia

Plasmodium vivax presents a major challenge for malaria elimination in the Asia-Pacific and causes a significant public health burden. During the liver stage of the *P. vivax* life cycle, parasites can become dormant hypnozoites that can reactivate months

to years later and cause a blood-stage infection. These hypnozoites cannot be detected by traditional diagnostic methods and are responsible for up to 80% of active *vivax* malaria infections.

Infection with *P. vivax* induces short-lived and long-lived antibody responses. Understanding the underlying mechanisms behind antibody production not only helps us learn more about our immunity to *P. vivax*, but can be used to identify likely hypnozoite carriers. Research has shown that individuals infected in the last 9 months may be harbouring silent hypnozoites. This work presents a mathematical approach to utilise serological data to understand more about the humoral immune response and identify the time of infection for an individual. Using a biphasic antibody decay model and Bayesian statistics, we address the diagnostic gap for identifying hypnozoite infections to lead to better treatment of *P. vivax* and assist in the eradication of malaria.

ID: 175 / CP5.1: 4

Contributed abstract

Conference Topics: Protozoa

Keywords: Cryptosporidium, outbreaks, molecular typing, gp60

A preliminary investigation on a recent human *Cryptosporidium* outbreak in Perth, Western Australia

Sugandika Bulumulla¹, Amanda.D Barbosa^{1,2}, Amanda Ash¹, Una Ryan¹

¹Harry Butler Institute, Murdoch University, Australia; ²CAPEs Foundation, Ministry of Education of Brazil, Brasilia

Cryptosporidium is a leading cause of moderate-to-severe, unrelenting diarrhoea in humans, particularly in low-income countries (Ryan et al., 2016). The environmental stage of *Cryptosporidium* (oocysts), are transmitted directly by faecal-oral contamination or by ingestion of food or water contaminated with oocysts (Ryan et al., 2021). These oocysts are robust and resistant to chlorine disinfection (Ryan et al., 2021) and are a major cause of waterborne outbreaks in Australia and worldwide (Braima et al., 2021; Bourli et al., 2023). Currently more than 23 species have been reported in humans but two species; *C. parvum* and *C. hominis* are responsible for ~95% of human infections (Yang et al., 2021; Guo et al., 2022). Outbreak investigations using molecular typing tools are crucial to better understand infection sources and transmission dynamics and implement improved control strategies. The most widely used subtyping tool for *Cryptosporidium* is based on sequence analysis of the hypervariable 60 kDa glycoprotein (gp60) gene ((Ryan et al., 2021). Preliminary analysis of gp60 typing data from a cryptosporidiosis outbreak in March-April 2025, in Perth, WA, will be presented.

ID: 197 / CP5.1: 5

Contributed abstract

Conference Topics: Malaria

Keywords: Anopheles, malaria, salivary antigen, antibody biomarker, serosurveillance

Novel anti-*Anopheles* salivary antibody biomarkers for malaria serosurveillance in the Asia-Pacific region

Asleigh S. Heng-Chin^{1,2,3,4}, Ellen A. Kearney^{1,3,4}, Mei Hawe¹, Katherine O'Flaherty^{1,3,4}, James G. Beeson^{1,3,5,6,7}, Freya J. I. Fowkes^{1,2,3,4}, Australian Centre of Research Excellence in Malaria Elimination Investigators³

¹Burnet Institute, Melbourne, Australia; ²Department of Epidemiology and Preventative Medicine, Monash University, Melbourne, Australia; ³Australian Centre of Research Excellence in Malaria Elimination, The University of Melbourne, Melbourne, Australia; ⁴Centre for Epidemiology and Biostatistics, The University of Melbourne, Melbourne, Australia; ⁵Department of Immunology, Monash University, Melbourne, Australia; ⁶Department of Medicine, The University of Melbourne, Melbourne, Australia; ⁷Department of Microbiology and Immunology, The Peter Doherty Institute for Infection and Immunity, The University of Melbourne, Melbourne, Australia

To progress towards malaria elimination, sensitive and scalable surveillance tools are required to monitor human interactions with *Anopheles* mosquito vectors. Current methods of entomological surveillance are logistically challenging and only provide population-level estimates of mosquito human biting rates (HBR) that cannot capture heterogeneous transmission. Measurement of antibodies against *Anopheles* salivary proteins injected during a bite could provide a proxy measure of individual-level bite exposure. While measurement of antibodies against the leading candidate antigen from the African vector *An. gambiae* has been shown a reliable marker of biting in Africa, inconsistent correlations between seroprevalence and HBR were found in the Asia-Pacific, where 19 dominant vector species coexist. Currently, there are no identified anti-salivary antibody biomarkers of Asian-Pacific *Anopheles* bites.

To address this, we measured IgG responses against salivary peptides from 6 leading Asian-Pacific vectors (*An. culicifacies*, *An. dirus*, *An. farauti*, *An. minimus*, *An. maculatus*, and *An. stephensi*) in samples collected from Bangladesh, Myanmar, Laos, Cambodia, Malaysia, Papua New Guinea, and Kenya. All antigens elicited IgG responses, which varied significantly across study sites ($p < 0.01$), therefore suggesting their potential utility as biomarkers of Asian-Pacific *Anopheles* bites. Further investigation of these candidates may improve vector serosurveillance and aid malaria elimination efforts in the Asia-Pacific.

ID: 299 / CP5.1: 6

Contributed abstract

Conference Topics: One Health, Veterinary Parasitology, Wildlife parasitology

Keywords: Gastrointestinal Parasites, Foxes

Gastrointestinal Parasites of Foxes around Melbourne

Bridget Graffeo, Charles G. Gauci, Leonardo Brustenga, Tharaka Liyanage, Kabir Brar, Ian Beveridge, Jasmin Hufschmid, Abdul Jabbar

Melbourne Veterinary School, Werribee, Victoria 3030, Australia

The red fox (*Vulpes vulpes*) is an introduced species to Australia whose population and spatial dispersal have grown irreversibly. Due to their opportunistic feeding habits, extensive populations of foxes now inhabit urban and peri-urban environments, where they coexist with humans and domesticated animals. The proximity of these predators presents public and animal health concerns as they harbour diseases that can cross between species. Accordingly, monitoring potential disease risk and prevalence in urban foxes is warranted. This study aims to investigate the occurrence of gastrointestinal

parasites in foxes around Melbourne. The gastrointestinal tracts of 52 foxes collected in the greater Melbourne area were washed thoroughly to collect adult worms present in the stomach and the small and large intestines. Current results showed that 92.3% of foxes were positive for at least one gastrointestinal helminth infection. Based on a gross examination of worms, the detected nematode parasites were *Toxocara canis*, *Uncinaria stenocephala* and *Trichuris vulpis*, while the identified tapeworms included *Dipylidium caninum*, *Spirometra* sp. and *Taenia* spp. This study is likely to highlight a significant human and animal health risk, as a crossover of parasitic diseases is possible in areas where these diseases coexist.

CP6.1: Livestock Parasites 5 min talks sponsored by Elanco

Time: Tuesday, 01/July/2025: 2:45pm - 3:00pm · Location: Conference room 3

sponsored by Elanco and chaired by Liisa Ahlstrom & Monica Commons Elanco

ID: 242 / CP6.1: 1

Contributed abstract

Conference Topics: Epidemiology, Helminthology, Veterinary Parasitology

Keywords: Dairy goats, gastrointestinal nematodes, prevalence, faecal egg counts, Australia

Epidemiology of strongylid nematodes in Australian dairy goats

Endris Ali¹, Abdul Ghafar¹, Ghazanfar Abbas¹, Mark A. Stevenson¹, Sandra Baxendell², Charles G. Gauci¹, Elysia Ling¹, Ian Beveridge¹, Abdul Jabbar¹

¹University of Melbourne, Australia; ²Goat Veterinary Consultancies- goatvetoz, Brisbane, Queensland, Australia

Gastrointestinal nematodes (GINs) pose major health, production and welfare concerns for the global goat industry, resulting in significant economic losses. However, knowledge regarding the prevalence and intensity of GINs in Australian dairy goats remains limited. This study determined the prevalence and intensity of GINs in Australian dairy goats. Between November 2023 and July 2024, 1,028 faecal samples collected from 68 dairy goat farms were analysed using the modified McMaster technique. Eggs per gram (EPG) of faeces were determined for strongyles, *Nematodirus* spp. and *Trichuris* spp. Overall, the prevalence of GINs was 90.5%, with the highest prevalence for strongyles (88.0%) followed by *Trichuris* spp. (13.8%) and *Nematodirus* spp. (7.3%). Adult goats had the highest prevalence of strongyles (92.6%) followed by weaners (86.4%) and kids (73.1%). The mean EPG was highest for strongyles (1,085) followed by *Trichuris* spp. (16) and *Nematodirus* spp. (6). Adults had a higher mean EPG of strongyles (1,255) than weaners (798) and kids (701). *Haemonchus contortus*, *Teladorsagia circumcincta* and *Trichostrongylus* spp. were the main GINs infecting Australian dairy goats. The high prevalence of GINs among Australian dairy goats poses potential production losses. These findings underscore the need for national efforts to achieve sustainable GINs control in grazing ruminants.

ID: 191 / CP6.1: 2

Contributed abstract

Conference Topics: Diagnostics, Livestock Parasites, Veterinary Parasitology

Keywords: *Haemonchus contortus*, diagnostic, infection surveillance, Loop-mediated Isothermal Amplification (LAMP)

Comparison of different diagnostic methods to measure *Haemonchus contortus* infection for surveillance across Victoria

Rebecca Farnell¹, David Piedrafita¹, Andrew Greenhill¹, Christiane Bahlo¹, Steve Cotton², Sarah Preston¹

¹Federation University Australia; ²Dynamic Ag Pty Ltd

Gastrointestinal nematode infections are the second most important endemic disease impacting sheep in Australia, and there are increasing concerns of *Haemonchus contortus* infections becoming endemic in Victoria. Surveillance must be completed to track these infections; however, many sheep owners are not testing due to costs, time and labour. The current industry standard test is larval culture, which takes 1-2 weeks, and requires a trained specialist. This is an issue as *H. contortus* infection can cause death quickly, so rapid diagnostics would be valuable.

This research compared colorimetric loop-mediated isothermal amplification (LAMP), Nemabiome deep-amplicon sequencing and larval culture (for samples submitted from a consulting company; n=27). LAMP and larval culture agreed in 74% of the samples (n=20), and LAMP and Nemabiome deep-amplicon sequencing agreed in 70% of these submitted samples (n=19). For a collection of samples collected from local saleyards, LAMP and Nemabiome deep-amplicon sequencing had 66% agreement (n=41). The disagreement between these results is being further evaluated. This research will enable investigation and surveillance of the current distribution of *H. contortus* infections throughout Victoria under changing climate conditions. Furthermore, the colourimetric LAMP shows to be a promising, rapid diagnostic, helping improving animal welfare with infection surveillance.

ID: 162 / CP6.1: 3

Contributed abstract

Conference Topics: Drugs, Livestock Parasites, Veterinary Parasitology

Keywords: Anthelmintic Resistance, Levamisole, Informed Drenching

Screening for Levamisole Resistance in *Teladorsagia circumcincta* and *Trichostrongylus colubriformis* from Sheep across New South Wales

Olivia Kelly¹, Emily Francis¹, Jan Slapeta¹, Janina McKay²

¹University of Sydney, Australia; ²Department of Primary Industries, New South Wales

Anthelmintic resistance in major parasitic nematodes *Teladorsagia circumcincta* and *Trichostrongylus colubriformis* encompasses a substantial economic and welfare concern for the Australian sheep industry, with estimated production losses exceeding \$500 million annually. Although molecular diagnostics for levamisole resistance detection has been established in *Haemonchus*, its application to *Trichostrongylus* and *Teladorsagia* remains unexplored. This study aims to discover the prevalence of levamisole resistance-associated single nucleotide polymorphism S168T, in *T. circumcincta* and *T. colubriformis*

populations across New South Wales. Archived larval field samples collected between 2020 and 2024 will be analysed using mixed amplicon metabarcoding targeting the *acr-8* gene region, to quantify resistance allele frequencies. Furthermore, spatial analyses will be conducted to visualise the NSW geographic distribution in SNP prevalence. We hypothesise that resistance-associated alleles will be prevalent (~ 80%) in both species, indicative of ongoing selection pressure since the last nationwide field survey based on phenotypic data from FECRTs. The research outcomes will provide critical insights into the contemporary status of levamisole resistance in *T. circumcincta* and *T. colubriformis*, supporting the development of evidence-based approaches for sustainable parasite control and resistance management on Australian sheep farms.

CP7: Omics 15 min talks

Time: Tuesday, 01/July/2025: 3:30pm - 4:45pm · *Location:* Conference room 1

Session Chair: Ghizal Siddiqui, Monash University

Session Chair: Balu Balan, Walter and Eliza Hall Institute

ID: 266 / CP7: 1

Contributed abstract

Conference Topics: Bioinformatics, Ectoparasites

Keywords: Ixodes ricinus, salivary glands, ovarian tissue, blood-feeding, ectoparasites

Transcriptomic Blueprint of ectoparasitic tick *Ixodes ricinus*

Amrita Vijay¹, Balu Balan¹, Louise Baker¹, Stefano Gaiarsa², Quentin Gouil³, Pradip Roy⁴, Alexander Gofton⁵, Alessandra Cafiso⁶, Ala Tabor⁷, Nathan Lo⁸, Olivier Plantard⁹, Jan Riemer¹⁰, Fabrizia Stavru¹¹, Clare A Anstead⁴, Peter Czabotar¹, Anthony Papenfuss¹, Davide Sasser^{12,13}, Aaron R Jex^{1,4}

¹WEHI, Australia; ²Department of Microbiology & Virology, Fondazione IRCCS Policlinico San Matteo, Pavia, Italy; ³Olivia Newton-John Cancer Research Institute, Australia; ⁴Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Victoria, Australia; ⁵CSIRO, Health and Biosecurity, Brisbane, Queensland, Australia; ⁶Department of Veterinary Medicine and Animal Sciences, University of Milan, Lodi, Italy; ⁷The University of Queensland, Queensland Alliance for Agriculture & Food Innovation, St Lucia, Queensland, Australia; ⁸School of Life and Environmental Sciences, The University of Sydney, New South Wales; ⁹Oniris, INRAE, BIOEPAR, 44300, Nantes, France; ¹⁰Department for Chemistry, Institute for Biochemistry, University of Cologne, Cologne, Germany; ¹¹Unité de Biologie Evolutive de la Cellule Microbienne, Institut Pasteur, Paris, France; ¹²Department of Biology and Biotechnology, University of Pavia, Pavia, Italy.; ¹³Fondazione IRCCS Policlinico San Matteo, Pavia, Italy

Tick-borne diseases present significant public health challenges in Europe, with the European castor bean tick, *Ixodes ricinus*, serving as a principal vector for Lyme borreliosis and tick-borne encephalitis. In this study, we generated a high-quality hybrid transcriptome assembly comprising 21,954 predicted transcripts, including 19,221 full-length open reading frames. High-confidence annotations of both 5' and 3' untranslated regions, along with alternatively spliced isoforms, were achieved using long-read RNA sequencing, providing an in-depth view of the genetic architecture underlying key biological processes such as chemosensation, hematophagy, and immune tolerance. Tissue-specific transcriptomic analyses of salivary glands and ovarian tissues were performed across distinct feeding phases: early, mid-feeding, and late-feeding. In the salivary glands, dynamic metabolic re-wiring, vesicle biogenesis, and secretome alterations were identified, facilitating anti-clotting, vasodilatory, anti-inflammatory, and immunomodulatory functions to overcome host defences. Complementary analysis of ovarian tissue revealed early transcriptomic reprogramming in cell fate determination, cytoskeletal organization, extracellular matrix dynamics, and immune regulation, potentially priming the ovary for fertilization and embryogenesis. Furthermore, our findings offer a valuable resource for future studies on tick molecular biology and the development of novel control strategies. This integrated approach expands our knowledge of tick ectoparasitism and supports the design of improved strategies for disease prevention.

ID: 173 / CP7: 2

Contributed abstract

Conference Topics: Genomics

Keywords: Ghana, Blackfly, Onchocerciasis, Genome Assembly, MOTUs

Genome Assembly of *Simulium damnosum* from Ghana: Implications for Onchocerciasis Transmission Dynamics

Millicent Opoku^{1,2}, Neha Sirwani¹, Emily N. Hendrickson^{1,5}, Kwadwo K. Frempong², Sedou Naniogue⁴, Sampson K. Otoo², Philomina Jackson², Franklin Ayisi³, Joseph H. N. Osei⁶, Sellase Pi-Bansa², Sindew M. Feleke^{1,7}, Daniel A. Boakye^{2,4}, Warwick Grant¹, Shannon Hedtke¹

¹Department of Ecological, Plant and Animal Sciences, La Trobe University, Australia; ²Department of Parasitology, Noguchi Memorial Institute for Medical Research, University of Ghana, Ghana; ³African Regional Graduate Programme in Insect Sciences, University of Ghana, Accra, Greater Accra, Ghana; ⁴The END Fund, New York, USA.; ⁵University of California, San Diego, USA.; ⁶Biomedical and Public Health Research Unit, Water Research Institute, Council for Scientific and Industrial Research, Ghana.; ⁷Ethiopian Public Health Institute, Addis Ababa, Ethiopia.

Human onchocerciasis, or river blindness, is caused by the nematode *Onchocerca volvulus*, which is transmitted by blood-sucking blackflies of the genus *Simulium*. Despite their medical importance, we lack molecular data characterizing *Simulium* species. Current classification is based largely on cytotoxicology, which is dependent on expert identification of chromosomal banding patterns in the larval silk glands, and poor adult morphological differentiation further complicates taxonomic efforts. Knowledge of species dispersal ranges, critical for understanding transmission dynamics, remains limited.

We present the first nearly complete (90.5%) genome assembly of an adult *Simulium damnosum* collected from Ghana. We used Oxford Nanopore long-read sequencing (2,915,146 million reads) and Illumina short-read sequencing (16,385,843 paired-end 2x150bp) to assemble the genome, which is ~260 million bp with 19,186 predicted genes. This was used as a reference for population genomics of 273 adult blackflies from 21 sites across Ghana. We identified three molecular operational taxonomic units (MOTUs), with further differentiation of one into two sub-MOTUs.

This genome assembly enabled us to identify the geographic distribution of genetically distinct blackflies, which we will incorporate into vector dispersal and transmission dynamics. Future improvements, including integrating Hi-C data for haplotype phasing, will enhance understanding of *Simulium* chromosomal evolution and speciation.

ID: 248 / CP7: 3

Contributed abstract

Conference Topics: Bioinformatics, Ectoparasites, Genomics, Livestock Parasites, Veterinary Parasitology

Keywords: Australian sheep blowfly, flystrike, chromosomal-level genome assembly, transcriptomics, insecticide resistance

The chromosomal level assembly of the Australian sheep blowfly, *Lucilia cuprina dorsalis* genome using third-generation DNA sequencing and Omni-C analysis

Shilpa Kapoor^{1,2}, Amrita Vijay¹, Balu Balan¹, Louise Baker¹, Laura Wines³, Vernon M. Bowles², Aaron R. Jex^{1,2}, Clare A. Anstead²

¹Infection and Global Health, Walter and Eliza Hall Institute of Medical Research, Parkville, VIC, Australia; ²Department of Veterinary Biosciences, Faculty of Science, The University of Melbourne, Parkville, VIC, Australia; ³School of Biosciences, Faculty of Science, The University of Melbourne, Parkville, VIC, Australia

The Australian sheep blowfly, *Lucilia cuprina dorsalis*, is a significant ectoparasite of sheep responsible for subcutaneous myiasis (flystrike) leading to production losses, injury or mortality. Current flystrike control management strategies predominantly rely on breech modification surgery (mulesing) and insecticide application; however, the widespread and accelerating development of insecticide resistance poses a major challenge. Despite this, the emergence and dissemination of resistance-associated alleles within Australian *L. c. dorsalis* populations remain poorly understood. To address this knowledge gap, we employed an integrated genomics approach, combining Oxford Nanopore Technologies (ONT) and Illumina sequencing with Omni-C proximity ligation technology, to achieve a chromosomal-level assembly of this important pest. Additionally, comprehensive long- and short-read RNA sequencing was utilized to construct a high-resolution *de novo* transcriptome. This multi-omics approach provides valuable insights into the genetic architecture, evolutionary history, and molecular mechanisms underlying key biological processes in *L. c. dorsalis*. Furthermore, the chromosomal-level genome assembly enables the investigation of genetic variation within blowfly populations across Australia, facilitating the tracking and monitoring of resistance allele emergence and dissemination, which is essential for developing sustainable control strategies.

ID: 284 / CP7: 4

Contributed abstract

Conference Topics: Epidemiology, Genomics

Keywords: genomics, Simulium, onchocerciasis, epidemiology, transmission

The genomes of African *Simulium* spp.: investigating onchocerciasis transmission zone delineation and epidemiology using vector population genomics.

Warwick Grant, Emily Hendrickson, Himlal Shrestha, Sindew Feleke, Millicent Opoku, Shannon Hedtke

La Trobe University, Australia

Blackflies of the genus *Simulium* are the vectors for the filarial parasite *Onchocerca volvulus*, the causative organism of onchocerciasis. The spatial pattern of transmission of *O. volvulus* is determined by the spatial pattern of vector (and infected host) movement but mass drug administration (MDAi) rarely takes this into account. In northwest Ethiopia, MDAi interrupted transmission in the Metema “sub-focus” and MDAi ceased in 2018. Recent post-treatment surveillance (based on detecting *O. volvulus* in blackflies and serology in children) revealed that transmission has resumed. We hypothesise that vector movement from neighbouring districts with ongoing transmission is the source of renewed transmission. Genomic analyses of blackflies from the Metema sub-focus and the neighbouring Metekel focus to the south showed high levels of blackfly genetic connectivity between the these foci, implying they belong to a single geographically large transmission zone. Landscape genetic modelling identified habitat corridors connecting the two foci that could facilitate blackfly movement, explain the genetic connectivity and support transmission. We conclude that vector movement from the south could have contributed to renewed transmission in the Metema sub-focus. We recommend that decisions to stop MDAi should use population genomic analysis to determine the “natural” boundaries of *O. volvulus* transmission zones.

ID: 118 / CP7: 5

Contributed abstract

Conference Topics: Apicomplexa Biology, Genomics, Malaria

Keywords: bromodomains, development, chromatin, epigenetics, acetylation

Epigenetic regulation of adaptation and differentiation in malaria parasites.

Michael Duffy¹, Lee M. Yeoh², Jingyi Tang³, Christopher Goodman⁴, Hannah Nguyen⁵, Myriam Grotz⁶, Scott Chisholm⁷, Kapil Pareek⁸, Suffian Azizan⁴, Chunhao Yu⁵, Anton Cozijnsen⁴, Karen Day¹, Geoffrey MacFadden⁴, Paul Gilson², Danae Schulz⁸, Tania De Koning-Ward³, Michaela Petter⁶

¹Department of Microbiology and Immunology, University of Melbourne, Australia; ²Burnet Institute, Melbourne, Australia;

³Deakin University; ⁴School of BioSciences, University of Melbourne; ⁵University of Melbourne; ⁶Erlangen University;

⁷Cambridge University; ⁸Harvey Mudd College

The malaria parasite *Plasmodium falciparum* derepresses heterochromatic genes for 1) initiating developmental transitions and 2) for environmental adaptation by generating clonally variant phenotypes through expression switching between genes. These epigenetic processes are regulated by chromatin proteins including histones, the enzymes that modify them and the bromodomain proteins that bind them. We have shown that a suite of bromodomain proteins are essential for survival of multiple lifecycle stages of parasites and regulate differentiation and adaptation. One of them, the histone acetyl transferase PfGCN5 is critical to these processes and maintains a dynamic heterochromatin boundary through acetylation of the variant histone Pf H2B.Z. Acetylated Pf H2B.Z activates gene promoters and is antagonised by the silencing protein and histone deacetylase PfSir2A at heterochromatin boundaries. This fundamental biology is essential to the parasites survival,

pathogenesis and transmission and involves three classes of proteins that are being pursued as drug targets in human disease, histone acetyl transferases, histone deacetylases and bromodomain proteins.

CP8: Arthropod Parasites & Vectors 15 min talks

Time: Tuesday, 01/July/2025: 3:30pm - 4:45pm · *Location:* Conference room 2

Session Chair: Charlotte Oskam, Murdoch University

Session Chair: Vern Bowles, The University of Melbourne

ID: 150 / CP8: 1

Contributed abstract

Conference Topics: Diagnostics, Parasites of companion animals, Parasites of dogs

Keywords: Metabarcoding, Oxford Nanopore Technologies, *Dirofilaria*, Haemoparasites

Nanopore metabarcoding for vector-borne pathogen detection, surveillance and characterisation of novel species

Lucas Huggins¹, Ushani Atapattu¹, Ugyen Namgyel², Virak Khieu³, Vito Colella¹

¹Veterinary Preclinical Sciences Building, University of Melbourne, Parkville, Victoria 3052, Australia; ²National Centre for Animal Health, Serbithang, Thimphu 11001, Bhutan; ³National Centre for Parasitology, Entomology and Malaria Control, Ministry of Health, Phnom Penh, Cambodia

Humans and animals are afflicted by diverse vector-borne pathogens (VBP) of which many can cause severe disease and be fatal. Accurate diagnosis of parasitic infections can be challenging due to intermittent parasitaemia, frequent coinfections and the wide range of rare, emerging, and novel VBP species encounterable. Nonetheless, advanced metabarcoding techniques can circumvent these challenges by detecting and characterising entire groups of parasites simultaneously, including those that are unexpected, cryptic and as yet undescribed. Through a series of case studies, we demonstrate how our recently developed metabarcoding assays conducted on the Oxford Nanopore Technologies (ONT) sequencing platform can be used to comprehensively elucidate VBPs such as bacteria, apicomplexans, kinetoplastids and filarial worms. We show how are VBP-targeting assays have assisted in characterising novel pathogens such as *Dirofilaria asiatica*, an *Ehrlichia chaffeensis*-like organism and haemotropic *Mycoplasma* spp. from companion animals in Bhutan, Cambodia, Mongolia and Sri Lanka that pose a significant risk to co-habiting human health. We envision that as these versatile deep-sequencing approaches become more cost-effective and user-friendly that they will deepen our understanding of parasite diversity and highlight novel methods through which they can be effectively controlled.

ID: 207 / CP8: 2

Contributed abstract

Conference Topics: Ectoparasites

Keywords: Scabies, Microbiota, Treatment

A novel topical scabicide demonstrates beneficial effects on the porcine microbiota after the clearance of crusted scabies

Sara Taylor, Martha Zakrzewski, Deepani D Fernando, Gangi R Samarawickrama, Nirupama A Nammunige, Katja Fischer

Infection and Inflammation Program, QIMR Berghofer Medical Research Institute, Brisbane, Australia

Scabies is a highly contagious infectious skin disease with an estimated prevalence of ~200 million cases annually. It is a neglected tropical disease and linked with secondary bacterial infections causing a substantial public health burden. Current treatment for this condition relies heavily on two broad-spectrum anti-parasitic agents (permethrin and ivermectin), that require repeated doses to be effective. Our research has demonstrated the *in vitro* efficacy of two novel compounds against *Sarcoptes scabiei* mites and eggs, anti-microbial properties against *Staphylococcus aureus* and *Streptococcus pyogenes*, and efficacy *in vivo* in a porcine scabies model. Throughout the preclinical trial, skin scrapings were collected and sequenced using 16s full-length rRNA amplicon sequencing, to determine the compositional changes in the skin microbiota over the course of infection and treatment. Across 190 samples 9931 amplicon sequence variants (ASVs) were identified. There was a significant effect of treatment on the microbial community composition and the combination treatment prevented colonisation with known pig skin pathogens. This research demonstrates the usefulness of a scabicide with anti-microbial properties in preventing over-colonisation with opportunistic pathogens.

ID: 271 / CP8: 3

Contributed abstract

Conference Topics: Ecology, Ectoparasites, Molecular Biology

Keywords: ticks, population ecology, microbiome, land-use, climate

Ticky Business: Integrating Population Genetics, Environment and Microbiome to Inform Tick Management Strategies

Xavier Barton¹, Joseph Fontaine², Shanan Tobe¹, Siobhon Egan¹, Charlotte Oskam¹

¹School of Medical, Molecular and Forensic Sciences, College of Environmental and Life Sciences, Murdoch University;

²School of Environmental and Conservation Sciences, College of Environmental and Life Sciences, Murdoch University

Ticks pose a global threat through pathogen transmission to humans, companion animals, and livestock. We investigated the population ecology of *Amblyomma triguttatum* (ornate kangaroo tick), Western Australia's most common human-biting tick, to understand implications for exotic tick and pathogen incursions. Using 379 *A. triguttatum* specimens, we employed three complementary approaches: (1) population structure analysis using ddRADseq, (2) examination of environmental drivers, and (3) bacterial community assessment across the landscape.

Results reveal distinct population structure, with major divisions between populations north and south of the Swan River and Perth city. Environmental factors including land use, soil type, climate, fire history, and water catchments were evaluated as predictors of observed structure. Preliminary microbiome analyses suggest complex relationships that may not directly correspond with the observed population structure, potentially complicating targeted control efforts.

Our research provides crucial information for how terrestrial pathogen vectors move around the landscape, how this interacts with land use and how composition of the microbiome changes. Our work enables development of predictive models with significant implications for public health, agriculture, and wildlife management.

ID: 165 / CP8: 4

Contributed abstract

Conference Topics: Diagnostics, Ectoparasites, Molecular Biology

Keywords: Scabies, skin and soft tissue infections, molecular diagnostics, point-of-care diagnostics

Development of a Rapid point-of-care molecular diagnostic for scabies and co-infections

Matthew Paxman^{1,2}, Hanh Nguyen¹, Wei Lee¹, Jack Richards¹, Deborah Holt², Josh Francis², Ella Meumann², Bart Currie²

¹ZiP Diagnostics; ²Menzies School of Health Research

Scabies is a neglected tropical disease caused by *Sarcoptes scabiei* affecting >200 million people worldwide, especially impoverished populations. Scabies mites burrow into the skin, causing severe itching, skin lesions and co-infection with *Streptococcus pyogenes* (StrepA) and *Staphylococcus aureus* (S. aureus), which can lead to life-threatening sequelae such as rheumatic fever and heart disease. Point-of-care (POC) diagnosis would assist low-income countries where disease burden is greatest. This project aims to develop a world-first rapid and accurate POC diagnostic for the detection of scabies and co-infection with StrepA and S. aureus.

Bioinformatic analysis identified conserved and highly expressed gene targets for each species. Using loop-mediated-isothermal-amplification (LAMP) techniques and the ZiP-P2 POC platform by Australian company ZiP Diagnostics, the assay detects mite fragments (< 1 mite/reaction) within 10 minutes. Since typical scabies presents with <20 mites per host, this rapid and sensitive assay enables the development of simple sampling methods without needing vigorous skin scrapings to obtain a whole mite. Assay development for StrepA and S. aureus is underway, with preliminary data showing detection within 15 mins. Upcoming clinical studies in collaboration with the Menzies School of Health Research will help inform clinical decision-making and address an unmet need for this neglected disease.

ID: 179 / CP8: 5

Contributed abstract

Conference Topics: Ectoparasites, Host-parasite interactions, One Health, Veterinary Parasitology, Zoonoses

Keywords: Ticks, Ixodes ricinus, Tick-borne diseases, Transmission, Artificial feeding, infection, Borrelia afzelii

Optimizing artificial models for studying tick-borne pathogen transmission

Vincent C. Duru^{1,2,3}, Helena Rohackova^{4,5}, Abdul Ghafar³, Abdul Jabbar³, Ryan O.M. Rego^{4,5}, Ard M. Nijhof^{1,2}

¹Institute of Parasitology and Tropical Veterinary Medicine, Freie Universität Berlin, Robert-Von-Ostertag-Str. 7, 14163 Berlin, Germany.; ²Veterinary Centre for Resistance Research, Freie Universität Berlin, Robert-Von-Ostertag-Str. 8, 14163 Berlin, Germany.; ³Department of Veterinary Biosciences, Melbourne Veterinary School, The University of Melbourne, Werribee, VIC, 3030, Australia.; ⁴Institute of Parasitology, Biology Centre CAS, Branišovská 31, 37005, České Budějovice, Czech Republic.; ⁵Faculty of Science, University of South Bohemia, Branišovská 1760, 37005, České Budějovice, Czech Republic.

Artificial feeding and infection models provide a controlled alternative to the use of animal models to study tick-borne diseases (TBDs), but their ability to replicate natural infection dynamics remains under investigation. This study examined *Borrelia afzelii* transmission dynamics using an artificial tick feeding system (ATFS) to identify transmission windows under *in vitro* feeding conditions. Two groups of mice (8 mice/group) were used: one infected with the *B. afzelii* (CB43 strain), the other uninfected. Approximately 150 *Ixodes ricinus* larvae fed per mouse per group. Of the resulting nymphs, 120 nymphs were selected from each group and fed on an ATFS. Attachment and engorgement rates were 90%/77% (infected) and 83/60% (control), respectively. Samples of the blood on which the ticks fed were collected twice daily and will be screened for spirochete transmission via qPCR. To complement these transmission studies, we are also optimizing multiple artificial infection methods, including blood-meal spiking with *in vitro* cultured pathogen, direct microinjection, and larval immersion techniques. Preliminary results support the potential of artificial infection models for studying tick-borne pathogen transmission, but further validation is needed, especially for intracellular pathogens. This work optimizes artificial models as alternative tools for studying tick-pathogen interactions.

CP9: Fish Parasites 15 min talks

Time: Tuesday, 01/July/2025: 3:30pm - 5:00pm · Location: Conference room 3

Session Chair: Cecilia Power, RMIT University

Session Chair: Storm Martin, Murdoch University

ID: 196 / CP9: 1

Contributed abstract

Conference Topics: Aquaculture, Diagnostics, Epidemiology, Fish parasitology, Veterinary Parasitology

Keywords: Blood fluke, non-lethal sampling, diagnostics, aquaculture, fish health

Optimising sampling protocol for current routine health monitoring of *Cardicola* spp. infection in ranched Southern bluefin tuna

Melissa J. Carabott¹, Cecilia Power¹, Maree Widdicombe¹, Jemma Hudson¹, Paul A. Ramsland¹, Barbara F. Nowak^{1,2}, Nathan J. Bott¹

¹School of Science, STEM College, RMIT University, Bundoora, Victoria, 3083, Australia; ²Institute for Marine and Antarctic Studies, University of Tasmania, Hobart, Tasmania, 7004, Australia

Aporocotylid blood flukes of the genus *Cardicola* are parasites affecting bluefin tunas in aquaculture. In Australia, *Cardicola forsteri* and *C. orientalis* affect ranched Southern bluefin tuna (*Thunnus maccoyii*, SBT). SBT are a species of great economic value to Australian aquaculture due to their extensive international exports. Like in other large aquaculture systems, it is often difficult and expensive to sample the individual traits of all fish within a given population. Samples collected for blood fluke monitoring require the lethal collection of primary infection sites, the gills and heart. Lethal sample collection combined with logistical constraints such as the large size of tunas and the high costs associated with sampling at offshore cages, limits routine sampling efforts. As a result, sampling is restricted to commercial harvest, where only a small number of fish are opportunistically collected to minimise operational disruption, hindering current epidemiological studies. The assessment of non-lethal sampling techniques offers an alternative to traditional protocol. Non-lethal sampling offers the potential for more frequent and informative monitoring and will lead to improvements in fish health and welfare in tuna aquaculture systems.

ID: 143 / CP9: 2

Contributed abstract

Conference Topics: Coral Reef Parasites, Ecology, Ectoparasites, Fish parasitology, Wildlife parasitology

Keywords: fish parasites; animal behaviour; self-remedy; disease ecology

How do fishes manage parasitic diseases?

Kate Hutson^{1,2}, David Vaughan³, Richard Saunders⁴

¹Cawthron Institute, Australia; ²James Cook University, Australia; ³Central Queensland University; ⁴University of Tasmania

Amidst the COVID-19 pandemic we observed drastic human behavioural changes from a disease threat. Indeed, disease drives the evolution of transformational and spectacular behaviours in fishes as it does in terrestrial animals. In this presentation I will talk about how fishes manage parasitic diseases through self-remedy, by utilizing natural resources around them. Some of these behaviours are well known while others are almost entirely unexplored. These behaviours include mass migrations, 'behavioural fever', zoopharmacognosy, fish visiting the doctor (=cleaning interactions) and bizarre interspecific associations - you'd have to be driven crazy by external parasites to approach a shark to rub on their skin. The need for self-remedies to prevent or manage parasitic diseases affects all individual organisms. It has driven considerable complexity in fishes and is in such demand in nature that many species have made treating other organisms their specialization.

ID: 140 / CP9: 3

Contributed abstract

Conference Topics: Aquaculture, Fish parasitology, Host-parasite interactions, Protozoa

Keywords: protozoan; notifiable; shellfish disease; aquatic animal health; aquaculture

Perkinsus olseni in green-lipped mussels: in vitro culture, infectivity, and disease progression

Kate Hutson^{1,2}, Joanna Copedo¹, Lizenn Delisle¹

¹Cawthron Institute, New Zealand; ²James Cook University, Australia

Perkinsus olseni is a WOA notifiable protozoan parasite that infects a wide variety of molluscs and gastropods. Regularly associated with mass mortality events, it causes significant economic loss in the aquaculture sector worldwide. In New Zealand, the presence of the parasite has never been clearly associated with green-lipped mussel *Perna canaliculus* mortality events, even though it is regularly detected in young and adult mussels. The emergence of *P. olseni* in commercially important species like the green-lipped mussel raises concerns for the aquaculture industry, warranting further investigation to assess associated risks. Following the establishment of the first in vitro culture of *P. olseni* from *Perna canaliculus*, we described the parasite's life cycle and define its thermal optimum around 22 °C. In vivo challenge experiments at Cawthron's biocontainment facility have allowed us to describe the effects of seawater temperature on parasite growth and infectivity, mussel immune competency, and disease progression.

ID: 229 / CP9: 4

Contributed abstract

Conference Topics: Biodiversity, Coral Reef Parasites, Fish parasitology, Wildlife parasitology

Keywords: Diplostomida, Schistosomatoidea, evolutionary history

Freshwater blood flukes in marine fishes

Storm Martin

Murdoch University, Australia

The blood flukes are among the most important, interesting and intensively studied parasitic helminths, yet remarkably little progress has been made towards understanding their evolutionary history. We do not know when, where or in which host

groups the blood-fluke life cycle originated. The predominance for tetrapod definitive hosts among the Diplostomida might suggest a relatively recent tetrapod and freshwater origin for the blood flukes with subsequent radiation via host-switching, whereas the diversity of blood flukes in both basal and derived lineages of fishes might suggest an earlier marine origin prior to the rise of tetrapods if cophyly is invoked. The two largest lineages of fish blood flukes are the Aporocotylidae in marine bony fishes and the Sanguinicolidae in freshwater bony fishes. I will report on discovery of a new sanguinicolid, a freshwater blood fluke, in a marine angelfish (Acanthuriformes: Pomacanthidae) at Ningaloo Reef, Western Australia. I will present new speculations on the evolutionary history of the blood flukes inspired by this remarkable worm.

ID: 161 / CP9: 5

Contributed abstract

Conference Topics: Aquaculture, Bioinformatics, Fish parasitology, Immunology

Keywords: Aquaculture, Aporocotylidae, Transcriptome, Immune response, Notch signalling

Transcriptome of ranched Southern bluefin tuna, *Thunnus maccoyii*, reveals a localised and systemic immune response during *Cardicola forsteri* (Trematoda: Aporocotylidae) infection

Maree Widdicombe¹, Alexis Marshall¹, Oliver White², Bronwyn Campbell¹, Melissa Carabott¹, Jemma Hudson¹, Paul Ramsland¹, Barbara Nowak³, Cinzia Cantacessi⁴, Nathan Bott¹

¹RMIT University, Australia; ²The Natural History Museum, England; ³University of Tasmania, Australia; ⁴University of Cambridge, England

Ranched Southern bluefin tuna (SBT), *Thunnus maccoyii*, are susceptible to infection by aporocotylids (Trematoda: Digenea), that represent an ongoing health concern for the Australian SBT aquaculture industry. Here, we performed a whole transcriptomic analysis of the gills and anterior kidney of ranched SBT infected with *Cardicola forsteri*, to characterise host immune responses against the parasite. SBT were sampled during the 2022 commercial harvest after 12 weeks of ranching. Total RNA was extracted from the gills (site of parasite infection- eggs) and anterior kidney (immune organ) of *C. forsteri*-infected and uninfected SBT. The mRNA was sequenced and the SBT transcriptome was assembled by mapping against the SBT reference genome. In the gills, a total of 180 differentially expressed genes (DEG) (105 upregulated, 75 downregulated) were identified between infected and uninfected SBT. A subset of annotated genes with putative immune roles in the gills showed an upregulation of the Notch signalling pathway. In the anterior kidney, 300 DEGs (142 upregulated, 153 downregulated) were identified from the total transcriptome. Transcripts putatively associated with host immune responses were associated with a downregulation of the ubiquitin mediated proteolysis and 4 innate immune signalling pathways, i.e. NOD-like, C-type lectin, toll-like, and the RIG-I-like signalling receptor pathways.

ID: 232 / CP9: 6

Contributed abstract

Conference Topics: Aquaculture, Fish parasitology, One Health, Zoonoses, Other

Keywords: seafood-borne parasites, diet, health, rural, regional

Unequal bites: Understanding disparities in seafood access, consumption, and parasitic risk between regional and metropolitan Australia

Natalie Jefferson¹, Alexa Seal², Colin Stack³, Shokoofeh Shamsi¹

¹Gulbali Institute, Charles Sturt University, Australia; ²University of Notre Dame, Australia; ³Western Sydney University, Australia

Seafood is internationally recognised for its health benefits, particularly as a rich source of omega-3 long-chain polyunsaturated fatty acids. Yet despite national dietary guidelines recommending regular seafood intake, many Australians, especially those in regional and rural areas, fail to meet these recommendations. Factors such as cost, availability, perceived quality, and food safety concerns all contribute to low seafood consumption. For communities located inland, limited access to fresh seafood may heighten both nutritional disadvantage and vulnerability to food safety risks, particularly those related to parasites in fish.

To investigate these issues, we conducted an online survey targeting Australians across regional and metropolitan settings to identify key drivers and barriers to seafood consumption. We also explored community knowledge around seafood safety, including awareness of zoonotic parasites and how these perceptions influence dietary behaviours. The most commonly consumed fish species in both inland and coastal areas were also documented to help assess potential differences in exposure to parasitic infections.

This study contributes novel insights into the intersection of food security, public health, and parasitology in an Australian context. Findings will inform risk communication strategies, support behaviour change initiatives, and promote equitable access to healthy, safe seafood for all Australians, regardless of postcode.

CP7.1: Omics 5 min talks

Time: Tuesday, 01/July/2025: 4:45pm - 5:00pm · Location: Conference room 1

Session Chair: Ghizal Siddiqui, Monash University

Session Chair: Balu Balan, Walter and Eliza Hall Institute

ID: 240 / CP7.1: 1

Contributed abstract

Conference Topics: Bioinformatics, Epidemiology, Genomics, Molecular Biology

Keywords: onchocerciasis, vector biology, comparative genomics, genome assembly

Exploring the Genomes of African *Simulium*: De Novo Genome Assembly, Annotation, and Insights into Genomic Diversity Across Three Black Fly Species

Neha Sirwani¹, Emily Hendrickson^{1,5}, Millicent Opoku^{1,3}, Sindew Feleke^{1,4}, Kwadwo Frempong³, Warwick Grant¹, Shannon Hedtke^{1,2}

¹Department of Ecological and Evolutionary Biology, School of Biomedicine, Agriculture and Environment, La Trobe University, Bundoora, VIC, Australia; ²La Trobe Institute of Molecular Science, La Trobe University, Bundoora, VIC, Australia; ³Noguchi Memorial Institute for Medical Research, Accra, Ghana; ⁴Ethiopian Public Health Ministry, Adis Abbaba, Ethiopia; ⁵University of California, Sandiego, USA.

Blackflies in the genus *Simulium* are the sole vectors of *Onchocerca volvulus*, the human filarial parasite responsible for onchocerciasis (river blindness), a neglected tropical disease endemic to sub-Saharan Africa. Species within this genus exhibit significant ecological and biological variation—including differences in host preference, breeding site selection, and vector competence—resulting in varied transmission dynamics across regions. A key challenge in vector surveillance is species delimitation within the *Simulium damnosum* complex, due to the lack of distinctive morphological markers in adults and reliance on cytotoxicology, which requires larval stages and is unsuitable for large-scale use. Here, we present the first nuclear genome assemblies for three *Simulium* species: two from the *S. damnosum* complex and one *S. neavei*, using a hybrid *de novo* assembly approach that integrates Oxford Nanopore and Illumina sequencing from individual flies. We conducted comparative genomic analyses to annotate orthologous gene clusters and assess divergence. Additionally, whole genome alignments revealed syntenic regions and high genetic differentiation between *Simulium damnosum* from East and West Africa. These new genomes serve as a foundational resource for studying genome evolution and adaptation in vector species. Our findings pave the way for integrating genomics into vector surveillance, supporting efforts toward onchocerciasis elimination.

ID: 216 / CP7.1: 2

Contributed abstract

Conference Topics: Ectoparasites, Genomics

Keywords: Ixodes holocyclus, Genome, Transcriptome, Ticks, Ectoparasite

The chromosome-scale assembly of the Australian Paralysis Tick, *Ixodes holocyclus*

Amrita Vijay¹, Thomas Karbanowicz², Quentin Gouil³, Alexander Gofton⁴, Balu Balan¹, Louise Baker¹, Stefano Gaiarsa⁴, Pradip Roy^{1,5}, Shilpa Kapoor⁵, Clare A Anstead⁵, Ala Tabor², Nathan Lo⁶, Jan Riemer⁷, Fabrizia Stavru⁸, Davide Sasser⁹, Peter Czabotar¹, Tony Papenfuss¹, Aaron Jex^{1,5}

¹WEHI, Australia; ²The University of Queensland, Queensland Alliance for Agriculture & Food Innovation, St Lucia, Queensland, Australia; ³Olivia Newton-John Cancer Research Institute, Australia; ⁴Zoonotic & Arboviral pathogens, Health & Biosecurity, CSIRO, Canberra, Australia; ⁵Microbiology and Virology unit at Policlinico San Matteo, Fondazione IRCCS, Pavia, Province of Pavia, Italy; ⁶Faculty of Veterinary and Agricultural Sciences, The University of Melbourne, Parkville, Victoria, Australia; ⁷School of Life and Environmental Sciences, The University of Sydney, New South Wales; ⁸Department for Chemistry, Institute for Biochemistry, University of Cologne, Cologne, Germany; ⁹Unité de Biologie Evolutive de la Cellule Microbienne, Institut Pasteur, Paris, France

Ixodes holocyclus, the Australian eastern paralysis tick, is a medically and veterinary significant ectoparasite capable of producing potent neurotoxins known as holocyclotoxins. These toxins can induce rapidly ascending flaccid paralysis in companion animals, livestock, and humans, often leading to fatal outcomes. Despite its importance, the molecular mechanisms governing toxin production, host specificity, and survival remain poorly understood, largely due to the scarcity of genomic and transcriptomic resources. To address this gap, we present the first high-quality, chromosomal-scale genome assembly of *I. holocyclus* using an integrated sequencing strategy combining Oxford Nanopore long reads, Illumina short reads, and Hi-C chromatin conformation capture. The resulting 1.9 Gb genome is robustly annotated with gene models derived from a comprehensive *de novo* transcriptome comprising 18,324 main transcript isoforms, enabling the identification of alternative splicing events. To explore genomic diversity and its potential links to ecological adaptation and vector capacity, we further sequenced 100 *I. holocyclus* individuals across Australia. Together, these genomic and transcriptomic resources represent a significant advancement in tick biology, offering valuable insights into the genetic basis of toxin production, host-parasite interactions, and the development of targeted control strategies against tick-borne diseases.

ID: 260 / CP7.1: 3

Contributed abstract

Conference Topics: Genomics, Protozoa

Keywords: *Dientamoeba fragilis*, Genomics, Nanopore

Stepping Towards a Genome of *Dientamoeba fragilis*

Luke Hall¹, John Ellis¹, Damien Stark²

¹Faculty of Science, University of Technology Sydney, Australia; ²SydPath, St Vincents Hospital, Sydney, Australia

Dientamoeba fragilis is a protozoan parasite mired in controversy regarding whether it is a pathogen and what the infective stage is: cyst or trophozoite with pinworm ova as a vector. Sequencing a genome could identify virulence factors and cyst wall proteins, developing a better understanding of the biology and pathology of this parasite building consensus. No attempt has been successful in sequencing the genome of *D. fragilis*. The main challenge is the large number of bacteria present in samples as there is no axenic culture method for *D. fragilis*, resulting in almost all sequence data generated matching non-

target bacterial species. Even replacing the bacterial community with a single species did not help as most data was still bacterial, just of less diverse origin. We are currently using the adaptive sampling acquisition option of Nanopore to overcome this challenge and decrease the abundance of this non-target data. Refinement of the non-target database is required for each sample based on the most prevalent bacterial species in it. Smaller non-target databases allow for more efficient rejection of bacterial reads in samples, enhancing sequencing outcomes. Further exploration of adaptive sampling will enhance the production of *D. fragilis* genome data.

CP8.1: Arthropod Parasites & Vectors 5 min talks

Time: Tuesday, 01/July/2025: 4:45pm - 5:00pm · *Location:* Conference room 2

Session Chair: Charlotte Oskam, Murdoch University

Session Chair: Vern Bowles, The University of Melbourne

ID: 181 / CP8.1: 1

Contributed abstract

Conference Topics: Ectoparasites, Livestock Parasites, Veterinary Parasitology

Keywords: Cattle tick, *Rhipicephalus australis*, microbiome, amplicon sequencing, 16S rRNA gene

Investigations on the microbiome of *Rhipicephalus australis*

Bahar E Mustafa¹, Abdul Ghafar¹, Swaid Abullah², Ian Beveridge¹, Charles G. Gauci¹, Alejandro Cabezas-Cruz³, Ard M. Nijhof^{4,5}, Abdul Jabbar¹

¹Melbourne Veterinary School, The University of Melbourne, Werribee, Victoria 3030, Australia; ²School of Veterinary Science, Faculty of Science, University of Queensland, Gatton, QLD 4343, Australia; ³UMR BIPAR, INRAE, ANSES, Ecole Nationale Vétérinaire d'Alfort, Université Paris-Est, Maisons-Alfort, France; ⁴Institute of Parasitology and Tropical Veterinary Medicine, Freie Universität Berlin, Robert-Von-Ostertag-Str. 7, 14163, Berlin, Germany; ⁵Veterinary Centre for Resistance Research, Freie Universität Berlin, Berlin, Germany

Ticks act as vectors for many pathogens affecting both humans and animals. For example, the cattle tick can transmit various pathogens such as *Anaplasma* and *Babesia* spp., resulting in significant economic losses to the global cattle industry. Despite its relevance, our understanding of the cattle tick's microbiome, particularly in Australia, is limited. This study aimed to characterise the microbiome of different life stages of *Rhipicephalus australis*, including larvae, nymphs and adults, by targeting the V3-V4 regions of the 16S rRNA gene. Specimens collected from cattle farms in Queensland in 2024 were decontaminated using a bleach solution and preserved in 70% ethanol before being subjected to DNA extraction and PCR. Sequencing of the libraries was performed using the Illumina NextSeq™ 1000 platform. Paired-end FastQ reads were uploaded in the QIIME2 environment for subsequent quality filtering and analysis using cutadapt, DADA2, VSEARCH, and BLASTn. Preliminary analyses revealed the presence of several bacterial taxa, including *Arsenophonus*, *Acinetobacter*, *Coxiella*, *Coxiella*-like endosymbionts, *Stenotrophomonas*, *Lactococcus*, *Morganella*, *Salmonella*, *Staphylococcus*, *Providencia* and other bacteria mainly belonging to the families Coxiellaceae, Enterobacteriaceae, Moraxellaceae, Staphylococcaceae and Streptococcaceae. The data obtained from this study will provide valuable insights into the microbiome of *R. australis* and support developing of improved control strategies.

ID: 208 / CP8.1: 2

Contributed abstract

Conference Topics: Ectoparasites

Keywords: Scabies, Microbiota

Identifying the core-microbiota of *Sarcoptes scabiei* life-stages

Sara Taylor, Martha Zakrzewski, Deepani D Fernando, Katja Fischer

Infection and Inflammation Program, QIMR Berghofer Medical Research Institute, Brisbane, Australia

Scabies is a neglected tropical disease with an estimated global prevalence of ~200 million cases. The obligate lifecycle of *Sarcoptes scabiei* has traditionally presented challenges to understanding the basic nature of the mite's biology. The establishment of an *ex-vivo* culture system using a porcine animal model has enabled a greater understanding of the mite's complex biology. To date however, there is limited research on the internal microbiota of the mite. The current understanding comes from an analysis performed on adult females and eggs, which found the bacterial genera *Klebsiella*, *Corynebacterium*, *Streptomyces*. Given the limited treatment options available for scabies, and the association with secondary bacterial infections established, this study has attempted to provide the first life-stage specific microbiota, to identify core components of the mite's microbiota, that could be essential endosymbionts and potential future drug targets. We collected washed and unwashed samples from all life-stages (females, males, nymphs, larvae and eggs) and performed 16s full-length rRNA amplicon sequencing on the PacBio platform. A total of 3500187 reads remained after primer trimming and quality filtering, with 487 taxa found, and ~38 per sample. This analysis found that the bacterial genera *Corynebacterium*, *Serratia* and *Acinetobacter* are present across all life-stages.

ID: 138 / CP8.1: 3

Contributed abstract

Conference Topics: Biodiversity, Ectoparasites

Keywords: Genetic barcoding, Mosquito

What Morphology Sweeps Under the Rug: The Hidden Genetic Diversity of Perth's Mosquito Populations.

Ashleigh Peck¹, Alan Lymber^{2,3}, Siobhon Egan⁴, Amanda Ash^{1,5}

¹School of Medical, Molecular and Forensic Sciences, Murdoch University; ²School of Environmental and Conservation Sciences, Murdoch University; ³Centre for Sustainable Aquatic Ecosystems, Harry Butler Institute, Murdoch University; ⁴Centre of Computational and Systems Medicine, Murdoch University; ⁵Centre for Biosecurity and One Health, Murdoch University

Mosquitoes are the most significant vectors for human and animal diseases. However, not all mosquito species are competent at disease transmission, so accurate species identification is crucial to determine which species are competent vectors. Traditionally, species identification has relied on time-consuming taxonomic methods, which depend on identifying species-specific features that can be damaged during field collection or are cryptic among species.

Genetic barcoding using the Cytochrome Oxidase I (COI) gene region aids in species identification by comparing sequences to established databases. This study used barcoding methods to generate COI barcodes for fifteen endemic species from 6 genera collected in Perth's metropolitan region. Phylogenetic and p-distance analysis of generated and collated COI barcodes offers insight into Perth mosquito populations' conspecific and congeneric diversity.

We observed greater genetic diversity than morphological diversity by identifying species clades for *Aedes notoscriptus*, *Aedes alboannulatus*, and *Culex annulirostris*. The identified conspecific diversity raises questions about whether vector competence is diffuse across species clades. Additionally, there is an undefined diversity within the important and morphologically similar mosquito group, the *Culex pipiens* complex. Therefore, the inability to distinguish species within complexes with overlapping morphology and genetic barcoding complicates the assessment of vector competence more than initially anticipated.

P2: Plenary Lecture: Don McManus Tropical Health Research Centre and QIMR Berghofer Prize

Time: Wednesday, 02/July/2025: 9:45am - 10:30am · Location: Conference plenary room
Session Chair: Darren Gray, QIMR Berghofer

ID: 142 / P2: 1

Contributed abstract

Conference Topics: Malaria, Molecular Biology

Keywords: Gene drives, malaria, Plasmodium, transmission

Building gene drives to directly target malaria parasites.

Mary-Louise Wilde, Christopher D. Goodman, Geoffrey I. McFadden

School of BioSciences, The University of Melbourne, Parkville, Victoria, Australia

Gene drives are self-propagating genetic elements that defy the usual laws of inheritance. A gene drive can spread rapidly through a population even if the inherited trait reduces fitness, offering a mechanism to eradicate pests and pathogens by 'driving' them to extinction. We designed a gene drive to wipe out the malaria parasite *Plasmodium* by forcing a sex bias onto the population. In this strategy, we removed the ability to make males, resulting in a female-only population. The loss of males from the population means that *Plasmodium* cannot infect its definitive host—the mosquito. Our gene drive in *Plasmodium berghei* collapsed the parasite population in 5 weeks. We then built a gene drive for the human malaria parasite *P. falciparum* which was similarly effective at converting all progeny to females in just one sexual cross. Modelling predicts that inoculating fewer than 0.1% of the at-risk human population with our gene drive could eradicate *falciparum* malaria in ~2 years, averting 253 million infections and 660,000 deaths annually. There are major safety, ethical, regulatory and social obstacles facing a *Plasmodium* gene drive. Nevertheless, current tools are facing an uphill battle to control malaria in resource poor countries thus gene drives warrant exploration.

CP10: Drugs & Drug Resistance 15 min talks

Time: Wednesday, 02/July/2025: 11:00am - 12:15pm · Location: Conference room 1

Session Chair: Jacinta Macdonald, Griffith University

Session Chair: Darren Creek, Monash University

ID: 188 / CP10: 1

Contributed abstract

Conference Topics: Drugs, Malaria, Proteomics

Keywords: Plasmodium falciparum, Antimalarials, Drug targets, Proteomics

Uncovering the mechanism of action of second generation bis-triazines, a potent new class of antimalarials

Jennifer Le¹, Carlo Giannangelo¹, Annaliese Dillon², Paul Stuppel², Ghizal Siddiqui¹, Darren Creek¹

¹Drug Delivery, Disposition and Dynamics, Monash Institute of Pharmaceutical Sciences, Monash University, Parkville, 3052, Australia; ²Medicinal Chemistry, Monash Institute of Pharmaceutical Sciences, Monash University, Parkville, 3052, Australia

Widespread resistance to all current antimalarials threatens the control and eradication of malaria. Second-generation bis-triazines are a new antimalarial class with fast-killing activity, low nanomolar potency and no known cross-resistance. However, the novel mechanism of action (MOA) remains unknown.

In vitro combination drug-pulse assays using various inhibitors was performed to identify potential modulators of bis-triazine activity. We also included an antimalarial candidate with an unknown MOA currently under preclinical development with MMV. Only E64d, a cysteine protease inhibitor, altered bis-triazine activity (6-fold increase in IC₅₀), whereas both E64d and chloroquine decreased activity of the preclinical candidate (10-fold IC₅₀ increase), indicating the involvement of the haemoglobin digestion pathway in its MOA.

To identify the molecular target of the bis-triazine analogues we used solvent proteome profiling. This method was validated using atovaquone which selectively stabilised 7 proteins of the parasite cytochrome bc1 complex, its known target, amongst ~4,500 detected proteins. Bis-triazine hits were mostly nuclear proteins, with the RING finger E3 ubiquitin-protein ligase of particular interest as it was previously identified as a potential target of our predecessor series. Further investigations will use proteomics and transcriptomics studies to confirm these targets and elucidate the antimalarial mechanism of this promising new antimalarial class.

ID: 274 / CP10: 2

Contributed abstract

Conference Topics: Diagnostics, Drugs, Malaria

Keywords: Malaria, Plasmodium falciparum, drug resistance, diagnostics

Evolution and Spread of Artemisinin- and Diagnostic-Resistant (AMRED) Malaria Parasites in Africa.

Rachael Yong^{1,2}, Alyssa E. Barry^{1,2}, Qixin He³, Gordon A. Awandare⁴, Charles Narh^{1,2}

¹School of Medicine, Deakin University, Waurn Ponds VIC 3216, Australia; ²Burnet Institute, Melbourne VIC 3004, Australia;

³Department of Biological Sciences, Purdue University, West Lafayette, IN 47907, USA; ⁴West African Centre for Cell Biology of Infectious Pathogens (WACCBIP), University of Ghana, Legon, Accra, Ghana

Artemisinin-based combination therapies (ACTs) are mainstay in malaria control, halving malaria cases and deaths in Africa since the early 2000s. Artemisinin resistant (ART) *Plasmodium falciparum* (*Pf*) parasites have emerged in East Africa (EAF), where these resistant parasites also have linked adaptations to evade detection on HRP2-based RDTs (i.e. diagnostic resistant). Artemisinin- and Diagnostic-Resistant (AMRED) parasites are now widespread in EAF, but their evolution and factors likely to drive their spread to the rest of Africa remain unknown.

We conducted a systematic review of published African studies between 2000 to 2024 using search terms that included ACTs, RDTs, and Pf resistance markers - K13 (artemisinin), crt/mdr1 (chloroquine and artemisinin partner drug resistance/PDR), and *hrp2/hrp3* deletions (RDTs); 118/453 were eligible for analysis.

Preliminary results showed over 10 ART K13 mutations in Africa; most below 5% in West Africa (WAF) but two validated mutations reaching >15% in EAF. Pf *hrp2/3* deletions were predominant in EAF reaching 80%, but <5% in WAF except in Ghana where one study reported >30% in 2018, warranting further investigations. Temporal analysis suggested ACTs selected for PDR in 2008-2018 in Africa before ART 2019-2022 in EAF. Pf prevalence data suggested declining malaria transmission played a role in AMRED emergence.

ID: 256 / CP10: 3

Contributed abstract

Conference Topics: Drugs, Malaria

Keywords: mETC, drugs, toxoplasmosis, malaria

Identification of a potent and selective inhibitor of the mitochondrial electron transport chain in the parasites that cause malaria and toxoplasmosis.

Rachel A Leonard, Yusheng Wang, Ruijia Lang, Deyun Qiu, Adele M Lehane, Giel G van Dooren

Australian National University, Australia

The apicomplexan parasites that cause severe malaria (*Plasmodium falciparum*) and toxoplasmosis (*Toxoplasma gondii*) are responsible for significant mortality and morbidity globally. Drugs are needed to combat these parasites, although the advent of parasite resistance to frontline treatments highlights the urgent need for new drugs targeting drug-resistant strains. *P. falciparum* and *T. gondii* both contain a mitochondrial electron transport chain (mETC) that is essential for their survival and a validated drug target. We have developed compound library screens to identify inhibitors that selectively target the parasite mETC over the human mETC. In these screens, we have identified a compound - MMV1794211 - that exhibits exceptional potency against *P. falciparum* and *T. gondii*. MMV1794211 exhibits selectivity for the parasite mETC over the human mETC, and remains active against parasites resistant to other mETC inhibitors. MMV1794211 belongs to a class of chemicals known as strobilurins. Our data indicate that the structure of strobilurins are critical determinants of their potency against parasites, and we are exploring the underlying basis for this. In sum, we have identified a potent mETC inhibitor that shows promise as a lead against the diseases caused by *T. gondii*, *P. falciparum* and related apicomplexans.

ID: 123 / CP10: 4

Contributed abstract

Conference Topics: Biochemistry, Drugs, Malaria

Keywords: Malaria, Plasmodium, Aminopeptidase, Drug discovery, Structure-based drug design

The development of a novel class of aminopeptidase inhibitors as potent antimalarials

Mahta Mansouri^{1,2}, Sheena McGowan³, Peter Scammells¹

¹Monash Institute of Pharmaceutical Sciences, Monash University, Melbourne, VIC.; ²Walter and Eliza Hall Institute of Medical Research, Melbourne, VIC.; ³Biomedicine Discovery Institute, Monash University, Melbourne, VIC.

Malaria is a widespread parasitic disease that continues to place a significant burden on global health. The evolution of resistance diminishes the efficacy of current therapies, highlighting an urgent need for novel therapeutics with new mechanisms of action. Haemoglobin digestion is a critical survival pathway in the intra-erythrocytic parasite in which metalloaminopeptidases play a central role. Previous studies have demonstrated that blocking the activity of *Plasmodium falciparum* M1 and M17 aminopeptidases leads to parasite death. Dual inhibition of these two enzymes is a promising avenue for discovering novel therapeutic agents with a novel mechanism of action.

In this work, a new class of inhibitors with a novel scaffold were identified. The new series demonstrated a three-fold improvement in activity which was retained in drug-resistant strains. They were highly effective when tested against the *P. vivax* and *P. berghei* target homologs, exhibiting both cross-peptidase and cross-species activity. Further studies revealed good stability in hepatocytes and blood, with improved pharmacokinetic properties following oral administration *in vivo*. Subsequent *in vivo* efficacy study showed that the lead inhibitor effectively treated mice infected with *P. berghei*, in a similar profile to artesunate. Altogether, this series presents promising potential for future development into potent antimalarials.

ID: 105 / CP10: 5

Contributed abstract

Conference Topics: Bioinformatics, Drugs, Malaria

Keywords: artemisinin; lumefantrine; Plasmodium falciparum; treatment failure

***pfk13* and *pfubp1* genotypes in African *Plasmodium falciparum* isolates exhibiting reduced susceptibility to the antimalarials artemisinin and lumefantrine**

Colin Sutherland

LSHTM London, United Kingdom

The effectiveness of artemether-lumefantrine for treating malaria in East Africa is threatened by newly-emerging *P. falciparum* populations with reduced susceptibility to both therapeutic components. *In vitro* studies suggest that partial resistance to the artemisinin component, mediated chiefly by genetic variants of the *pfk13* locus, may occur in concert with increased tolerance of lumefantrine. Two Ugandan lines from UK travellers, HL2208 and HL2210, display significantly reduced susceptibility to both artemisinin and lumefantrine, and carry variant alleles at several resistance-associated loci. In this study, phenotype-genotype associations are described in an extended panel of >40 *P. falciparum* lines established from UK travellers since 2016. Parasites with enhanced ring-stage survival against artemisinin were identified from Kenya, Uganda, Zambia and Namibia. These carried newly-identified or previously established variants in propeller or non-propeller domains of *pfk13* or harboured variant alleles of *pfap2mu*, *pfcoronin* or *pfubp1*. Among 25 isolates from ten different countries collected since 2022 that were evaluated for lumefantrine susceptibility, the seven least susceptible isolates were all from individuals who had travelled to

Uganda. Evidence will be presented that both *pfk13*-dependent and -independent partial artemisinin resistance has arisen in multiple African settings, and that some erosion of lumefantrine efficacy has occurred in Uganda.

CP11: Immunology & Vaccination 15 min talks

Time: Wednesday, 02/July/2025: 11:00am - 12:15pm · Location: Conference room 2

Session Chair: Li Jin Chan, The Walter and Eliza Hall Institute

Session Chair: Michael Duffy, University of Melbourne

ID: 227 / CP11: 1

Contributed abstract

Conference Topics: Apicomplexa Biology, Biochemistry, Cell Biology, Malaria, Molecular Biology, Vaccines

Keywords: monoclonal antibodies, immunity, vivax, synergy

Monoclonal antibodies targeting malaria combine to recruit immune components synergistically for greater efficacy

Lee M. Yeoh^{1,2}, Chiara L. Drago¹, D. Herbert Opi^{1,2,3}, Anna C. Winnicki⁴, Lenore L. Carias⁴, Alyssa N. Malachin⁴, Melanie H. Dietrich^{2,5}, Nicolai C. Jung⁵, Wai-Hong Tham^{2,5}, Jürgen Bosch^{4,6}, Christopher L. King^{4,7}, James G. Beeson^{1,2,3}

¹Burnet Institute, Melbourne, Australia; ²The University of Melbourne, Parkville, Australia; ³Monash University, Clayton, Australia; ⁴Case Western Reserve University, Cleveland, USA; ⁵Walter and Eliza Hall Institute of Medical Research, Parkville, Australia; ⁶InterRayBio LLC; ⁷Veterans Affairs Medical Center, Cleveland, USA

Monoclonal antibodies (mAbs) are effective therapeutics against diseases including cancer and COVID-19. There are three promising malaria mAbs in clinical trials.

However, we have only limited knowledge about how malaria mAbs work in vivo. There are two major areas where knowledge is lacking. Firstly, very little is known about the ability of malaria mAbs to recruit downstream components of the immune system. Secondly it is unclear how combinations of mAbs may work synergistically, giving greater efficacy than otherwise expected and increasing the potency of therapeutics.

In addition to therapeutics, mAbs can also provide details about epitopes of potential vaccine candidates. By testing permutations of mAbs simultaneously, we can recapitulate the natural polyclonal response to vaccines or infection, determining which epitopes work together and which do not, better informing vaccine design in the future.

We recently cloned and expressed over 20 mAbs specific to a malaria antigen (PvAMA1). Many mAbs strongly inhibit invasion of parasites in vitro. A number are also capable of stimulating strong immune responses. In addition, we have identified a group of mAbs that are capable of working synergistically. We have also modified the Fc region of these mAbs to increase immune recruitment and synergy.

ID: 204 / CP11: 2

Contributed abstract

Conference Topics: Immunology, Malaria

Keywords: Malaria, VAR2CSA, *P.falciparum*, Pregnancy, antibody

Characterising monoclonal antibodies to placental malaria antigen, VAR2CSA

Vivin Kokuhenadige¹, Christopher Gonelli², Olivia Wilhelm¹, Wina Hasang¹, Oscar Williams¹, Ching-Seng Ang³, Phantica Yambo⁴, Paula Tesine⁴, Alice Mengi⁴, Benishar Kombut⁴, Holger Unger^{5,6,7}, Adam Wheatley², Elizabeth Aitken^{1,2}, Stephen Rogerson^{1,8}

¹Department of Infectious Diseases, The Peter Doherty Institute of Infection and Immunity, University of Melbourne, Melbourne, Victoria, 3000, Australia; ²Department of Microbiology and Immunology, The Peter Doherty Institute of Infection and Immunity, University of Melbourne, Melbourne, Victoria, 3000, Australia; ³Mass Spectrometry and Proteomics Facility, Bio21 Molecular Science and Biotechnology Institute, University of Melbourne, Parkville, VIC 3052, Australia; ⁴Papua New Guinea Institute of Medical Research, Madang, Papua New Guinea; ⁵Department of Obstetrics and Gynaecology, Royal Darwin Hospital, Darwin, NT, Australia; ⁶Menzies School of Health Research, Charles Darwin University, Darwin, NT, Australia; ⁷Department of Clinical Sciences, Liverpool School of Tropical Medicine, Liverpool, United Kingdom; ⁸Department of Medicine (RMH), The Peter Doherty Institute of Infection and Immunity, University of Melbourne, Victoria, 3000, Australia

Placental malaria, due to the sequestration of *Plasmodium falciparum*-infected erythrocytes (IE), causes adverse pregnancy outcomes. The sequestration is mediated by VAR2CSA, a protein that binds to placental chondroitin sulfate A (CSA). VAR2CSA antibodies protect against adverse pregnancy outcomes; however, no pregnancy-specific vaccine or therapeutic exists to date. We identified and expressed VAR2CSA-specific IgG1 monoclonal antibodies (mAbs) using B cells of exposed Papua New Guinean women. VAR2CSA mAbs were characterised by their ability to recognise eight heterologous CSA-binding *P. falciparum* strains to neutralise CSA binding and/or induce phagocytosis of IEs by THP-1 monocytes. We identified 16 mAbs, and all targeted just two of the six domains of VAR2CSA. Cross-reactivity varied between mAbs, with 2D9 binding to all eight strains. Although individual mAbs did not promote phagocytosis, combinations targeting distinct VAR2CSA epitopes did. None of the mAbs inhibited IEs from binding to CSA. Structural studies using Hydrogen/Deuterium Exchange Mass Spectrometry (HDX-MS) with 2D9 revealed a broadly conserved epitope on globular VAR2CSA structure. Our findings revealed a conserved VAR2CSA epitope as a target for strain-transcending immunity and suggest that effective mAb therapies should combine mAbs targeting distinct epitopes. Altogether, we demonstrated how mAbs can dissect naturally acquired antibody responses to inform vaccine design.

ID: 128 / CP11: 3

Contributed abstract

Conference Topics: Malaria

Keywords: 6-cysteine proteins, transmission blocking, crystallography

Pfs48/45 nanobodies block *Plasmodium falciparum* transmission

Frankie Lyons^{1,2}, Jill Chmielewski^{1,2}, Mikha Gabriela^{1,2}, Li Jin Chan^{1,2}, Joshua Tong¹, Amy Adair¹, Kathleen Zeglinski^{1,2}, Quentin Gouil^{1,2,3,4}, Melanie Dietrich^{1,2}, Wai-Hong Tham^{1,2,5}

¹The Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria, Australia; ²Department of Medical Biology, The University of Melbourne, Parkville, Victoria, Australia; ³Olivia Newton-John Cancer Research Institute, Heidelberg, Victoria, Australia; ⁴School of Cancer Medicine, La Trobe University, Bundoora, Victoria, Australia; ⁵Research School of Biology, The Australian National University, Canberra, ACT, Australia

Malaria parasite fertilisation occurs within the *Anopheles* mosquito midgut. Interventions that inhibit parasite fertilisation prevent ongoing transmission and are necessary to achieve malaria elimination. A major target of transmission-blocking interventions is the *Plasmodium falciparum* 6-cysteine protein Pfs48/45. Pfs48/45 is expressed on the surface of gametocytes and gametes and is essential for parasite fertilisation. It forms a complex with Pfs230, another 6-cysteine protein and transmission-blocking target. We have generated the first collection of nanobodies against Pfs48/45, which bind to domain 3 with nanomolar affinities and recognize Pfs48/45 on the surface of gametocytes. In standard membrane feeding assays, the nanobodies significantly reduce parasite transmission to *Anopheles stephensi*. The crystal structure of our most potent nanobody in complex with Pfs48/45 reveals it binds a distinct epitope to TB31F, a leading transmission blocking monoclonal antibody currently undergoing clinical trial in humans. We have also generated bispecific nanobodies against Pfs48/45 and Pfs230 that recognize both antigens and have potent transmission-reducing activity. This work demonstrates the potential of nanobodies as a versatile antibody format that can reduce malaria transmission.

ID: 148 / CP11: 4

Contributed abstract

Conference Topics: Apicomplexa Biology, Malaria, Microscopy, Vaccines

Keywords: RH5, monoclonal antibodies, live-cell microscopy, synergy, invasion inhibition

Understanding how RH5 monoclonal antibodies work to inform future antimalarial therapies

Alysha Literski, Claudia Barnes, Oliver Looker, Hayley Bullen, Paul Gilson

Burnet Institute, Australia

Plasmodium falciparum (*Pf*) causes malaria and while vaccines targeting the liver-stage have been developed, they are not fully protective, underscoring the urgent need for novel vaccines that target the parasite blood-stage.

To survive in the bloodstream, *Pf* invades and replicates within red blood cells (RBCs), using its essential *Pf*RH5 protein to bind to the RBC receptor basigin, which helps trigger invasion. *Pf*RH5 is a leading blood-stage vaccine candidate as anti-*Pf*RH5 monoclonal antibodies (mAbs) sterically block the *Pf*RH5-basigin interaction, preventing RBC invasion.

However, as mAb concentrations are reduced their blocking efficiency steeply declines. To boost the efficiency of RH5 mAbs, we have successfully combined them with invasion-slowing compounds to give the RH5 mAbs more time to inhibit invasion.

Additionally, we observed that the *Pf*RH5 mAbs induce structural changes in extracellular parasites, transforming them into amoeboid forms known as "pseudo-rings." Through live-cell microscopy, we demonstrated that RH5 mAbs trigger pseudo-ring formation in 30% of parasites in approximately 10 minutes.

As the pseudo-rings are no longer able to invade RBCs, we aim to enhance their formation to augment the direct invasion-blocking activity of RH5 mAbs and inform the development of a more effective blood-stage malaria vaccine.

ID: 253 / CP11: 5

Contributed abstract

Conference Topics: Bioinformatics, Genomics, Malaria

Keywords: antigenic diversity, polymorphisms, malaria vaccines, haplotypes

Characterising The Global Genetic Diversity Of Plasmodium Falciparum Merozoite Surface Protein 1 (MSP1) To Inform Malaria Vaccine Design

Katelyn M. Stanhope^{1,2}, Myo T. Naung², Kirsty M. McCann², Lee M. Yeoh^{1,3}, James G. Beeson^{1,3,4}, Alyssa E. Barry^{1,2}

¹Burnet Institute, Melbourne, Australia; ²Centre for Innovation in Infectious Disease and Immunology Research, Institute for Mental and Physical Health and Clinical Treatment (IMPACT), School of Medicine, Deakin University, Geelong; ³The University of Melbourne, Parkville, Victoria, Australia; ⁴Monash University, Clayton, Victoria, Australia

Malaria causes significant morbidity and mortality worldwide, yet efforts to develop a highly efficacious vaccine are challenged by the extensive genetic diversity of *Plasmodium falciparum* antigens. Vaccines based on single antigen variants may not capture the full extent of variation circulating in parasite populations, limiting cross-strain protection. The full-length *P. falciparum* merozoite surface protein 1 (*MSP1*) is a promising blood-stage vaccine candidate, which has recently re-entered clinical trials. However, the genetic diversity in global parasite populations remains under-characterised. We aimed to characterise the global genetic diversity of key *MSP1* domains to inform vaccine design and serotype selection using over 20,000 publicly available *P. falciparum* genomes. The analysis included characterisation of the sequence variation in 33 countries based on *MSP1* gene sequences extracted from the publicly available *P. falciparum* genomes. Variant (haplotype) distributions and network analyses identified potentially antigenically distinct variants for immunological profiling. This work will support the identification of 'serotypes' using immunological assays and contributes to a deeper understanding of *MSP1* diversity and supports the rational design of next-generation, broadly protective malaria vaccines.

CP12: One Health 15 min talks

Time: Wednesday, 02/July/2025: 11:00am - 12:30pm · Location: Conference room 3

Session Chair: Amanda Ash, Murdoch University

Session Chair: Lucas Huggins, University Of Melbourne

ID: 293 / CP12: 1

Contributed abstract

Conference Topics: Diagnostics, Zoonoses

Keywords: uncommon zoonotic cases

Zoonotic parasite cases: the role of e-Diagnosis as a collaborative approach

Harsha Sheorey¹, Richard Bradbury², Anson Kohler³, Robin Gasser³

¹St Vincent's Hospital, Melbourne, Australia; ²James Cook University, Australia; ³The University of Melbourne, Australia

Accurate diagnosis of parasitic infections often relies heavily on the morphological identification of parasites—an area that demands substantial expertise and experience. However, with the retirement of many seasoned parasitology morphologists, there is a growing gap in this critical field. To help bridge this gap, an international voluntary e-group has been established, comprising expert parasitologists, including morphologists, molecular biologists, histopathologists, entomologists, and both human and veterinary parasitology specialists (including wildlife experts).

This talk will introduce the structure and function of this collaborative e-group, which supports the diagnosis of difficult and uncommon zoonotic parasite cases. The workflow involves submitting case-related images or queries to a central coordinator, who then circulates them among the group for rapid consultation and expert input. Many cases are confirmed by molecular sequencing by a member of the group.

A live demonstration of how the group operates will be presented, along with selected case examples primarily from Australia and New Zealand. The discussion will highlight the value of such a collaborative model in addressing current diagnostic challenges, while also acknowledging its limitations and areas for improvement.

ID: 249 / CP12: 2

Contributed abstract

Conference Topics: Diagnostics, Epidemiology, One Health, Zoonoses

Keywords: Zoonoses, Epidemiology, One Health

The first biological validation of *Taenia solium* risk assessment tools

Amanda Ash¹, Oula Bouphakaly², Bounnaloth Insiengmay³, Malavanh Chittavong², Davina Boyd¹, Andrew Larkins¹

¹College of Environmental and Life Sciences, Murdoch University, Australia; ²Department of Agricultural Sciences, National University of Laos; ³Department of Communicable Disease Control, Ministry of Health, Laos

Taenia solium places substantial burden on communities in low and middle-income countries, where neurocysticercosis is a leading cause of preventable epilepsy. Current diagnostic tests for *T. solium* are not suited for low resource settings or perform poorly. Several risk-assessment tools, based on risk rather than disease data, have been developed but have yet to be validated against biological data.

We trialled 5 risk-assessment tools; 2 national quantitative risk-assessment tools (LISA and MCDA), 2 local semi-quantitative tools, and 1 local qualitative tool and assessed their performance against biological *T. solium* taeniasis data from 28 villages.

T. solium positive villages had higher odds of being high-risk villages for all tools, however, this was only significant for the LISA and qualitative tools. Other tools that calculated risk scores also showed similar results, however, were not significant. All positive villages were hotspots for unimproved toilets by the LISA tool. The open defaecation component of the local rapid tool was the only component of any tool to demonstrate a significant relationship with positive *T. solium* villages.

This is the first biological validation of *T. solium* risk assessment tools and our results demonstrate that there are multiple tools that should be considered for further development.

ID: 164 / CP12: 3

Contributed abstract

Conference Topics: Epidemiology, One Health, Zoonoses

Keywords: Cysticercosis, Epidemiology, Neglected Tropical Diseases, One Health, Zoonoses

Cysticercosis in Laos: Prevalence, risk factors and predictive mapping

Andrew Larkins¹, Somphou Sayasone², Sarah Gbariel³, Amanda Ash¹

¹Murdoch University, Australia; ²Lao Tropical and Public Health Institute, Lao PDR; ³Ghent University, Belgium

Taenia solium cysticercosis is a leading cause of epilepsy in low and middle-income countries. There have been no nationally representative surveys in southeast Asia and decision makers rely on research from other populations. To support the control of cysticercosis in Laos, a national serosurvey was conducted in 150 villages in 2019 using the B158/B60 antigen ELISA.

The prevalence of cysticercosis in Laos was 5.47% (CI: 4.58 – 6.49%). Prevalence ranged from 0 – 43.75% between provinces and 0.85 – 15.74% between villages. Individuals living in northern or southern regions were almost twice as likely to have cysticercosis.

A predictive risk map was created using explanatory census variables and also identified northern and southern Laos as the highest risk areas. The proportion of pig households and poverty in a village were significant risk factors.

These results support and validate previous risk mapping efforts in Laos, with highest prevalences in northern and southern regions. Northern Laos has been the historical focus of cysticercosis research, however, this study highlights that Southern Laos should also be included. Given the insignificance of most individual risk factors, more general risk factors and the conditions in which people live may have a greater impact on cysticercosis.

ID: 102 / CP12: 4

Contributed abstract

Conference Topics: Helminthology, Livestock Parasites, One Health, Vaccines, Zoonoses

Keywords: Taenia solium, control, vaccination, pigs

Control of Taenia solium transmission by vaccination of pigs

Marshall Lightowers

University of Melbourne, Australia

Taenia solium is a zoonotic cestode parasite which causes neurocysticercosis in humans. Pigs transmit the parasite by acting as the intermediate host. A recombinant antigen vaccine, TSOL18, was developed for pigs which achieved high levels of protection against experimental challenge infections with *T. solium*. Field trials have been completed using the vaccine in Peru, Nepal, Cameroon, Uganda, Tanzania, Zambia and the largest and most recent being undertaken in Madagascar, completed in 2023. In several of these trials, pre-vaccination levels of porcine cysticercosis were determined by necropsy to be >30%. To date, a total of 168,707 pigs have been vaccinated in the field trials. As part of the assessment of the trials, a total of 768 slaughter-age pigs have been assessed for infection by total body necropsy. No vaccinated animal has been found to have a viable *T. solium* cyst. The vaccine is manufactured and registered as Cysvax by Indian Immunologicals Limited and is included in the WOAHA Terrestrial Manual and Terrestrial Code. The vaccine, together with the treatment of humans for taeniasis, has the potential to control transmission of *T. solium* and reduce the burden of neurocysticercosis.

ID: 170 / CP12: 5

Contributed abstract

Conference Topics: Diagnostics, Fasciolosis/Liver fluke, Molecular Biology

Keywords: geographical distribution, liver flukes, molecular genotyping

Challenging current geographic distributions of fish-borne liver flukes in Laos and their implications for liver disease and cancer

Breanna Knight¹, Boulay Keokhamphavanh², Kelly Taggart¹, Sarah Keatley¹, Andrew Larkins¹, Bounnaloth Insisiengmay², Amanda Ash¹

¹Murdoch University, Australia; ²Laos Ministry of Health

Fish-borne liver flukes, particularly *Opisthorchis viverrini* and *Clonorchis sinensis*, represent significant public health concerns in Southeast Asia, estimated to affect 17 million people globally. These liver flukes are associated with severe health complications, including cholangiocarcinoma, and are classified as Neglected Tropical Diseases by the WHO. An apparent distinct geographic distribution exists for these flukes with only *C. sinensis* present in China and Vietnam while only *O. viverrini* is present in Laos. Current diagnostic methods in-country rely on the detection of liver fluke eggs through faecal microscopy, which cannot differentiate between these species or less pathogenic flukes such as *Haplorchis taichui*. Microscopy analysis of 3500 faecal samples from a 2022-2023 survey in Laos detected 2.9% small fluke eggs, with *O. viverrini* being the assumed liver fluke species in the region. However, genotyping results revealed 35.3% were *H. taichui*, 2% were *O. viverrini*, and 18.6% were *C. sinensis*. These findings challenge the long-standing assumption that *O. viverrini* is the predominant liver fluke in Laos. These findings could guide more targeted public health interventions and address the actual species distribution and associated health risks, ultimately improving disease control and reducing the burden of liver disease and cancer in the region.

ID: 134 / CP12: 6

Contributed abstract

Conference Topics: Education/Outreach, Helminthology

Keywords: Opisthorchis viverrini, health promotion, child, randomised-controlled trial, neglected tropical diseases

Examining the acceptability and impact of “The Magic Glasses: Opisthorchiasis” on schoolchildren’s knowledge, attitudes and practices surrounding Opisthorchis viverrini in the Lower Mekong Basin

Suji O'Connor

The Australian National University, Australia

Opisthorchis viverrini (OV), a foodborne trematode, is a major health concern in the Lower Mekong Basin, estimated to affect 10 million. Transmission occurs via consumption of raw or undercooked cyprinid fish. Acute cases are often asymptomatic, however chronic infection can cause cholangiocarcinoma, a bile duct cancer that is often fatal.

The primary control strategy for OV is mass drug administration, but this does not prevent future infection. Furthermore, hepatobiliary abnormalities caused by OV infection can persist after treatment. As such, alternative strategies are needed.

Children are an important group for health promotion and disease prevention. “The Magic Glasses” is a school-based educational cartoon that has demonstrated success in improving knowledge, attitudes and practices (KAP) surrounding soil-transmitted helminths in Asia. We devised “The Magic Glasses: Opisthorchiasis” with the aim of improving OV KAP among schoolchildren in the Lower Mekong region. This is the first “Magic Glasses” to target a new disease, and multiple countries.

A cluster-randomised trial was conducted in Cambodia, Lao PDR and Thailand in 2023-2024 to assess the acceptability of the cartoon and its impact on schoolchildren’s OV KAP.

This presentation will summarise study findings and their implications for scaling up “The Magic Glasses: Opisthorchiasis” in the Lower Mekong.

CP10.1: Drugs & Drug Resistance 5 min talks

Time: Wednesday, 02/July/2025: 12:15pm - 12:30pm · Location: Conference room 1

Session Chair: Jacinta Macdonald, Griffith University

Session Chair: Darren Creek, Monash University

ID: 132 / CP10.1: 1

Contributed abstract

Conference Topics: Apicomplexa Biology, Drugs, Malaria

Keywords: antimalarial, malaria, Plasmodium, mechanism of action

Characterisation of novel antimalarial chemotypes for which resistance is associated with PfCARL

Brad Sleebs^{1,2}, Madeline Dans^{1,2}, Qingmiao Zhou¹, Kyle Awalt^{1,2}, Stephen Brand³, Paul Jackson⁴

¹Walter and Eliza Hall Institute, Australia; ²University of Melbourne, Australia; ³Medicines for Malaria Venture, Geneva, Switzerland.; ⁴Global Public Health, Janssen R&D LLC, La Jolla, USA.

Malaria is a devastating disease caused by the *Plasmodium* parasite. Due to the threat of emerging drug resistance, the current arsenal of clinically used artemisinin combination therapies and drug candidates undergoing clinical assessment may not be sufficient to eliminate the disease. Thus, novel chemotypes that target multiple stages of the parasite lifecycle are required to continually populate the antimalarial clinical portfolio.

To contribute to the global effort to treat and eliminate malaria we have performed a high throughput screen of the EscuLab library of drug-like small molecules against the asexual stage of *P. falciparum* parasite. Several hit classes with unique scaffolds were identified and shown to exhibit sub-micromolar EC₅₀ values against asexual *P. falciparum* and did not display cytotoxicity towards human cell lines highlighting their attractiveness as starting points for antimalarial investigation.

This presentation will focus on the mechanism of action studies and the asexual stage characterisation of two unique hit scaffolds. PfCARL was established as the mechanism of resistance of the two novel scaffolds using forward genetic studies, phenotypic and protein export models, and drug-resistant clinical strains. The two new antimalarial classes may represent an alternative to KAF156 as a multistage antimalarial agent.

ID: 201 / CP10.1: 2

Contributed abstract

Conference Topics: Drugs

Keywords: histone deacetylase inhibitor, antimalarial, hydroxamic acid, malaria

Hydroxamic acid-based HDAC inhibitors as antimalarial drug leads: a structure activity relationship analysis

Wisam Dawood¹, Christian Anzenhofer², Jacinta Macdonald¹, Thomas Kurz², Kathy Andrews^{1,3}

¹Institute for Biomedicine and Glycomics, Griffith University, Queensland, Australia; ²Institut für pharmazeutische und medizinische Chemie, Heinrich-Heine Universität, Germany; ³School of Environment and Science, Griffith University, Nathan, Brisbane, Queensland, Australia

Malaria parasite drug resistance is driving the need for new antimalarials with novel mechanisms of action. Histone/lysine deacetylases (HDACs) are enzymes that reversibly modify the acetylation state of lysine residues on both histones and non-histone proteins and are validated drug targets for cancer and other diseases. HDACs are also prospective targets for malaria due to their essential role in regulating key processes in *Plasmodium* parasites, including transcription and developmental control. To better understand the structure activity relationship (SAR) of antiplasmodial HDAC inhibitors of the hydroxamic acids chemotype, we assembled a database of >700 compounds with data available on *in vitro* activity (IC₅₀) against asexual-stage *P. falciparum* parasites. SAR analyses compared *in vitro* potency, *P. falciparum* versus human cell Selectivity Indices, and physicochemical properties. These data provide new insights that will aid in the investigation of this chemotype for further development as drug leads for malaria.

ID: 147 / CP10.1: 3

Contributed abstract

Conference Topics: Apicomplexa Biology, Biochemistry, Drugs, Malaria

Keywords: Antimalarial; Drug resistance; Collateral sensitivity; Cipargamin

Protecting PfATP4-targeting antimalarials by exploring collateral sensitivity and roads to cipargamin resistance.

Ruijia Liang, Deyun Qiu, Adele Lehane

Australian National University, Australia

Combating malaria caused by *Plasmodium falciparum* is intertwined with overcoming drug resistance. The clinical candidates cipargamin and (+)-SJ733 kill parasites by inhibiting Na⁺-pump PfATP4. The G358S mutation in PfATP4 confers clinically-significant resistance to both compounds but increases parasite sensitivity to some other PfATP4 inhibitors. This suggests an opportunity to exploit collateral sensitivity to prevent parasite resistance. We investigated the ability of 'hypermutator' parasites to simultaneously acquire resistance to two different PfATP4 inhibitors. Resistance to the combination of cipargamin and (+)-SJ733 arose quickly via the PfATP4-G358S mutation, as expected. We also paired cipargamin with compounds that are more potent against PfATP4^{G358S} parasites than PfATP4^{WT} parasites. For one such combination, no viable parasites emerged, suggesting a high barrier to resistance for this pair. For a different combination, parasites acquired resistance via a L354V mutation in PfATP4. This mutation confers 300-fold resistance to cipargamin and protects parasites from cipargamin-induced dysregulation of cytosolic [Na⁺]. The L354V mutation is associated with an elevated resting [Na⁺] in the parasite and a growth defect in the blood stage. The study revealed that exploiting collateral sensitivity is an approach worth exploring for PfATP4 inhibitors, and uncovered a second avenue to high-level cipargamin resistance.

CP11.1: Immunology & Vaccination 5 min talks

Time: Wednesday, 02/July/2025: 12:15pm - 12:30pm · Location: Conference room 2

Session Chair: Li Jin Chan, The Walter and Eliza Hall Institute

Session Chair: Michael Duffy, University of Melbourne

ID: 160 / CP11.1: 1

Contributed abstract

Conference Topics: Immunology, Malaria, Vaccines

Keywords: RTS, S, malaria, vaccine, boosting, antibody

Declining antibody responses in children given the RTS,S malaria vaccine with repeated yearly booster doses

Alexander Harris^{1,2}, Liriye Kurtovic^{1,2}, Alassane Dicko³, Jean-Bosco Ouedraogo³, Daniel Chandramohan³, Brian Greenwood³, James Beeson^{1,2,4,5}, NCT04319380 Clinical trial team³

¹Burnet Institute, Australia; ²School of Translational Medicine, Monash University, Australia; ³RTS,S SMC clinical trial NCT04319380 team; ⁴Department of Medicine, Doherty Institute University of Melbourne, Australia; ⁵Department of Microbiology, Monash University, Australia.

There remains an urgent need for effective malaria interventions to reduce disease burden in children. Combining RTS,S vaccination with seasonal anti-malarial chemoprevention was shown to enhance efficacy against clinical malaria among children by ~72% over the first year compared to either intervention alone. However, over four years, efficacy of this combination steadily decreased despite annual boosters. RTS,S induces antibodies, which are associated with protection from clinical malaria. One antibody type, IgG, was shown to peak following primary vaccination, but became progressively lower after each annual booster. It is unknown what drives poor antibody responses to boosters, which likely varies between different antibody isotypes and antigenic targets. A deeper understanding is essential to inform the next generation of malaria vaccines with greater efficacy and longevity.

To address these knowledge gaps, we studied young children in West Africa who received RTS,S and subsequent boosters with or without seasonal chemoprevention in a phase-III clinical trial. We quantified the induction and boosting of antibodies and functional activity against two major subdomains of the vaccine antigen in plasma samples (n=1,929) collected over four years. We observed novel differences in the kinetics of antibodies to each subdomain, shedding light on why vaccine efficacy wanes over time.

ID: 228 / CP11.1: 2

Contributed abstract

Conference Topics: Epidemiology, Immunology, Malaria

Keywords: Malaria, *P. vivax*, antibodies, parasite clearance, epidemiology

Investigation of the relationship between naturally acquired antimalarial antibodies and the duration and clearance of ultra-low density *Plasmodium vivax* infections

Katherine O'Flaherty^{1,2}, Sophie Zaloumis¹, Rhea Longley^{3,4,5}, Merryn Roe², D. Herbert Opi², Kael Schoffer³, David Price⁶, Rupam Tripura^{5,7}, Chea Nguon⁸, Koukeo Phommason^{9,10}, Mayfong Mayxay^{7,11}, Paul Newton^{7,9}, Thomas Peto^{5,7}, James Callery⁵, Mehul Dhorda⁵, Nicholas Day^{5,7}, Arjen Dondorp^{5,7}, Eizo Takashima¹², Takafumi Tsuboi¹², Julie Simpson^{1,7}, James Beeson^{2,13,14}, Ivo Mueller^{3,4}, Nicholas White^{5,7}, Lorenz von Seidlein^{5,7}, Freya Fowkes^{1,2,15}

¹Centre for Epidemiology and Biostatistics, The University of Melbourne, Melbourne, Australia; ²Disease Elimination Program, Burnet Institute, Melbourne, Australia; ³The Walter and Eliza Hall Institute of Medical Research, Melbourne, Australia; ⁴Department of Medical Biology, The University of Melbourne, Melbourne, Australia; ⁵Faculty of Tropical Medicine, Mahidol University, Bangkok, Thailand; ⁶Department of Infectious Diseases, The University of Melbourne at the Peter Doherty Institute for Infection and Immunity, Melbourne, Australia; ⁷Centre for Tropical Medicine and Global Health, University of Oxford, Oxford, United Kingdom; ⁸National Centre for Parasitology, Entomology, and Malaria Control, Phnom Penh, Cambodia; ⁹Lao-Oxford-Mahosot Hospital-Wellcome Trust Research Unit, Vientiane, Lao PDR; ¹⁰Amsterdam Institute for Global Health & Development, Amsterdam, Netherlands; ¹¹Institute of Research and Education Development, University of Health Sciences, Vientiane, Lao PDR; ¹²Division of Malaria Research, Proteo-Science Center, Ehime University, Matsuyama, Japan; ¹³Departments of Medicine, Microbiology and Immunology, and Infectious Diseases, The University of Melbourne, Melbourne, Australia; ¹⁴Central Clinical School and Department of Microbiology, Monash University, Melbourne, Australia; ¹⁵Departments of Epidemiology and Preventative Medicine and Infectious Diseases, Monash University, Melbourne, Australia

Spontaneous clearance of subclinical peripheral *Plasmodium* spp. parasitemia, including *P. vivax*, occurs frequently in low transmission settings. Subclinical *P. vivax* infections contribute significantly to ongoing malaria transmission, therefore understanding the role of antibody mediated immunity in the spontaneous clearance of parasitemia is essential to the broader understanding of the epidemiology and transmission potential of *P. vivax*. We sought to identify IgG responses associated with clearance of peripheral *P. vivax* parasitemia. IgG against 30 *P. vivax* antigens was determined in participants of a nested cohort study of subclinical *Plasmodium* spp. infection in Laos (n=202) and Cambodia (n=150) sampled monthly for 12 months (n=3,041). *Plasmodium* spp. infections detected by ultrasensitive qPCR. Accelerated failure time models were used to determine the association between IgG and time to spontaneous clearance of peripheral *P. vivax* parasitemia. A total 293 subclinical *P. vivax* infections were detected. Spontaneous clearance was observed in 79% and 67% of participants in Laos and Cambodia, respectively. Anti-*P. vivax* IgG responses were higher in *P. vivax* infected compared to uninfected participants during the study period. Antigen-specific IgG responses were not associated with time to clearance of peripheral *P. vivax* parasitemia. Future investigations will include analysis of multi-antigen responses. These findings will further our understanding of immunity in the epidemiology of *P. vivax* infections.

ID: 217 / CP11.1: 3

Contributed abstract

Conference Topics: Immunology, Malaria, Vaccines

Keywords: Malaria; Immunity; Vaccines

Investigating heterologous prime/boost regimens for malaria vaccines

Lirive {Lydia} Kurtovic^{1,2,3}, Adam Thomas^{1,2}, D. Herbert Opi^{1,2,3}, James Beeson^{1,2,3}

¹Burnet Institute, Australia; ²Department of Immunology, Monash University; ³Department of Infectious Diseases, The University of Melbourne

Malaria remains a major global health burden, especially in young children under the age of 5 years. Current malaria vaccines consist of virus-like particles expressing the major surface antigen on malaria sporozoites, the circumsporozoite protein (CSP). However, they provide only modest vaccine efficacy against disease in target populations. Efforts to modify the vaccine antigen and/or platform are needed to achieve highly efficacious vaccines and eliminate malaria.

In collaboration with GeoVax Inc., we evaluated a novel malaria vaccine candidate using the modified vaccinia ankara (MVA) viral vector-based platform. We evaluated an MVA encoding the malaria CSP antigen in mouse immunogenicity studies. Three groups of mice (n=4 per group) were given a combination of the MVA with recombinantly expressed CSP, or MVA-prime/protein-boost, or protein-prime/MVA-boost. We assessed the magnitude, functionality, and durability of vaccine-induced antibodies, and found the protein-prime/MVA-boost regimen was significantly less immunogenic than the other regimens. This was most notable for functional responses such as C1q-fixation, which is an established mechanisms of immunity to sporozoites.

These data provide new insights into how heterologous prime/boost regimens can impact the quality of vaccine-induced antibodies. We have expanded this work to explore other MVA-based construct and mRNA-based vaccines encoding CSP and other malaria antigens.

CP13: Cell & Molecular Biology 15 min talks

Time: Wednesday, 02/July/2025: 1:30pm - 2:30pm · *Location:* Conference room 1

Session Chair: Fleur Sernee, The University of Melbourne

Session Chair: Paul Gilson, Burnet Institute

ID: 282 / CP13: 1

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Host-parasite interactions, Parasites of cats

Keywords: IFA imaging, macro software, metabolomics, tissue culture

Understanding metabolic sensing during *Toxoplasma* differentiation

Amber Simonpietri^{1,2}, Alessandro Uboldi^{1,2}, Nicholas Katris³, Malcolm McConville³, Christopher Tonkin^{1,2}

¹The Walter and Eliza Hall Institute of Medical Research, 1G Royal Parade, Parkville, Melbourne, Victoria, Australia, 3052;

²Department of Medical Biology, The University of Melbourne, 1G Royal Parade Parkville; ³Department of Biochemistry and Pharmacology, Bio21 Molecular Science and Biotechnology Institute, The University of Melbourne, Parkville, Australia

Toxoplasma gondii is an intracellular parasite found globally that establishes untreatable latent infection in muscle and neural tissue. It is not known what triggers the differentiation into this latent form or why it is resistant to current therapies. Our laboratory undertook a CRISPR screen to determine which genes were required for the utilization of glutamine or glucose *in vitro*. The results uncovered that a switch of carbon source can trigger differentiation, and identified several genes in central carbon metabolism, suggesting a link between these two processes. I utilised a newly reported human muscle cell line (KD3) to study the link between host and parasite central carbon metabolism and differentiation. Overall, we found that KD3 cells as compared to the canonical 'alkaline stress' model have significant benefits and reveal novel phenotypes. We generated several parasite mutant strains lacking genes involved in central carbon metabolism, multiple showed defective differentiation which was most pronounced in the KD3 myotubes. In particular we show that a glutamate dehydrogenase (GDH2) is particularly important for differentiation and localises to the parasite nucleus, suggesting a link to gene regulation. Together this work suggests that central carbon metabolism is linked to *Toxoplasma* differentiation in unappreciated ways.

ID: 283 / CP13: 2

Contributed abstract

Conference Topics: Apicomplexa Biology, Bioinformatics, Cell Biology, Genomics, Microscopy, Molecular Biology, Parasites of cats, Proteomics

Keywords: mass spectrometry, computational analysis, proteomics, cloning, microscopy

Role of Ubiquitination in *Toxoplasma* Differentiation

Karan Singh^{1,2}, Alessandro Uboldi^{1,2}, Amalie Jayawickrama^{1,2}, Simon Cobbold^{1,2}, Christopher Tonkin^{1,2}

¹The Walter and Eliza Hall Institute of Medical Research, Australia; ²The University of Melbourne, Australia

A quarter of the global population is chronically infected with *Toxoplasma gondii*. Following infection, *T.gondii* switches from fast-growing tachyzoites, to slow-growing bradyzoites characteristic of chronic infection. Both immune system and therapies fail to clear chronic infection. BFD1, a myb-like transcription factor regulates the initiation of differentiation. BFD1 is thought to be regulated post-transcriptionally, where protein levels accumulate upon a differentiation signal. Through a CRISPR screen, we identified an E3 ubiquitin ligase complex called the GiD complex, that regulates differentiation. We used mass spectrometry to map, for the first time, the global proteome during differentiation and showed that loss of GiD resulted in a loss of expression of known bradyzoite proteins, in a similar manner to loss of BFD1, suggesting a strong link of this E3 ligase in regulation of differentiation. We identified that GiD acts to regulate differentiation through the 3'-utr of the BFD1 transcript, likely by modulating translation repression of this master regulator. We also performed paired RNAseq to reveal proteins that are regulated post-transcriptionally and plan to use orthogonal methodologies to reveal substrates of the GiD. Overall, this study will provide the first evidence as to the role of ubiquitination during differentiation in *Toxoplasma* and other apicomplexan parasites.

ID: 169 / CP13: 3

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Malaria, Microscopy, Molecular Biology
Keywords: Expansion microscopy, Image analysis

Rhoptries, too small to see! How to overcome the diffraction limit using simple light microscopy.

Shamit Singla^{1,2}, **Sonja Frolich**^{1,2}, **Joshua Morrow**³, **Ghizal Siddiqui**³, **Danny Wilson**^{1,2,4}

¹Research Centre for Infectious Diseases, School of Biological Sciences, University of Adelaide, Adelaide, SA 5005 Australia.; ²Institute for Photonics and Advanced Sensing, The Braggs Building, University of Adelaide, Adelaide, SA 5005 Australia.; ³Drug Delivery, Disposition and Dynamics, Monash Institute of Pharmaceutical Sciences, Monash University, Parkville Campus, Parkville, Victoria, Australia; ⁴Burnet Institute, 85 Commercial Road, Melbourne, VIC 3004, Australia

To cause disease, malaria parasites need to invade host cells using specialised secretory organelles called the rhoptries. While we have a relatively good understanding of secreted rhoptry proteins, our knowledge of proteins that control rhoptry function is limited. Rhoptry proteins exposed to the cytoplasm are in the right place at the right time to facilitate rhoptry secretion due to their ability to mediate interactions with other parts of the cell. However, the rhoptries are too small to be observed using conventional light microscopy, making it challenging to determine rhoptry protein localisation or detect changes in rhoptry structure. We have applied a newly developed technology termed expansion microscopy that overcomes this problem by increasing the size of the parasite by ~4.5x. In conjunction with advanced image analysis, we have used this technique to create a 3D map of the parasite to determine the location of individual protein molecules and visualise small changes in the rhoptry structure. Early results suggest that the essential rhoptry protein CERL1 has a discontinuous distribution along the rhoptry surface, potentially highlighting areas where key interactions occur. These findings will help us better define rhoptry biology and understand the role of this organelle during host cell invasion.

ID: 199 / CP13: 4

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Host-parasite interactions, Malaria, Microscopy
Keywords: inner membrane complex, gametocyte development, invasion

Functional characterisation of the GAPM proteins in asexual and sexual stage development

Katrina Larcher^{1,2}, **Yuri Shibazaki**^{1,2}, **Cindy Evelyn**³, **Niall Geoghegan**³, **Kelly Rogers**³, **Sash Lopatnicki**^{1,2}, **Hayley Buchanan**^{1,2}, **James McCarthy**^{1,2}, **Matthew Dixon**^{1,2}

¹Department of Infectious Diseases, Doherty Institute, The University of Melbourne, Victoria; ²Infection and Global Health Division, Walter and Eliza Hall Institute, Victoria; ³Centre for Dynamic Imaging, Walter and Eliza Hall Institute, Victoria

Plasmodium falciparum uses a highly conserved organelle to facilitate transition between different cellular niches throughout its lifecycle. The inner membrane complex (IMC) acts as a scaffold for cytoskeletal components to provide cell structure and rigidity and acts as an anchor for the actin-myosin motor essential for motility and invasion. Despite being well studied we know little about the organisation and assembly of the IMC and the function of many of its resident proteins. In this work, we used a gene tagging and conditional knockout (cKO) approach, integrated with super-resolution microscopy (Lattice Light Sheet Microscopy and 3D-SIM) and proteomic analyses, to determine the location and function of all three Glideosome Associated Protein(s) with Multiple membrane spans (GAPM) within the IMC. Quantitative proteomics indicated that loss of either GAPM1, GAPM2 or GAPM3 causes global dysregulation of many IMC components and key invasion ligands. This manifested in the inability for cKO merozoites to reinvade new red blood cells. Additionally, absence of GAPM1/2/3 prevented gametocytes from elongating and maturing beyond stage-three. cKO parasites showed abnormal tubulin deposits and incorrect coupling of the IMC to the microtubule network. This work proposes the GAPM protein family acts as key anchors of cytoskeletal components to the IMC.

CP14: Helminth Biology 15 min talks

Time: Wednesday, 02/July/2025: 1:30pm - 2:45pm · Location: Conference room 2

Session Chair: Sarah Preston, Federation University Australia

Session Chair: Anson Koehler, University of Melbourne

ID: 290 / CP14: 1

Contributed abstract

Conference Topics: Fasciolosis/Liver fluke, Immunology, Proteomics
Keywords: *Opisthorchis viverrini*, human liver fluke infections

Immunomics-guided biomarker discovery for human liver fluke infection and infection-associated cholangiocarcinoma

Alex Loukas¹, **Lakkhana Sadaow**², **Rutchanee Rodpai**², **Michael Smout**¹, **Rie Nakajima**³, **Patcharaporn Boonroumkaew**², **Philip L Felgner**³, **Wanchai Maleewong**², **Paul Brindley**⁴, **Pewpan Intapan**²

¹Australian Institute of Tropical Health and Medicine, James Cook University, Cairns, QLD, Australia; ²Department of Parasitology, Faculty of Medicine, and Mekong Health Science Research Institute, Khon Kaen University, Khon Kaen, Thailand.; ³Vaccine R&D Center, Department of Physiology and Biophysics, University of California Irvine, Irvine, CA, USA; ⁴Department of Microbiology, Immunology and Tropical Medicine, School of Medicine and Health Sciences, George Washington University, Washington, DC, USA

Sensitive diagnostics are needed to improve management and surveillance of the human liver fluke infections, opisthorchiasis and clonorchiasis. These flukes are considered by the WHO to be group 1 biological carcinogens because of the undisputed association between liver fluke infection in East Asia and bile duct cancer, or cholangiocarcinoma (CCA). We generated and screened an *Opisthorchis viverrini* recombinant secreted proteome to identify antibody biomarkers of liver fluke infection and CCA with sera from study participants in endemic populations and evaluate their utility as point-of-care immunochromatographic tests (PoC-ICTs). Two of the most promising antigens from the proteome array screen, P1 and P9, were each incorporated into PoC-ICTs to further validate their diagnostic performance. The P9-IgG4 PoC-ICT was superior amongst the single recombinant antigen tests for diagnosing fluke infection as well as fluke-induced CCA, and out-performed

parasite crude extract-IgG ICTs. Here we identified two biomarkers of *O. viverrini* infection and infection-associated CCA that could form the basis of novel antibody serodiagnostic tests for human liver fluke infection and associated cancer.

ID: 195 / CP14: 2

Contributed abstract

Conference Topics: Bioinformatics, Fasciolosis/Liver fluke, Host-parasite interactions, Immunology

Keywords: MicroRNAs, Fasciola hepatica, host-parasite interaction

The non-coding RNA landscape in macrophages is altered by liver flukes to promote parasite survival and gain immune dominance

Dayna Sais¹, Sumaiya Chowdhury², Sheila Donnelly^{2,3}, Nham Tran¹

¹School of Biomedical Engineering, Faculty of Engineering and Information Technology, University of Technology Sydney, Sydney, NSW, Australia; ²School of Life Sciences, Faculty of Science, University of Technology Sydney, Sydney, NSW, Australia; ³School of biological and chemical sciences, University of Galway, Galway, Ireland

Helminths are adept at modulating host immunity to enable long term survival. Increasing evidence points to non-coding RNAs (ncRNAs) such as microRNAs (miRNAs), as key regulators of host-pathogen interactions. We propose that the liver fluke *Fasciola hepatica* exploits host miRNA machinery to suppress immune responses by transferring parasite-derived miRNAs to be taken up by host macrophages. Mice were infected with *F. hepatica*, and peritoneal macrophages were isolated 6h, 18h and 5 days post-infection for miRNA and total RNA sequencing. Several parasite miRNAs (fhe-miR-277b-3p, fhe-miR-71a-5p, fhe-miR-125b-5p, fhe-miR-125a-5p) were enriched in the macrophages at 6h and 18h but absent by day 5. Target analysis indicated these miRNAs suppress host genes (Hif1a, Nod1, Nlrp6 and P2rx7) associated with M1 macrophage polarisation and pro-inflammatory signalling. In parallel, a shift in host ncRNA (miRNA and long non-coding RNA) expression was observed, with predicted targeting of genes including Adar1, Igf1 and Plex, implicated in M2 polarisation and tissue repair. This dual modulation suggests a coordinated ncRNA-driven program that dampens host defence, while promoting tissue homeostasis, facilitating parasite persistence. Our findings reveal a complex layer to host-parasite communication via ncRNAs, offering insights into immune evasion strategies and highlighting potential targets for intervention.

ID: 101 / CP14: 3

Contributed abstract

Conference Topics: Host-parasite interactions, Immunology

Keywords: Macrophage, hookworm, Ac-FAR-2, RNA sequence, arthritis

Hookworm-derived biologic Ac-FAR-2 binds arachidonic acid on macrophages to reduce inflammation in a mouse model of rheumatoid arthritis

Suchandan Sikder¹, Haleagrahara Nagaraja², Connor McHugh¹, Darren Pickering¹, Kim Miles¹, Rachael Ryan¹, Manoharan Kumar¹, Matt Field¹, Paul Giacomini¹, Roland Ruscher¹, Alex Loukas¹

¹Australian Institute of Tropical Health and Medicine, James Cook University, Cairns, Australia; ²College of Medicine and Dentistry, James Cook University, Townsville, Australia

Macrophages are key players in driving inflammation in autoimmune diseases and are thus a target for the development of novel therapeutics. Hookworms are parasitic nematodes that live for years in the intestines of mammalian hosts. To facilitate parasitism, hookworms secrete proteins that have evolved to modulate human immune system. We have discovered that a recombinant fatty-acid and retinol-binding protein (Ac-FAR-2) from the secretome of the hookworm *Ancylostoma caninum* potently attenuates inflammatory cytokine production by cultured human peripheral blood mononuclear cells and THP-1 monocyte-derived macrophages. Moreover, impaired macrophage function was linked with reduced co-stimulatory marker expression and associated reduction of T-cell proliferation in co-cultures. Mechanism of action studies demonstrated that Ac-FAR-2 interacts with surface molecules of macrophages and impedes arachidonic acid-prostaglandin E2 pathway. Transcriptome profiling and pathway analysis revealed that Ac-FAR-2 downregulates genes involved in arachidonic acid, prostaglandin E2 and NF-κB pathway signaling, chemokinetic and inflammatory functions, and inflammasome-related pro-inflammatory molecules in LPS-stimulated human macrophages, providing insight into its mechanism of action. Finally, treatment with Ac-FAR-2 reduced clinical and histological scores of collagen-induced arthritis modelling autoimmune rheumatoid arthritis in mice. Our data suggests Ac-FAR-2 as a novel anti-inflammatory drug candidate for the treatment of macrophage-dependent autoimmune conditions such as rheumatoid arthritis.

ID: 185 / CP14: 4

Contributed abstract

Conference Topics: Biochemistry, Host-parasite interactions, Immunology, Molecular Biology

Keywords: Therapeutic, Inflammatory disease, Hookworm, Recombinant proteins

Necator americanus recombinant proteins as novel therapeutics for inflammatory disease

Connor McHugh, Suchandan Sikder, Kim Miles, Yide Wong, Maggie Veitch, Maxine Smith, Stephanie Ryan, Darren Pickering, Roland Ruscher, Paul Giacomini, Alex Loukas

Australian Institute of Tropical Health and Medicine, James Cook University

Experimental and naturally acquired human helminth infections have been shown to impart varying degrees of protection against a suite of inflammatory diseases. The proclivity of helminths to regulate their host immune response and suppress inflammation is attributed to the active release of excretory/secretory proteins (ESP) into the host tissues. Experimental infection of humans with helminths presents significant complications as a therapeutic modality due to their complex lifecycles, likely poor adoption, and unavoidable side effects in some subjects. As such, there is now considerable interest in identifying bioactive ESPs and making them more drug-like. We therefore created a recombinant library of *N. americanus* ESPs from both the adult and larval stage secretomes and are screening the library in a range of *in vitro* and *in vivo* assays to identify proteins with potent immunoregulatory properties. Thus far, we have identified potential candidate proteins that could form the basis of novel therapeutics for treating type 2 diabetes, inflammatory bowel disease and rheumatoid arthritis based on their *in vitro*

and/or *in vivo* bioactivities. Using *in silico* screening method, potential binding partners and MOA are being elucidated, facilitating pre-clinical development.

ID: 103 / CP14: 5

Contributed abstract

Conference Topics: Helminthology, Host-parasite interactions, Immunology, Vaccines

Keywords: Hookworm, subunit vaccine, B cell memory

Rational discovery of a novel hookworm subunit vaccine based on immunomic profiling of irradiated parasite-induced protective immunity in humans

Eti Sarkar¹, Connor Mchuge¹, LI Liang², Yide Wong¹, Philip Felgner², Paul Giacomin¹, Suchandan Sikder¹, Alex Loukas¹

¹James Cook University, Australia; ²School of Medicine, University of California Irvine, USA

Hookworms infect half-a-billion people, causing approximately 2.1 million disability-adjusted life-years lost. While anthelmintic drugs are available, they do not prevent reinfection. A vaccine could provide long-term immunity, but none currently exist for human hookworm infections. We recently demonstrated proof-of-concept that human subjects can be safely vaccinated with irradiated hookworm larvae, eliciting humoral and cellular immune responses that protect against challenge infection with non-irradiated parasites. We identified IgG targets in vaccinated individuals containing recombinant proteins sourced from the secretome of *Necator americanus*. We characterized the immunodominant *N. americanus* proteins and identified their orthologs in the rodent hookworm *Nippostrongylus brasiliensis*. These *Nb* proteins were tested for efficacy in a mouse challenge model. Our lead vaccine candidate provided robust protection against infection, evidenced by >90% reductions in intestinal worm burden and fecal egg counts, and elevated antigen-specific IgG levels. Vaccination also stimulated IgG¹⁺/IgA⁺/IgE⁺ B cells suggesting protection against gut immunity and systemic immunity. The presence of IgG¹⁺/IgA⁺ memory B cells in both the lymph nodes and spleen indicates potential long-term immunity against reinfection. Our findings suggest that a *Nb* L3 protein homolog in *Na* could serve as a promising subunit vaccine candidate targeting the human hookworms.

CP15: Pet Parasites 15 min talks

Time: Wednesday, 02/July/2025: 1:30pm - 3:00pm · *Location:* Conference room 3

Session Chair: Ryan O'Handley, Adelaide University

Session Chair: Bonny Cumming, AMRRIC

ID: 288 / CP15: 1

Contributed abstract

Conference Topics: Helminthology, Parasites of dogs, Veterinary Parasitology, Zoonoses

Keywords: *Ancylostoma caninum*, *Uncinaria stenocephala*, dogs helminths

Molecular detection of canine hookworms in faeces collected from urban parks across New Zealand

Madisson Nam¹, Patsy A. Zendejas-Heredia¹, Ushani Atapattu¹, Carl Eden², Maureen Forsyth², Sara Ashby², Frédéric Beugnet³, Neil D. Young¹, Robin B. Gasser¹, Rebecca Traub⁴, Vito Colella¹

¹Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Victoria 3010, Australia; ²Boehringer Ingelheim Animal Health, Mount Wellington 1060, New Zealand; ³Boehringer Ingelheim Animal Health, 69007 Lyon, France; ⁴City University of Hong Kong, Tat Chee Avenue, Kowloon 999077, Hong Kong

Dogs are family members in more than one-third of households across New Zealand. These companion animals are susceptible to a range of pathogens, including helminths. Among these pathogens, hookworms are blood-feeding nematodes, capable of causing disease in dogs and potentially infecting humans. However, little is known about the nature and extent of hookworm contamination in the environment, urban parks, and the parasite species present. This study addressed this knowledge gap by using a multiplex quantitative PCR assay to detect and identify hookworm species in dog faeces collected from urban parks in New Zealand. It also evaluated risk factors potentially associated with canine hookworm contamination of urban parks. A total of 739 faecal samples from 123 urban parks were collected across NZ. *Ancylostoma caninum* and *Uncinaria stenocephala* were detected in 18.7% (23/123; 95% CI 12.8-26.5) of sampled parks. Dog population density ($p = 0.049$) and distance from the nearest city centre ($p = 0.05$) were significantly associated with the probability of a park being contaminated with canine hookworms. These findings highlight the importance of implementing effective management strategies to prevent hookworm infections in dogs and reduce the risk of zoonotic transmission in NZ's urban park environments.

ID: 206 / CP15: 2

Contributed abstract

Conference Topics: Apicomplexa Biology, Livestock Parasites, Parasites of cats, Protozoa, Veterinary Parasitology

Keywords: *Toxoplasma gondii*, Infection Model, Lifecycle

Establishing a complete *Toxoplasma gondii* *in vivo* model of infection using a South Australian field isolate

Ornella Romeo^{1,2}, Connor Bury³, Maria Gancheva^{1,2,3}, Milton McAllister³, Ryan O'Handley³, Danny Wilson^{1,2}

¹Research Centre for Infectious Diseases, School of Biological Sciences, The University of Adelaide, 5005, SA, Australia;

²Institute for Photonics and Advanced Sensing (IPAS), The University of Adelaide, 5005, SA, Australia; ³School of Animal and Veterinary Sciences, University of Adelaide, Roseworthy Campus, SA 5371

The complex lifecycle of the apicomplexan parasite *Toxoplasma gondii* consists of replicative tachyzoites, semi-dormant bradyzoites that reside in tissue cysts of the host and oocysts excreted into the environment by definitive host felids. However, obtaining these morphologically distinct stages requires access to felid and intermediate animal hosts, greatly limiting the ability to establish the full lifecycle for clinical investigations. Here, we present a South Australian field isolate, derived from a seropositive mouse on Kangaroo Island (TgKIM1), and demonstrate its successful adaptation across the full lifecycle. Using

cultured TgKIM1 tachyzoites, we established long-term infection in Swiss mice and produced bradyzoites within brain tissue within 49 days post-infection. Clinical symptoms of disease, including ruffled coats and weight loss, were minimised with prophylactic antimicrobial treatment. TgKIM1 bradyzoite-infected tissue was harvested from the Swiss mice and fed to seronegative cats resulting in the successful production of oocysts shed in faeces across 9 days. The successful generation of TgKIM1 oocysts allows replication of the natural infection pathway, critical for challenge models for livestock vaccine research. This model establishes the full lifecycle of an Australian *Toxoplasma gondii* field isolate that can be used for understanding parasite biology, therapeutic development and clinical trials.

ID: 198 / CP15: 3

Contributed abstract

Conference Topics: Livestock Parasites, Parasites of cats, Protozoa, Veterinary Parasitology

Keywords: Toxoplasma gondii, Sheep, Serology, Pathology

Presenting an oocyst challenge profile for a South Australian field isolate of *Toxoplasma gondii* in pregnant Merino ewes.

Connor Bury^{1,2}, Ornella Romeo¹, Maria Gancheva^{1,2}, Danny Wilson¹, Ryan O'Handley²

¹Research centre for infectious diseases, School of Biological Sciences, University of Adelaide, Adelaide 5005, South Australia; ²School of Animal and Veterinary Sciences, University of Adelaide, Roseworthy 5371, South Australia

Toxoplasma gondii (*T. gondii*) is a protozoan parasite of the cat, with a vast range of intermediate hosts and is of concern for its clinical manifestation as toxoplasmosis. The reproductive failure associated with exposure to *T. gondii* is of particular concern in sheep (*Ovis aries*), with each 10% increase in seroprevalence seeing an approximate 5% decrease in lamb marking. *T. gondii* is widespread amongst South Australian sheep with flock-level seroprevalence approaching 100%. So too is the feral cat definitive host, responsible for environmental contamination with orally infective oocysts. Using a field isolate derived from a seropositive mouse on Kangaroo Island (*TgKIM1*), we successfully induced oocyst production from cats and performed a sporulated oocyst challenge in pregnant Merino ewes as part of an ongoing vaccine trial. Previously seronegative Merino ewes scanned in-lamb experienced abortive events 8-12 days post oocyst challenge. We performed serology to assess both the maternal humoral response to challenge and the in-utero vertical transmission of the parasite via detection of *T. gondii*-specific IgG. Through pathological investigation of aborted foetal and placental tissue and comparison with serological assessment of in-utero parasite transmission, we demonstrated that South Australian *T. gondii* strain *TgKIM1* is an abortive agent in sheep.

ID: 144 / CP15: 4

Contributed abstract

Conference Topics: Parasites of companion animals, Parasites of dogs, Veterinary Parasitology

Keywords: Anthelmintics, Canine health, Deep sequencing, Greyhounds, Multiple anthelmintic drug resistant (MADR)

Benzimidazole resistance in canine hookworm in Australia

Swaid Abdullah¹, Thomas Stocker², Hyungsuk Kang¹, Ian Scott³, Douglas Hayward⁴, Susan Jaensch⁴, Michael P Ward², Malcolm K Jones¹, Andrew C Kotze¹, Jan Slapeta²

¹The University of Queensland, Australia; ²The University of Sydney, Australia; ³Massey University, New Zealand; ⁴Vetnostics NSW, Australia.

Canine hookworm (*Ancylostoma caninum*) is a common parasite in dogs that can also affect humans. In the USA, this parasite has shown resistance to multiple deworming drugs. We investigated benzimidazole resistance in *A. caninum* from dogs in Australia and New Zealand using advanced DNA sequencing techniques. Our study found that 90% of the hookworm samples were *A. caninum*, while 11% were another species, *Uncinaria stenocephala*. We discovered two mutations (F167Y and Q134H) in *A. caninum* that are linked to drug resistance, with 49% and 67% of samples showing these mutations, respectively. Notably, the F167Y mutation was also found in *U. stenocephala* for the first time. No other common resistance mutations were detected. Egg hatch tests confirmed a strong link between the F167Y mutation and increased drug resistance and about 14% of dogs had a high level of this mutation, indicating widespread resistance. To combat this, we recommend a risk-based approach to limit unnecessary use of deworming drugs.

ID: 152 / CP15: 5

Contributed abstract

Conference Topics: One Health, Parasites of companion animals, Parasites of dogs

Keywords: rat lungworm, *Angiostrongylus cantonensis*, qPCR, ELISA, Australia

Climate driven emergence of canine neural angiostrongyliasis in eastern Australia, 2020-2024

Phoebe Rivory¹, Rogan Lee², Michael P. Ward¹, Jan Slapeta¹

¹The University of Sydney, Australia; ²Westmead Hospital, Australia

Neural angiostrongyliasis (NA), caused by the rat lungworm (*Angiostrongylus cantonensis*), is an emerging disease on Australia's east coast, with cases increasing since 2010. This study examined the diagnosis, genetic diversity, and environmental factors influencing canine NA (CNA). We analysed cerebrospinal fluid from 180 suspected cases (2020-2024) using qPCR, confirming 93 infections. Most cases were near Brisbane and Sydney, peaking in 2022 (32 cases). Statistical modeling showed CNA occurrence is linked to rainfall (1 and 10-12 month lags) and temperature changes (5-7 month lags). Genetic analysis identified Ac13 as the dominant haplotype. Comparison with an ELISA test showed substantial agreement ($\kappa = 0.66$). Many cases likely go undiagnosed, making NA a significant ongoing health concern in Australia.

ID: 192 / CP15: 6

Contributed abstract

Conference Topics: Education/Outreach, Parasites of companion animals, Veterinary Parasitology

Keywords: informed consent, videos, accessibility, knowledge translation

Enhanced Understanding: Using Educational Videos to Support the Informed Consent Process

Jessica Hoopes, Bonny Cumming

Animal Management in Rural and Remote Indigenous Communities Ltd (AMRRIC), Australia

Voluntary informed consent is a fundamental ethical requirement in parasitology research. It ensures participants understand the study's purpose, procedures, and potential risks and benefits for themselves, their pets, and the community. Beyond ethics, the consent process is also an opportunity to raise community awareness and understanding of research topics.

However, traditional written consent forms often fall short. These documents frequently use complex language and technical jargon that can confuse participants, particularly those from marginalised or vulnerable populations. Language barriers and low literacy levels can further hinder comprehension, leaving participants feeling pressured to consent without fully understanding the study.

To address these challenges, researchers must adopt clearer communication and innovative methods. One effective approach is the use of educational videos to complement written materials. Visual aids simplify complex information, accommodate diverse learning styles, and make the consent process more accessible. These tools enhance participant understanding, helping ensure truly informed consent.

Such videos can also be produced with minimal resources, making them a cost-effective way to support both informed consent and broader community education. By integrating accessible language and visuals, this approach promotes ethical research practices and empowers participants with the knowledge they need to make informed decisions.

CP13.1: Cell & Molecular Biology 5 min talks

Time: Wednesday, 02/July/2025: 2:30pm - 3:00pm · Location: Conference room 1

Session Chair: Fleur Sernee, The University of Melbourne

Session Chair: Paul Gilson, Burnet Institute

ID: 154 / CP13.1: 1

Contributed abstract

Conference Topics: Apicomplexa Biology, Malaria, Molecular Biology

Keywords: malaria, nanobody, structural biology, fertilisation, mosquito transmission

Nanobodies to a HAP2-like protein in malaria parasite fertilisation

Li Jin Chan^{1,2}, Jill Chmielewski^{1,2}, Melanie Dietrich^{1,2}, Frankie Lyons^{1,2}, Amy Adair¹, Mikha Gabriela^{1,2}, Wai-Hong Tham^{1,2,3}

¹The Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria, Australia.; ²Department of Medical Biology, The University of Melbourne, Parkville, Victoria, Australia.; ³Research School of Biology, The Australian National University, Canberra, ACT, Australia.

Malaria remains a devastating global parasitic disease, causing 619,000 deaths annually, with 80% of deaths in children under five. To reduce the malaria burden in communities, we need vaccines that block transmission. By inducing antibodies against the sexual stages of the parasite within the mosquito, we can prevent mosquito infection and halt subsequent transmission to humans.

Hapless 2 (HAP2) is an ancient fusion protein that mediates gamete fusion during sexual reproduction across diverse eukaryotic branches. In *Plasmodium*, disruption of hap2 abolishes the ability of male and female gametes to fuse. *Plasmodium* also encodes two structurally related proteins: HAP2p, which is also essential for gamete fusion, and an uncharacterised protein that we refer to as HAP2-like.

We aim to investigate whether HAP2-like is involved in *Plasmodium* fertilisation and whether it functions as a fusion protein. To this end, we have recombinantly expressed the soluble *Plasmodium vivax* HAP2-like protein. Using a nanobody discovery platform, next-generation sequencing, and a dedicated bioinformatics pipeline, we have generated and characterised a panel of nanobodies targeting *P. vivax* HAP2-like. These nanobodies will be tested in transmission-blocking assays. We also aim to determine high-resolution structures of *P. vivax* HAP2-like to gain mechanistic insights into its function.

ID: 246 / CP13.1: 2

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Malaria

Keywords: sugar transporter, SWEET, mutualism, parasitism, *P. berghei*

When SWEETs turn sour: a hypothesis for the origin(s) of parasitism in Apicomplexa

Joana Ferreira Costa¹, Vanessa Mollard¹, Mary-Louise Wilde¹, Giada Tortorelli^{1,2}, Eleanor Nicholson¹, Geoff I. McFadden¹

¹School of BioSciences, The University of Melbourne, Melbourne, Australia; ²Hawai'i Institute of Marine Biology, University of Hawai'i, Kaneohe, United States of America

The Myozoa supergroup includes both apicomplexan parasites like *Plasmodium* and photosynthetic dinoflagellates, some of which form symbioses with animal hosts. In mutualistic relationships, such as those between dinoflagellate algae and corals, metabolic exchange is key: the algae export photosynthetically derived glucose to their host using SWEET (Sugars Will Eventually be Exported Transporter) proteins, and in return, receive nitrogen and carbon. SWEETs are passive, bidirectional sugar transporters with seven transmembrane domains. This mutualistic model led us to hypothesise that apicomplexan SWEETs could mediate sugar uptake from the host. We identified a SWEET gene (*PbSWEET*) in the rodent malaria parasite *Plasmodium berghei* and generated a knockout line. Surprisingly, *PbSWEET* was dispensable across the entire life cycle, including blood, mosquito, and liver stages, making it one of a handful of malaria parasite genes with no observable knockout phenotype. This suggests that *PbSWEET* may be functionally redundant, consistent with prior reports that a 12-transmembrane hexose transporter mediates glucose uptake during blood stages. We are currently characterising *PbSWEET*'s

localisation and substrate specificity to better understand its potential contribution to the evolution of parasitism in Apicomplexa.

ID: 231 / CP13.1: 3

Contributed abstract

Conference Topics: Bioinformatics, Cell Biology, Genomics, Molecular Biology, Protozoa, Proteomics

Keywords: *Giardia duodenalis*, Evolutionary Biology, Transcriptomics, Splicing, Gene Regulation

Insights into the eukaryotic-emerged spliceosome and spliceosomal introns in early-diverging protist pathogen *Giardia duodenalis*

Nic Risteovski¹, Balu Balan¹, Aaron Jex^{1,2}

¹Walter and Eliza Hall Institute for Medical Research, Parkville, Victoria, Australia; ²Faculty of Science, The University of Melbourne, Parkville, Victoria, Australia

Spliceosomal splicing is a eukaryotic-originated process involving the processing of *cis*-acting elements (spliceosomal introns and exons) within pre-mRNA transcripts by *trans*-acting factors (spliceosomal proteins and snRNAs). Spliceosomal (canonical) splicing is an ancient driver of eukaryotic complexity, having emerged before the Last Eukaryotic Common Ancestor (LECA). Studies of splicing in yeast are often used to understand the evolution of eukaryotic splicing, however, there are extant organisms more basal than yeast. *As protists evolved more than 500 million years prior to yeast, exploring splicing in Giardia duodenalis*, an early-diverging eukaryote, may offer insights into the evolutionary origins of the spliceosome and spliceosomal introns. *Cis*-splicing elements and *trans*-splicing factors in *Giardia* are understudied. We conducted a comprehensive bioinformatic analysis of annotated intronic and proteomic data from the basal protist *Giardia* to complex protist *Plasmodium falciparum*, in addition to higher-branching model eukaryotes. Our preliminary analyses suggest that *Giardia* likely performs non-canonical splicing; spliceosomal proteins and introns in *Giardia* are minimal, and splicing mechanisms are divergent. Our study represents one of the first attempts to comprehend how spliceosomal splicing developed in early eukaryotes and has since expanded, contributing to the diversification of eukaryotic lineages.

ID: 286 / CP13.1: 4

Contributed abstract

Conference Topics: Apicomplexa Biology, Malaria

Keywords: malaria parasite biology

Investigating Cytosolically-exposed Rhoptry Interacting Proteins (C-RIPS) in *P. falciparum* sporozoites

Leonhard Satrio Arinanto

University of Melbourne, Australia

This research project investigates the roles of cytosolically-exposed rhoptry-interacting proteins (C-RIPs) during *Plasmodium falciparum* sporozoite invasion of Anopheles mosquito salivary glands and human liver cells. In particular, my work focuses on the proteins CERL1 and CERL2, which play critical roles in regulating rhoptry secretion—a vital process for host cell invasion. In asexual blood stages, disruption of these proteins has been shown to impair the secretion of essential invasion-related proteins, highlighting a potential shared invasion mechanism between merozoites and sporozoites. By employing expansion microscopy technique and super-resolution microscopy, along with molecular assays such as semi-quantitative PCR and proximity labeling via BioID, my study aims to provide deeper insight into malaria parasite biology and identify novel therapeutic targets.

ID: 146 / CP13.1: 5

Contributed abstract

Conference Topics: Apicomplexa Biology, Malaria, Molecular Biology, Vaccines

Keywords: *Plasmodium falciparum*, Malaria, Merozoite, MSP, MSP4

Defining the Role of MSP4 in *Plasmodium falciparum* Merozoite Invasion and Its Potential as a Therapeutic Target

Jarryd Tiu^{1,2}, Jill Chmielewski^{1,2,3}, Maria Gancheva^{1,2}, Danny Wilson^{1,2,3}

¹Research Centre for Infectious Diseases, School of Biological Sciences, University of Adelaide, Adelaide 5005, South Australia, Australia; ²Institute for Photonics and Advanced Sensing, University of Adelaide, Adelaide 5005, South Australia, Australia; ³The Walter and Eliza Hall Institute of Medical Research, Parkville 3052, Victoria, Australia

The *Plasmodium* merozoite surface is a dense, molecularly complex interface critical for host red blood cell (RBC) invasion. While many merozoite surface proteins (MSPs) remain poorly characterised, their putative roles in early attachment and invasion are increasingly called into question. Among these, MSP4 – a glycosylphosphatidylinositol-anchored protein, has been proposed as a prospective vaccine candidate in the most lethal malaria parasite, *P. falciparum*. Using conditional knockout (cKO) lines, we demonstrated that *PfMSP4* is indispensable for *P. falciparum* proliferation. We investigated this proliferation defect using live-cell microscopy of cKO parasites, revealing a pronounced defect in invasion efficiency, positioning MSP4's function post egress but prior to RBC entry. To resolve whether *PfMSP4* has a role in mediating merozoite attachment to the RBC, we are employing optical tweezer-based force spectroscopy, a novel approach to quantify merozoite-RBC interaction strength. Together, these findings will define the role of MSP4 in *P. falciparum* merozoite invasion of the host RBC and provide tools to investigate *PfMSP4* as a therapeutic target.

CP14.1: Helminth Biology 5 min talks

Time: Wednesday, 02/July/2025: 2:45pm - 3:00pm · Location: Conference room 2

Session Chair: Sarah Preston, Federation University Australia

Session Chair: Anson Koehler, University of Melbourne

ID: 130 / CP14.1: 1

Contributed abstract

Conference Topics: Helminthology, Vaccines

Keywords: Schistosoma, mansoni, schistosomiasis, vaccine

A novel multi-domain *Schistosoma mansoni* vaccine

Alexander Tynan¹, Shokoofeh Shamsi², Justin Roby³, Bernd Kalinna¹

¹Rural Health Research Institute, Australia; ²School of Agricultural, Environmental and Veterinary Sciences, Charles Sturt University, New South Wales, Australia; ³Gulbali Institute, Wagga Wagga, New South Wales, Australia

Schistosomiasis impacts over 200 million people globally, with treatment limited to one drug that is facing issues like drug resistance and failure to prevent reinfection. This study aims to develop a multi-domain vaccine that targets key proteins in *Schistosoma mansoni*, enhancing immune response and efficacy.

Five antigens—FABP, TPI, TSP, Calpain, and Cathepsin B—were combined into a single protein construct through epitope prediction and structural modelling. The construct is expressed in *E. coli* and yeast systems, with immunogenicity assessed in mice through antibody and cytokine profiling. Using tools like AlphaFold, the 3D structure was optimised to closely mimic native proteins, improving immune recognition potential. This multivalent approach provides a promising pathway towards sustainable schistosomiasis control.

ID: 117 / CP14.1: 2

Contributed abstract

Conference Topics: Bioinformatics, Cell Biology, Host-parasite interactions, Immunology, Molecular Biology, Proteomics

Keywords: Galectins, Immunomodulation, Hookworm, Whipworm, Inflammation

Galectin 3 and 9 homologues of gastrointestinal soil transmitted helminths and the search for anti-inflammatory molecules

Elizabeth Mullens¹, Gemma Zerna², Rob Bischof¹, Farah Ahmady³, David Piedrafita¹, Sarah Preston¹

¹Federation University Ballarat, Australia; ²Latrobe University Australia; ³Fiona Eley Cancer Research Institute Ballarat

The search for novel treatments for chronic inflammatory conditions has led to an increased interest in gastrointestinal nematodes and the molecules they produce to evade the human immune system. Galectins are carbohydrate-binding proteins that are potent, multifunctional signalling proteins for the immune system, and form a large component of the excretory/secretory molecules nematodes produce during infection.

The aim of this research was to determine if *Necator americanus* (New World Hookworm) and *Trichuris trichiura* (Human Whipworm), produced functional galectin homologues of human galectin 3 and 9, and determine if they display anti-inflammatory properties.

Protein databases for *N. americanus* and *T. trichiura* were analysed and significant sequence and structural similarity to human galectin 3 and 9 was detected. Four proteins were expressed using *E. coli* and separated by lactose affinity purification.

Hookworm-galectin-3 increased proliferation in the human epithelial HCA-7 colon carcinoma cell line, 20% on average at 5ug/ml, compared to untreated cells (p -value <0.05).

In-vitro results suggest that synthetic parasitic galectin may interfere with host cells. Experiments to assess if these galectins can alter the cytokine expression of human immune cells in vitro are underway. This research may further our understanding of how nematodes alter the host immune system to modulate inflammation.

ID: 291 / CP14.1: 3

Contributed abstract

Conference Topics: Helminthology

Keywords: transgenic hookworms, therapeutics

Production of transgenic hookworms that secrete therapeutic foreign molecules.

Deonne Walther^{1,2}, Michael Smout¹, Paul Giacomini¹, Paul Brindley³, Makedonka Mitreva⁴, Alex Loukas¹

¹Australian Institute of Tropical Health and Medicine, James Cook University, Cairns, Queensland, Australia; ²College of Medicine and Dentistry, James Cook University, Cairns, Queensland, Australia; ³Department of Microbiology, Immunology & Tropical Medicine, and Research Centre for Neglected Diseases of Poverty, School of Medicine & Health Sciences, George Washington University, Washington, DC, USA; ⁴The McDonnell Genome Institute, Washington University in St. Louis, St. Louis, Missouri

Clustered Regularly-Interspaced Palindromic Repeats (CRISPR) and CRISPR-associated (Cas) protein technology has only very recently been applied to the study of parasitic helminths, unlike the free-living counterparts (*Caenorhabditis elegans*) where there is a wealth of published information. Recent unpublished studies from our US collaborators have shown insertion of foreign genes into the germline stage of parasitic helminths is possible. This ensures heritable lines of transgenic worms can be developed where every cell in the organism carries the transgene. Given that parasitic hookworms live for many years in the gastrointestinal tract and secrete proteins into the gut tissue, they provide the ideal delivery tool for directing protein therapeutics directly to the gut. I will discuss how we plan to develop transgenic hookworms that secrete therapeutic antibodies or biologics that are already used in the clinic to treat people with inflammatory bowel disease (IBD). The transgenic hookworms will constitutively deliver the drugs to the affected tissues, and efficacy of the approach can be tested in different mouse models of IBD that are widely used in our laboratory.

P3: Elsevier Plenary Lecture Series International Journal for Parasitology: Drugs and Drug Resistance (IJP:DDR) Invited Lecturer

Time: Thursday, 03/July/2025: 9:00am - 9:45am · *Location:* Conference plenary room
Session Chair: Kevin Saliba, Australian National University

ID: 297 / P3: 1

Invited speaker abstract

Dissecting drug-target networks and resistance pathways in *Giardia* with mass spectrometry

Samantha Emery-Corbin

Monash University, Australia

Giardia duodenalis, a deeply branching eukaryote and a leading global cause of parasitic diarrhoea, is treated exclusively with the nitroheterocycle metronidazole (in clinical use for over 70 years) and the benzimidazole albendazole. Escalating compound toxicity, patient non-compliance and laboratory-derived resistance threaten the continued efficacy of its limited front-line therapies, compounded by a critical lack of system-wide insight into drug-target engagement and resistance mechanisms across genetically distinct human-infecting assemblages. Here, we show that integrated mass spectrometry and chemoproteomic workflows can resolve these gaps, delivering unprecedented molecular resolution of drug mode-of-action and resistance evolution in *Giardia*. Interrogation of isogenic metronidazole-resistant assemblage A lines by high-multiplex tandem-mass-tag quantitation revealed extensive rewiring of redox metabolism, thiol-dependent detoxification and NAD-dependent pathways, which align with *Giardia*-specific lysine-acetylation hotspots that coordinate stress adaptation, protein folding and energy metabolism identified via acetylome proteomics. Broader comparative proteomics analyses along the “road to resistance” between assemblages A and B uncovered strikingly divergent signatures, dramatic whole-proteome shifts in assemblage B versus modest, fold-change-reduced shifts in assemblage A. However, both assemblages showed concordant, limited perturbation of protein expression under albendazole selection. Consequently, more detailed, refined drug-screens including 13 BZ compounds and 7 Alb structural analogues teased apart the nature of beta-tubulin-specific resistance in Assemblage A, and identified the first protistan β -tubulin point mutation (E198K) linked to albendazole insensitivity. Together, these studies underscore the power of proteomics for precision diagnostics and the rational design of next-generation anti-giardial therapies.

P4: Elsevier Plenary Lecture Series International Journal for Parasitology: Parasites and Wildlife (IJP:PAW) Invited Lecturer

Time: Thursday, 03/July/2025: 9:45am - 10:30am · Location: Conference plenary room
Session Chair: Ian Beveridge, University of Melbourne

ID: 298 / P4: 1

Invited speaker abstract

Tales from a wandering (wildlife) parasitologist

Di Barton

Charles Sturt University, Australia

This presentation will take you on a wander through the wonderful world of the many different parasites of Australian wildlife. We will explore the past, present and the potential future of wildlife parasitology in Australia. Using examples from projects involving parasites of native and introduced animals, from snails to cane toads to wedgetail eagles and feral deer, I will highlight how little we still know about the diversity, ecology and relationships of the parasites involved. Then, we will finish with some hopes - and maybe some pleas - for the future of the teaching and research of wildlife parasitology in Australia.

CP16: Drugs & Drug Resistance 15 min talks

Time: Thursday, 03/July/2025: 11:00am - 12:15pm · Location: Conference room 1

Session Chair: Kathy Andrews, Griffith University

Session Chair: Adele Lehane, Australian National University

ID: 184 / CP16: 1

Contributed abstract

Conference Topics: Biochemistry, Drugs, Malaria, Molecular Biology, Protozoa

Keywords: Malaria, antimalarial, drug discovery, aminoacyl-tRNA synthetases

Natural product-mediated reaction hijacking mechanism validates *Plasmodium* aspartyl-tRNA synthetase as an antimalarial drug target

Nutpaka Ketprasit¹, Chia-Wei Tai¹, Vivek Kumar Sharma², Yogavel Manickam², Yogesh Khandokar³, Xi Ye¹, Con Dogovski¹, David H. Hilko^{4,5}, Craig J. Morton⁶, Anne-Sophie C. Braun^{4,5}, Michael G. Leeming⁷, Bagale Siddharam⁸, Gerald J. Shami¹, P.I. Pradeepkumar⁸, Santosh Panjekar^{3,9}, Sally-Ann Poulsen^{4,5}, Michael D.W. Griffin¹, Amit Sharma², Leann Tilley¹, Stanley C. Xie^{1,10}

¹Department of Biochemistry and Pharmacology, Bio21 Molecular Science and Biotechnology Institute, The University of Melbourne, Melbourne, VIC 3010, Australia; ²Molecular Medicine - Structural Parasitology Group, International Centre for Genetic Engineering and Biotechnology, New Delhi 110067, India; ³Australian Synchrotron, ANSTO, Clayton, VIC 3168; ⁴Institute for Biomedicine and Glycomics, Griffith University, Nathan, QLD 4111, Australia; ⁵School of Environment and Science, Griffith University, Nathan, QLD 4111, Australia; ⁶Biomedical Manufacturing Program, CSIRO, Clayton South, VIC 3169, Australia; ⁷Melbourne Mass Spectrometry and Proteomics Facility, Bio21 Molecular Science and Biotechnology Institute, The University of Melbourne, Melbourne, VIC 3010, Australia; ⁸Department of Chemistry, Indian Institute of Technology Bombay, Powai, Mumbai 400 076, India; ⁹Department of Biochemistry and Molecular Biology, Monash University, Clayton, VIC, 3800, Australia; ¹⁰Drug Delivery Disposition and Dynamics, Monash Institute of Pharmaceutical Sciences, Monash University, Parkville, VIC 3052, Australia

With ever-increasing resistance to currently deployed antimalarials, new targets and starting point compounds with novel mechanisms of action need to be identified. Here, we explore the antimalarial activity of the *Streptomyces* spp. natural product, dealanylascamycin (DACM), and compare it with the synthetic 5-O-sulfamoyladenine (AMS). These nucleoside sulfamates exhibit potent inhibition of *P. falciparum* growth and protein translation. We show that DACM targets multiple amino acyl tRNA synthetase (aaRS) targets, including aspartyl tRNA synthetase (AspRS). The mechanism involves hijacking of the reaction product, leading to the formation of tightly bound inhibitory amino acid-sulfamate conjugates. We show that recombinant *P. falciparum* and *P. vivax* AspRS are susceptible to hijacking by DACM and AMS, generating Asp-DACM and Asp-AMS adducts that stabilize these proteins. X-ray crystallography reveals that *P. vivax* AspRS exhibits a stabilized flipping loop over the active site that is poised to bind substrates. However, human AspRS exhibits disorder in an extended region around the flipping loop as well as in a Motif II loop, likely decreasing the susceptibility of human AspRS to reaction-hijacking by DACM and AMS. Our work reveals *Plasmodium* AspRS as a promising target and highlights structural features that underpin differential susceptibility of aaRSs to reaction hijacking inhibition.

ID: 255 / CP16: 2

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Malaria, Microscopy, Molecular Biology, Protozoa

Keywords: malaria, resistance, microscopy, expansion

Cytostome formation in artemisinin resistant *Plasmodium* parasites

Long K Huynh¹, Ben Liffner², Niall Geoghegan³, Dawson Ling³, Sabrina Absalon², Stuart A Ralph¹

¹Department of Biochemistry and Pharmacology, Bio21 Molecular Science and Biotechnology Institute, The University of Melbourne, Parkville, VIC, 3010; ²Department of Pharmacology and Toxicology, Indiana University School of Medicine, Indianapolis, IN, USA; ³The Walter & Eliza Hall Institute of Medical Research, Department of Medical Biology, The University of Melbourne, Parkville, VIC, Australia

Resistance to the frontline antimalarial, artemisinin, is predominantly driven by mutations in the gene encoding the Kelch 13 (K13) protein. K13 plays a crucial role in the regulation of a double-membraned invagination termed the cytostome; the apparatus which facilitates haemoglobin uptake from the host red blood cell into the *Plasmodium* parasite. Haemoglobin digestion supplies essential amino acids for parasite growth, whilst also releasing haem-iron as a waste by-product required for

artemisinin activation. Parasites with mutant K13 exhibit slower feeding, reduced haem levels, and delayed growth, leading to decreased artemisinin activation and parasite death. However, the exact mechanism linking K13 mutations to impaired feeding remains unclear.

We propose that K13 mutations reduce its stability and abundance, impairing cytosome formation/maintenance and thus parasite feeding. Using immunofluorescence assays and ultrastructure expansion microscopy (u-ExM) combined with super-resolution imaging, we resolved K13 as ring-shaped structures ~160nm in diameter at the cytosomal neck. Lattice light-sheet microscopy and u-ExM throughout the asexual blood cycle revealed that mutant parasites still form cytosomal rings, but at a slower rate than wild-type. Some cytosomes in K13 mutants also displayed aberrant morphologies. These findings provide new insights into how K13 mutations could reduce haemoglobin uptake, linking this defect to artemisinin resistance.

ID: 252 / CP16: 3

Contributed abstract

Conference Topics: Apicomplexa Biology, Biochemistry, Drugs

Keywords: Cryptosporidium, aspartyl protease, drug

Targeting an Aspartyl Protease to develop new drugs to treat the diarrheal pathogen

Cryptosporidium parvum

Susie Wang^{1,2}, Kharizta Wiradiputri^{1,2}, Simona Seizova^{1,2}, Jerzy Dziekan^{1,2}, Lena Chng^{1,2}, Anna Ngo^{1,2}, Ying Zhang^{1,2}, Kate Jarman^{1,2}, Kym Lowes^{1,2}, David Olsen³, Alan Cowman^{1,2}, Christopher Tonkin^{1,2}

¹WEHI, Australia; ²University of Melbourne, Australia; ³Merck Sharp & Dohme

Cryptosporidium spp. are a leading cause of diarrhea and contribute to severe morbidity and mortality in young children and immunocompromised adults. Despite the significant global health impact, there are currently no vaccines or effective treatments for cryptosporidiosis. The only FDA-approved drug, Nitazoxanide, has limited efficacy in AIDS patients and young children, where therapy is most needed. *Cryptosporidium spp.* are enteric protozoan parasites that is related to the malaria-causing *Plasmodium spp.* We have recently shown that a new class of drugs that target *Plasmodium* aspartyl proteases is also efficacious against *Cryptosporidium*. We show that WM382, previously identified to potently inhibit *Plasmodium* growth, also works to kill *Cryptosporidium*, by targeting the aspartyl protease CpASP4, as determined by solubility-based proteomic methodologies. We have built on these initial findings and identified more potent inhibitors by screening a library of 5,000 related compounds that were built as part of the *Plasmodium* drug discovery program. Furthermore, the lead compound that has entered clinical trials for the treatment of malaria also kills *Cryptosporidium*. A structure activity relationship is being developed with a focus on in vivo killing of this devastating diarrheal parasite.

ID: 278 / CP16: 4

Contributed abstract

Conference Topics: Apicomplexa Biology, Drugs, Malaria, Molecular Biology

Keywords: Plasmodium, Anopheles, PlasmeprinIX/X, invasion, inhibitors

Dual inhibition of Plasmeprins IX and X in *Plasmodium falciparum* sporozoites inhibits development within *Anopheles stephensi* mosquitoes

Elena Lantero-Escobar^{1,2}, John A. McCauley³, David B. Olsen³, Alan F. Cowman^{1,2}, Justin A. Boddey^{1,2}

¹The Walter and Eliza Hall Institute of Medical Research, Parkville, VIC 3052, Australia; ²University of Melbourne, Melbourne, VIC 3010, Australia; ³Merck & Co., Inc., 770 Sumneytown Pike, West Point, PA 19486, USA

Plasmeprin IX (PMIX) and X (PMX) are essential aspartyl proteases in all *Plasmodium spp.* that cleave a catalogue of substrates required for egress and invasion of host cells across the lifecycle. The dual PMIX and PMX inhibitor WM382 prevents merozoites egress and invasion of erythrocytes, blocks transmission of ookinetes to mosquitoes and attenuates the egress of liver-merozoites in merosomes and the first wave of erythrocytic infection. Despite strong expression of PMIX and PMX in sporozoites, their function remains unknown. Treatment of mice with WM382 4-hours prior to intravenous injection with *P. berghei* sporozoites had no effect on the rate of liver-stage infection nor did it cause any delay in the prepatent period, suggesting these aspartyl proteases have an important function in sporozoites as they develop within the mosquito vector. Like atovaquone, dosing *An. stephensi* mosquitoes with WM382 dramatically impaired the number of sporozoites located within the salivary glands. This suggests that PMIX and PMX are required for processing of substrates required for sporozoite invasion of salivary glands, such as MAEBL and AMA1 that contain cleavage sites. We will present our up-to-date understanding of the function of PMIX and PMX in *P. falciparum* sporozoites as they develop within the mosquito host.

ID: 127 / CP16: 5

Contributed abstract

Conference Topics: Biochemistry, Drugs, Malaria

Keywords: Malaria, Plasmodium, drug target, ion channel, membrane protein

Aryl N-acetamide compounds exert antimalarial activity by acting as agonists of rhomboid protease PfROM8 and cation channel PfCSC1.

Paul Gilson¹, Coralie Boulet¹, Joyanta Modak², Zelda Smith⁴, Deyun Qiu⁴, Natalie Counihan², Molly Parkyn Schneider¹, William Nguyen³, Madeline Dans³, Claudia Barnes¹, Zahra Razzok¹, Kirsty McCann², Alyssa Barry², Brendan Crabb¹, Brad Sleebs³, Tania de Koning-Ward², Adele Lehane⁴

¹Burnet Institute, Australia; ²Deakin University; ³Walter and Eliza Hall Institute; ⁴Australian National University

With resistance to frontline antimalarials spreading, new drugs are needed. We previously screened the 'Pathogen Box' for compounds that prevent *Plasmodium falciparum* parasites from invading erythrocytes. MMV020512 was identified in this screen and live cell imaging here established that it does not specifically inhibit invasion but likely inhibits intraerythrocytic parasite growth. Parasites selected for resistance to MMV020512 had mutations in the membrane protease PfROM8 and/or ion channel PfCSC1. Knockdown of PfROM8 or PfCSC1, or the introduction of the L562R mutation in PfROM8, reduced parasite growth, indicating that both proteins are functionally important. Parasites with these changes also became less sensitive to MMV020512, suggesting that MMV020512 does not act by inhibiting PfROM8 or PfCSC1. MMV020512, as well as

more potent analogues and other PfCSC1-linked compounds, gave rise to an increase in the parasite's internal [Na⁺]. Parasites with a L562R mutation in PfROM8, a L954F mutation in PfCSC1, or that had reduced expression of PfCSC1 were less susceptible to Na⁺ dysregulation by the compounds. Our data suggest that there is a functional interaction between PfROM8 and PfCSC1, and that the compounds stimulate ion flow through PfCSC1, perhaps by acting as agonists of PfCSC1 or by affecting the interaction between the two proteins.

CP17: Immunology & Vaccination 15 min talks

Time: Thursday, 03/July/2025: 11:00am - 12:15pm · Location: Conference room 2

Session Chair: Denise Doolan, University of Queensland

Session Chair: Lee M. Yeoh, Burnet Institute

ID: 119 / CP17: 1

Contributed abstract

Conference Topics: Epidemiology, Immunology, Malaria, Vaccines

Keywords: malaria, immunology, vaccinology, plasmodium vivax

Naturally acquired functional antibody responses to *Plasmodium vivax* vaccine candidates are associated with protection against clinical malaria infections

Pailene S. Lim^{1,2}, Francisco J. Martinez³, Arunaditya Deshmukh³, Ivo Mueller^{1,2}, Chetan E. Chitnis³, Rhea J. Longley^{1,2,4}

¹Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria; ²Department of Medical Biology, The University of Melbourne, Parkville, Victoria; ³Malaria Parasite Biology and Vaccines Unit, Institut Pasteur, Paris, France; ⁴Faculty of Tropical Medicine, Mahidol University, Bangkok, Thailand

Malaria infections caused by *Plasmodium vivax* continue to be a major global health burden. An effective long-lasting vaccine that can specifically target *P. vivax* infection would contribute greatly to efforts against malaria morbidity. Currently all approved malaria vaccine candidates target *P. falciparum* only. By studying naturally acquired immunity to *P. vivax* in endemic populations, a better understanding of target antigens and immune mechanisms for effective vaccines can be gained. We aimed to determine if functional antibody responses to potential blood and liver-stage *P. vivax* vaccine candidates were associated with protection against clinical malaria infection. We utilised a multiplexed Luminex assay to measure functional antibody responses to 6 *P. vivax* antigens in plasma samples from a well described longitudinal cohort of children aged 1 – 3 years old from Papua New Guinea. Statistical methods were then used to assess the association of functional antibody responses with protection against subsequent clinical malaria infections during 16 months follow-up. We observed that total IgG, IgG1, IgG3 responses to PvEBPII and DBPII were associated with protection against clinical diseases whilst FcγR responses to PvEBPII only were protective. These results suggest that there is potential in studying PvEBPII further.

ID: 222 / CP17: 2

Contributed abstract

Conference Topics: Apicomplexa Biology, Bioinformatics, Epidemiology, Host-parasite interactions, Malaria, Vaccines

Keywords: clinical malaria, naturally acquired immunity, allele specific immune response, genetic diversity, antigenic diversity, Plasmodium falciparum, Papua New Guinea

Protective immune responses to *Plasmodium falciparum* malaria are associated with antibodies to conserved but not diverse parasite antigens

Myo Naung^{1,2,3,10}, Ramin Mazhari², Rhea Longley^{2,3,4}, Somya Mehra^{1,2,4}, Wilson Wong², Paolo Bareng^{1,10}, Moses Laman⁵, Benson Kiniboro⁵, Maria Ome-Kaius⁵, Pascal Michon⁶, James Beeson^{1,7}, Eizo Takashima⁸, Takafumi Tsuboi⁸, Leanne Robinson^{1,2,3}, Ivo Mueller^{2,9}, Alyssa Barry^{1,2,3,10}

¹Burnet Institute, Australia; ²Population Health and Immunity Division, Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria, Australia; ³Department of Medical Biology, University of Melbourne, Parkville, Victoria, Australia; ⁴Faculty of Tropical Medicine, Mahidol University, Bangkok, Thailand; ⁵Vector Borne Diseases Unit, Papua New Guinea Institute of Medical Research, Madang, Papua New Guinea; ⁶Papua New Guinea Institute of Medical Research, Madang, Papua New Guinea; ⁷Department of Immunology and Pathology, Monash University, Melbourne, VIC, Australia; ⁸Division of Malaria Research, Proteo-Science Center, Ehime University, Japan; ⁹Department of Parasites and Insect Vectors, Pasteur Institute, Paris, France; ¹⁰Centre for Innovation in Infectious Diseases and Immunology Research (CIIDIR), Institute of Mental and Physical Health and Clinical Translation (IMPACT) and School of Medicine, Deakin University, Geelong, Victoria, Australia

Measuring antibody responses to malaria antigens helps prioritize vaccine candidates, but genetic and physicochemical diversity may influence these responses. To explore this, we measured total IgG against the 3D7 allele of 25 *Plasmodium falciparum* antigens in two cohorts of Papua New Guinean children (ages 5–14, n=696) from high (~10 infections/year) and moderate (<1 infection/year) transmission settings. IgG was assessed at baseline and study endpoint using a Luminex multiplex array. Protection was defined by the association between baseline IgG levels and risk of clinical episodes. Using Cox regression and a Bayesian antibody kinetics model, we assessed how protection correlated with antigen diversity, selection pressure, and structure. In high transmission, IgG levels were broadly maintained, but only antibodies to conserved antigens were linked to protection. Antibodies to highly diverse or immune-selected antigens showed weaker protection. In moderate transmission, IgG responses were variable, short-lived, and indicative of recent exposure rather than immunity. Notably, the most protective antigens were conserved and structurally unique, with high proportions of disordered and alpha-helical regions. These findings emphasize the need to account for genetic and structural diversity when selecting malaria vaccine targets.

ID: 120 / CP17: 3

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Malaria

Keywords: Gametocyte, transmission, inhibition, malaria

Identification of novel anti-gametocyte transmission blocking vaccine targets

Fiona Angrisano^{*1}, Hayley Bullen^{*1}, Ameila Ford², Senna Steel¹, Paul Gilson¹, Brad Sleebs³, William Nguyen³, Andrew Blagborough²

¹Burnet Institute, Australia; ²University of Cambridge; ³Walter and Eliza Hall Institute

Inhibiting transmission of *Plasmodium* is a central strategy in malaria eradication, and the biological process of gamete fusion during fertilisation is a proven target for this approach. The lack of knowledge of the mechanisms underlying fertilisation have been a hindrance in the development of transmission-blocking interventions. Here, for the first time we describe a protein disulphide isomerase essential for malarial transmission (PDI-Trans/PBANKA_0820300) to the mosquito. We show that PDI-Trans activity is male-specific, surface expressed, essential for fertilisation/transmission, and exhibits disulphide isomerase function which is up-regulated post-gamete activation. We demonstrate that PDI-Trans is a viable anti-malarial drug and vaccine target blocking malarial transmission with the use of repurposed the PDI inhibitors and anti-PDI-Trans peptide antibodies. Additionally, we have found that PDI-Trans is linked to the folding of invasion proteins in asexual parasites. These results reveal that protein disulphide isomerase function is crucial for arresting malaria transmission and circumventing clinical disease, highlighting the potential of anti-PDI agents to act as dual stage inhibitors, facilitating the development of novel antimalarials to eliminate malaria.

ID: 176 / CP17: 4

Contributed abstract

Conference Topics: Immunology, Malaria, Vaccines

Keywords: Malaria, Antibodies

Targets of Fc-dependent functional antibody responses in immunity to severe *Plasmodium falciparum* malaria in children

Grace Wright^{1,2}, D. Herbert Opi^{1,2,3}, Liriye Kurtovic^{1,3}, Kaitlin Pekin^{1,4}, Sandra Chishimba^{1,2}, Adam Thomas¹, Moses Laman⁵, Laurens Manning⁶, Ivo Mueller⁷, Stephen J. Rogerson², James G. Beeson^{1,2,3}

¹Burnet Institute, Melbourne, Australia; ²Department of Medicine, University of Melbourne, Melbourne, Australia; ³Central Clinical School and Department of Microbiology, Monash University, Melbourne, Australia; ⁴School of Biological Sciences, University of Adelaide, Adelaide, Australia; ⁵Papua New Guinea Institute of Medical Research, Madang, Papua New Guinea; ⁶Department of Infectious Diseases, University of Western Australia, Perth, Australia; ⁷Walter and Eliza Hall Institute of Medical Research, Melbourne, Australia

Plasmodium falciparum malaria is a major cause of severe and fatal disease in young children. Current vaccines do not target the merozoite parasite form, which replicates in the blood causing disease and associated symptoms. Identifying the targets and mechanisms of immunity that prevent severe malaria will aid the development of vaccines with greater efficacy. This study used a novel multi-antigen, multi-functional antibody assay platform to identify merozoite antigens targeted by Fc-dependent functional antibodies that are associated with protection against severe malaria in children from Papua New Guinea (severe malaria, n=201; uncomplicated malaria, n=163). We evaluated the functional activity of antibodies to interact with Fcγ receptors I, IIa, and IIIa/b to 33 merozoite antigens. Higher antibody Fcγ receptor binding activity to several key antigens were associated with a significantly reduced risk of severe malaria. These included known vaccine candidate antigens and less-studied antigens. Furthermore, higher functional antibodies to multiple antigens were associated with a greater reduction in the risk of severe malaria compared to antibodies to single antigens. Identification of merozoite antigens targeted by functional antibodies and associated with protection against severe malaria will enable the development of highly efficacious malaria vaccines to reduce the burden of severe and life-threatening malaria.

ID: 258 / CP17: 5

Contributed abstract

Conference Topics: Immunology

Keywords: neutrophils, *Plasmodium falciparum*, neutrophil extracellular traps

Neutrophil extracellular trap (NET) formation is inhibited by both *Plasmodium falciparum*-infected and uninfected erythrocytes.

Akachukwu Onwuka¹, Agersew Alemu¹, Wina Hasang¹, Elizabeth Aitken^{1,2}, Stephen Rogerson^{1,3}

¹Department of Infectious Diseases, The Peter Doherty Institute of Infection and Immunity, University of Melbourne, Melbourne, Australia.; ²Department of Microbiology and Immunology, The Peter Doherty Institute of Infection and Immunity, University of Melbourne, Melbourne, Australia.; ³Department of Medicine, The Peter Doherty Institute of Infection and Immunity, University of Melbourne, Melbourne, Australia.

Neutrophils are innate immune cells, but their role in clearing *Plasmodium falciparum* in humans is unclear and requires investigation. Neutrophil extracellular traps (NETs) are DNA and protein webs that trap and stop the spread of infection. We developed a simple assay to assess the effect of infected erythrocytes (IE) and uninfected erythrocytes (UE) on NET formation. In the assay, SYTOX green-stained primary neutrophils stimulated with phorbol 12-myristate 13-acetate (PMA) and calcium ionophore (Cal) were cocultured with different doses of IE and UE for 5 hours at neutrophil:erythrocyte ratios of 1:5, 1:10, 1:20, and 1:40. NET formation was measured by fluorometric DNA quantification and confocal imaging. Extracellular myeloperoxidase (MPO), a NET enzyme, was measured by ELISA in the supernatant. IE and UE markedly reduced NETs and MPO release in a dose-dependent manner, with pronounced inhibition at a 1:40 neutrophil:erythrocyte ratio. However, UE showed stronger inhibition than IE, suggesting a non-parasite-specific NET suppression. Microscopy shows reduced NET formation with IE and UE. IE causes less NET inhibition. This study highlights a potential novel immune evasion strategy of *P. falciparum* and suggests that erythrocytes, regardless of infection status, can modulate neutrophil responses. The inhibitory mechanism is under investigation.

CP18: Wildlife Parasites 15 min talks

Time: Thursday, 03/July/2025: 11:00am - 12:15pm · Location: Conference room 3

Session Chair: Kate Hutson, Cawthron Institute

Session Chair: Abdul Jabbar, University of Melbourne

ID: 149 / CP18: 1

Contributed abstract

Conference Topics: Diagnostics, Epidemiology, Protozoa, Veterinary Parasitology, Wildlife parasitology, Zoonoses

Keywords: Toxoplasma gondii, Recombinant antigens, ELISA, Marsupials, Australia.

A novel recombinant antigen-based multi-species enzyme-linked immunosorbent assay (ELISA) to detect *Toxoplasma gondii* exposure in Australian marsupials

Tharaka D. Livanage K.L.D.¹, Brett R. Gardner^{1,2}, Tian Chen¹, Anita Tolpinrud¹, Simon M. Firestone¹, Charles G. Gauci¹, Denise O'Rourke³, Abdul Jabbar¹, Jasmin Hufschmid¹

¹Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Werribee 3030, Victoria, Australia; ²Zoos Victoria, Parkville 3052, Victoria, Australia; ³Asia-Pacific Centre for Animal Health, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Werribee, VIC 3030, Australia

Infection with the zoonotic protozoan parasite *Toxoplasma gondii* is a health concern for Australian marsupial species. However, serological detection of *T. gondii* exposure in marsupials is often hindered by the lack of sensitive diagnostic methods. To address this knowledge gap, a recombinant antigen-based indirect enzyme-linked immunosorbent assay (ELISA) was developed to detect *T. gondii* exposure in multiple marsupial species. This new ELISA, alongside a modified agglutination test (MAT) and qPCR, was used to test 756 serum samples from 11 marsupial species, with 249 samples paired with tissues for qPCR. Bayesian latent class models estimated the diagnostic sensitivity and specificity of the novel ELISA as 95.9% and 80.1% for Tasmanian devils (*Sarcophilus harrisii*), and 84.0% and 91.8% for eastern grey kangaroos (*Macropus giganteus*), respectively. Overall prevalence varied based on the diagnostic method (ELISA 14.2%, MAT 9.8%, qPCR 5.6%) and host species. Higher trophic-level species showed greater exposure, with Tasmanian devils exhibiting the highest prevalence (65.1%). Notably, no exposure was detected in arboreal species. This novel ELISA offers a practical serological tool for routine *T. gondii* detection in diverse marsupial species. Further diagnostic performance evaluation of this novel multispecies ELISA is recommended before widespread application in diagnosis and surveillance.

ID: 238 / CP18: 2

Contributed abstract

Conference Topics: Ecology, Fish parasitology, Host-parasite interactions, Wildlife parasitology

Keywords: Behaviour, metabolic rate, sickness, compensatory growth

Personality changes in infected hosts may reflect hunger, not parasite manipulation

Alan Lymbery¹, Nuwandi Pathirana², Samuel Lymbery¹

¹Murdoch University, Australia; ²University of Peradeniya, Sri Lanka

Infected hosts sometimes have altered behaviours, particularly those related to the personality traits of activity and boldness, and these changes are frequently ascribed to parasite manipulation. We could not repeat the results of previous studies, showing that rodents infected with *Toxoplasma gondii* are bolder and more active, which would enhance trophic transmission. By contrast, we found that fishes infected with *Lernaea cyprinacea* or the bacterium *Photobacterium damsela* did have increased activity and boldness, although these changes should have no effect on transmission. We investigated the cause of host behavioural changes by challenging the native fish, *Nannoperca vittata*, with controlled doses of *P. damsela*, and measuring activity, boldness and metabolic rate for three consecutive weeks before and after challenge. Challenged fish significantly increased activity, boldness and metabolic rate, with differences among individuals in the plasticity of response. We then measured the same traits, over the same time period, in fish that had been either challenged with an immune system activator (lipopolysaccharide) or starved for seven days. The behavioural changes observed following infection were partially replicated by starvation, but not by lipopolysaccharide. We suggest that behavioural and metabolic changes following infection often relate to increased feeding behaviour to support compensatory growth.

ID: 115 / CP18: 3

Contributed abstract

Conference Topics: Helminthology, Wildlife parasitology

Keywords: Cestodes, marsupials, Australasia

Anoplocephalid cestodes of Australasian mammals: their diversity and origins

Ian Beveridge¹, Charles Cauci², Abdul Jabbar³

¹University of Melbourne, Australia; ²University of Melbourne, Australia; ³University of Melbourne, Australia

Marsupials and rodents in Australasia harbour a diverse assemblage of anoplocephalid cestodes, most endemic to the region. Currently, 72 species are recognised but knowledge of the full extent of the fauna is limited as many host species have not yet been examined due to difficulties (geographical and legal) in sampling hosts for parasites. Molecular methods have revealed several species complexes only some of which have been resolved using morphological data. The phylogenetic affinities of this group of cestodes remain uncertain as Australasian herbivorous marsupials evolved in isolation and are derived from ancestral carnivorous hosts. None of the current hypotheses for their evolutionary relationships within the family has been satisfactory, either through primary diversification within Australasia or by the introduction of eutherian mammals invading from Asia. New molecular evidence from the Australasian cestode genera together with additional advances in Australian mammalian palaeontology provide novel possibilities to explain the phylogenetic origins of these cestodes in Australasia.

ID: 202 / CP18: 4

Contributed abstract

Long-term monitoring and genetic characterisation of *Cryptosporidium* and *Giardia* in wildlife from water catchments in Victoria, Australia.

Anson Koehler¹, Tao Wang¹, Shane Haydon², Robin Gasser¹

¹University of Melbourne, Australia; ²Melbourne Water

This study is part of a nine-year (Dec 2015–July 2024) pathogen monitoring program across water catchments supplying Melbourne, Victoria, Australia. *Cryptosporidium* and *Giardia* were detected and genetically characterised in faecal samples from wildlife, primarily deer and kangaroos, but also rabbits, rodents, wombats, wallabies, canids, waterbirds, and emus across nine reservoir areas. PCR targeting the small subunit ribosomal RNA (SSU) and 60 kDa glycoprotein (gp60) genes was used to characterise *Cryptosporidium*, and the triose-phosphate isomerase (tpi) gene for *Giardia*. Sequence data underwent phylogenetic analysis to delineate genotypes and assess zoonotic potential. Of 8,695 samples analysed, *Cryptosporidium* was detected in 3.15% (n = 274), with 37 distinct SSU sequence types identified, including *C. parvum*, *C. ubiquitum*, and *C. viatorum*, with 22 novel genotypes. *Giardia* was detected in 0.16% (n = 14), with three tpi sequence types, all belonging to *G. duodenalis* assemblage A. Further analysis of all 14,960 samples (collected from 2009–2024) will investigate annual and seasonal trends. This study highlights the significant genetic diversity of *Cryptosporidium* and *Giardia* in catchment wildlife and identifies novel genotypes with undetermined zoonotic potential. Continued surveillance will monitor pathogen prevalence and diversity and expand to human populations to assess potential public health risks.

ID: 108 / CP18: 5

Contributed abstract

Conference Topics: Ecology, Host-parasite interactions, Wildlife parasitology

Keywords: hairworm, behaviour manipulation, parasite-host interaction, microbiome

Who's In Control? Microbiome Effects on Parasite-Host Interactions

Rosemary Presburger, Robert Poulin, Priscila Madi Salloum

Department of Zoology, University of Otago, New Zealand

Parasite-host manipulation is a commonly observed phenomenon. For instance, in order to transition from a terrestrial host to an aquatic environment, hairworms (phylum Nematomorpha) induce hydrophilic behaviour in their host. However, there is variation in the level of behavioural manipulation induced by individuals of the same species of parasite on their hosts. Differences in the microbiome of parasites and hosts have been proposed as a mechanism potentially underlying such variation in behavioural manipulation. In this study, the New Zealand native hairworm *Gordius paranensis* and its cave wētā hosts (Rhaphidophoridae) are models for studying the microbiome effects on parasite-host manipulation. Infected and uninfected wētā, as well as free-living mature hairworms, were collected during the summer of 2024-2025. A laboratory behavioural study was undertaken, and the behaviours of infected/uninfected wētā were recorded. Afterwards, worm tissue and host haemolymph were used for microbiome characterisation. We hypothesise that variation in the parasite's and host's microbiome correlates with the intensity of behavioural manipulation. I will present the preliminary results of this project, emphasising the microbiome as a valuable tool in understanding how parasites interact with their hosts and environment.

CP16.1: Drugs & Drug Resistance 5 min talks

Time: Thursday, 03/July/2025: 12:15pm - 12:30pm · Location: Conference room 1

Session Chair: Kathy Andrews, Griffith University

Session Chair: Adele Lehane, Australian National University

ID: 122 / CP16.1: 1

Contributed abstract

Conference Topics: Apicomplexa Biology, Biochemistry, Drugs, Malaria

Keywords: Malaria, Drug discovery, Apicomplexa

Discovery and Characterisation of Aryl N-Acetamide Antimalarials which are Susceptible to Mutations in ROM8 and CSC1

William Nguyen^{1,2}, Coralie Boulet³, Madeline Dans^{1,2}, Paul Gilson³, Brad Sleebs¹

¹The Walter and Eliza Hall Institute of Medical Research, Parkville, Australia; ²The University of Melbourne, Parkville, Australia;

³Macfarlane Burnet Institute for Medical Research and Public Health, Melbourne, Australia

The continued development of antimalarials which possess novel mechanisms of action is paramount to combating the rising resistance of the malaria parasite, *P. falciparum*, to gold standard artemisinin-based therapies.

A phenotypic screen on the Medicines for Malaria Venture (MMV) Pathogen Box employing transgenic *P. falciparum* parasites, identified an aryl N-acetamide hit (MMV020512) as an inhibitor of ring stage parasite development, exhibiting modest activity in a 72-hour asexual parasite assay. Additional phenotyping established that this hit inhibited the growth of ring stage and trophozoite parasites, but did not show specificity for merozoite invasion or egress.

Chemical optimisation of this series resulted in significant enhancement in *P. falciparum* asexual stage potency, accompanied with no human cell cytotoxicity. Optimised analogues had a moderate rate of sexual kill and potently inhibited both gametocytes and gametes and blocked transmission of the parasite to the mosquito.

N-Aryl acetamide *P. falciparum* resistant clones were generated and genome sequencing uncovered mutations found in rhomboid protease 8 (ROM8) and a putative calcium channel, CSC1. As the role of these proteins are still unknown, optimised analogues will serve as important tools to investigate the role of ROM8 and CSC1 in *P. falciparum* development and survival.

ID: 177 / CP16.1: 2

Contributed abstract

Conference Topics: Apicomplexa Biology, Biochemistry, Drugs, Genomics, Malaria, Molecular Biology, Protozoa
Keywords: Plasmodium, Malaria, Antimalarials, Pantothenate analogues, Drug resistance

Studies into the mechanism of action of pantothenate analogues in *Plasmodium falciparum*

Zaynab Radih, Kevin J Saliba

Research School of Biology, Australian National University, Canberra, ACT, Australia

Plasmodium falciparum is responsible for the most severe form of malaria in humans. Due to the parasite's resistance to all current antimalarials, identifying new drug targets is crucial. The coenzyme A (CoA) biosynthesis pathway is under investigation as a novel drug target against *P. falciparum*. There are five enzymes responsible for converting pantothenate (vitamin B₅) into CoA for utilisation by the parasite. Pantothenate analogues have been put forward as promising drug leads for the development of novel antimalarials that interfere with this pathway. The mechanism of action of various pantothenate analogues is not fully understood. One such pantothenate analogue is AH-2-45. This compound has been suggested to act by virtue of its metabolism by *PfPank*, *PfPPAT* and *PfDPCCK*, three of the five enzymes involved in CoA biosynthesis, generating a CoA antimetabolite. To investigate the mechanism of action of AH-2-45 in more detail, we will use *in vivo* evolution to generate AH-2-45-resistant parasites coupled with whole-genome sequencing to identify proteins involved in its mechanism of action. We will also investigate the effect of overexpressing *PfPPAT* or *PfDPCCK* on parasite sensitivity to AH-2-45 and various other pantothenate analogues.

ID: 236 / CP16.1: 3

Contributed abstract

Conference Topics: Apicomplexa Biology, Biochemistry, Drugs, Malaria, Protozoa

Keywords: Malaria, Drugs, Antiplasmodials, Apicomplexa, Protozoa

The role of *PfAcAS* and *PfACS11* in the antiplasmodial mechanism of action of pantothenate analogues

Christy-Amber Radmann, Kevin J Saliba

Research School of Biology, Australian National University, Canberra, Australia

A rise in antimalarial resistance in *Plasmodium falciparum* highlights the importance of identifying novel drug targets. Coenzyme A (CoA) synthesising and utilizing enzymes serve as promising drug targets. Analogues of pantothenate, the precursor of CoA biosynthesis and an essential nutrient for the intraerythrocytic stage of the malaria parasite, form antimetabolites that interfere with CoA synthesis or utilization. It has previously been shown that mutations in the CoA utilizing-enzymes, acetyl-CoA synthetase (*PfAcAS*) and acyl-CoA synthetase 11 (*PfACS11*), render the parasites resistant to a specific class of pantothenate analogues called inverted pantothenamides. However, the effect of overexpressing *PfAcAS* and *PfACS11* on the sensitivity of the parasite to pantothenate analogues has not been investigated. To address this, we will generate transgenic parasites overexpressing GFP-tagged copies of the wild-type (WT) or mutated forms of *PfAcAS* and *PfACS11*. This will not only allow us to localise the proteins within the parasite and investigate their effect on the parasite's sensitivity to a wider range of pantothenate analogues, but anti-GFP immunoprecipitation will also allow us to investigate the catalytic activity of these proteins and the effect the mutations have on enzyme activity and drug binding.

CP17.1: Immunology & Vaccination 5 min talks

Time: Thursday, 03/July/2025: 12:15pm - 12:30pm · Location: Conference room 2

Session Chair: Denise Doolan, University of Queensland

Session Chair: Lee M. Yeoh, Burnet Institute

ID: 131 / CP17.1: 1

Contributed abstract

Conference Topics: Malaria, Vaccines

Keywords: Malaria vaccine, virus-like particles, multi-stage immunisation, antigen display

Developing New Nanoparticle Delivery Platforms for Multi-stage Malaria Vaccinology.

Anne Nguyen¹, Daniel Luque^{1,2}, Michael Johnson¹, Jake Baum¹

¹School of Biomedical Sciences, University of New South Wales; ²Electron Microscopy Unit, University of New South Wales

In 2022, malaria caused an estimated 249 million cases and 608,000 deaths, yet no vaccine has achieved sterile protection. The *Plasmodium* parasite's complex lifecycle—spanning both human and mosquito hosts—features distinct antigenic profiles at each stage, making single-stage targeting insufficient for long-term control or eradication. RTS,S, the first WHO-approved malaria vaccine, targets the circumsporozoite protein (CSP) of sporozoites using a virus-like particle (VLP) platform fused with hepatitis B surface antigen and adjuvant AS01. Although RTS,S induces robust immune responses and shows 30–50% efficacy, its protection is limited to the liver stage and varies among individuals. To overcome these limitations by developing multi-antigen VLP-based platforms that target several life cycle stages of *Plasmodium*. The approach involves engineering VLPs—such as those based on the woodchuck hepatitis B core antigen (WHcAg)—to present key malaria antigens, including CSP, PIRH5, and Pf230. Technologies like tandem core fusion and split-core co-expression are employed to enable high-density and modular antigen display, with control over orientation, spacing, and stoichiometry. These strategies allow for either multi-antigen display on a single VLP or the combination of multiple VLPs. The goal is to produce next-generation malaria vaccines with enhanced immunogenicity, broader coverage, and more durable protection.

ID: 137 / CP17.1: 2

Contributed abstract

Conference Topics: Immunology, Malaria, Vaccines

Keywords: Malaria, Plasmodium falciparum, Vaccines, Immunology

Antibody specificity and promiscuity are linked with protective efficacy with the leading malaria vaccine in children

Alessia Hysa^{1,2}, **Liriye Kurtovic**^{1,3}, **D. Herbert Opi**^{1,2,3}, **David Wetzel**^{4,5}, **Michael Piontek**⁴, **Jahit Scaralal**^{6,7}, **Carlota Dobaño**^{7,8}, **James G. Beeson**^{1,3,9}

¹Burnet Institute, Melbourne, Australia; ²Department of Infectious Diseases, The University of Melbourne, Melbourne, Australia; ³Department of Immunology and Pathology, Monash University, Melbourne, Australia; ⁴ARTES Biotechnology GmbH, Langenfeld, Germany; ⁵Laboratory of Plant and Process Design, Technical University of Dortmund, Dortmund, Germany; ⁶Centro de Investigação em Saúde de Manhiça, Maputo, Mozambique; ⁷Faculdade de Medicina, Universidade Eduardo Mondlane (UEM), Maputo, Mozambique; ⁸ISGlobal, Hospital Clínic Universitat de Barcelona, Barcelona, Catalonia, Spain; ⁹Department of Medicine, The University of Melbourne, Melbourne, Australia

The RTS,S and R21 malaria vaccines are being implemented among young children in Africa, but their efficacy is modest and variable across populations. These vaccines are based on the *Plasmodium falciparum* circumsporozoite protein (CSP), whereby IgG to the NANP-repeat is strongly induced, but variably associated with clinical protection in vaccinated children. Notably, both vaccine constructs exclude the junction epitope of the CSP that shows similarities to the NANP-repeat but is a known target of antibodies that inhibit liver invasion. Little is known about the specific CSP targets of protective antibody responses against malaria, hindering the development of highly efficacious malaria vaccines.

We evaluated antibody specificity to different peptides across NANP-repeat and junction regions of CSP in young Mozambican children vaccinated with RTS,S in a phase IIb clinical trial (n=735). Antibodies that could bind short NANP-repeat epitopes were significantly associated with protection against malaria. Furthermore, a subset of children generated promiscuous antibodies that recognised the junction epitope (missing in RTS,S), which was associated with higher vaccine efficacy. Identifying key parasite targets of protective antibody responses could enable the redesign of CSP-based malaria vaccines to achieve highly protective next-generation malaria vaccines.

ID: 145 / CP17.1: 3

Contributed abstract

Conference Topics: Immunology, Malaria

Keywords: antibodies, vivax, knowlesi, adaptive-immunity, merozoite surface proteins

Plasmodium vivax merozoite surface proteins are targets of protective multi-functional antibodies

Kaitlin Pekin^{1,2}, **Liriye Kurtovic**^{1,3,4}, **Gaoqian Feng**^{1,5}, **Jill Chmielewski**², **Isabelle Henshall**², **Daisy Mantila**⁶, **Benishar Kombut**⁶, **Maria Ome-Kaius**⁶, **Moses Laman**⁶, **Ivo Mueller**⁷, **Leanne Robinson**^{1,7,8}, **Danny Wilson**², **D. Herbert Opi**^{1,3,9}, **James Beeson**^{1,3,9,10}

¹Burnet Institute, Melbourne, Australia; ²School of Biological Sciences, University of Adelaide, Adelaide, Australia; ³Department of Immunology, Monash University, Melbourne, Australia; ⁴Department of Infectious Diseases, University of Melbourne, Melbourne, Australia; ⁵Department of Pathogen Biology, Nanjing Medical University, Nanjing, China; ⁶Papua New Guinea Institute of Medical Research, Madang, Papua New Guinea; ⁷Walter and Eliza Hall Institute of Medical Research, Melbourne, Australia; ⁸School of Public Health and Preventive Medicine, Monash University, Melbourne, Australia; ⁹Department of Medicine, Doherty Institute, University of Melbourne, Melbourne, Australia; ¹⁰Department of Microbiology, Monash University, Clayton, Australia

Developing efficacious vaccines against *Plasmodium vivax*, a major cause of malaria outside Africa, will greatly support malaria elimination efforts. However, no licensed vaccines exist, and few candidates are under development.

Merozoites, which invade red blood cells, are ideal vaccine targets as they are exposed to the immune system with antibodies playing a major role. A deeper understanding of key antigenic targets and mechanisms of immunity are needed. In a study of malaria exposed Papua New Guinean children, we evaluated the ability of antibodies to induce potent protective functional responses, to six merozoite surface proteins (MSPs). We assessed multiple functional activities, including Fcγ receptor-binding (FcγRI, FcγRIIa, FcγRIIIa), complement fixation, and opsonic phagocytosis. Antibodies to *P. vivax* MSPs could engage Fcγ-receptors at varying levels, and strongest for FcγRI. Antibodies could also promote complement fixation with activity varying between antigens. Antibody opsonic phagocytosis activity was seen for all antigens and strongly correlated with FcγRI-binding. These findings will be further investigated using live *P. knowlesi* parasites as a model to overcome the inability to culture *P. vivax in vitro*. These results support a role for *P. vivax* MSPs as targets of protective immunity and as potential vaccine candidates to reduce the global malaria burden.

CP18.1: Wildlife Parasites 5 min talks

Time: Thursday, 03/July/2025: 12:15pm - 12:30pm · *Location:* Conference room 3

Session Chair: Kate Hutson, Cawthron Institute

Session Chair: Abdul Jabbar, University of Melbourne

ID: 203 / CP18.1: 1

Contributed abstract

Conference Topics: Biodiversity, Ecology, Invasive Species, Protozoa, Wildlife parasitology

Keywords: Sarcocystidae, wildlife

How far do they go; Protozoans in Aussie Marsupials

Keira Brown¹, **Shkoofeh Shamsi**¹, **Diane Barton**¹, **Anthony Rendall**²

¹School of Agricultural, Environmental and Veterinary Sciences, Gulbali Institute, Charles Sturt University, Australia; ²School of Life and Environmental Sciences, Faculty of Science Engineering and the Built Environment, Deakin University, Geelong, Burwood Campus, Victoria, Australia

Australia's ecosystems are unique with a high level of endemism. Substantial biodiversity loss has been observed since the arrival of European settlers, largely attributed to direct predation by invasive species (i.e. Red Foxes *Vulpes vulpes* and Feral Cats *Felis catus*). As a result, biodiversity and conservation of Australian natives is of high priority. Limited research however considers how parasites, transmitted by these invasive species, could impact native mammal communities. With new research finding cases of protozoan parasites of the Sarcocystidae family in both invasive and native animals around Australia, identification of transmission pathways and potential effects is vital. My research aims to bridge these gaps by identifying any

parasites from the Sarcocystidae family that may be present in both native and invasive mammals. Through these identifications possible parasite assemblages, hot spot areas of parasite interaction, or the identification of more susceptible animals may be identified. This work can then be used to assist conservation efforts for a more complete assessment of the environment.

ID: 182 / CP18.1: 2

Contributed abstract

Conference Topics: Veterinary Parasitology, Wildlife parasitology

Keywords: tick(s), tick(-)borne pathogens, wildlife, Rickettsia, Theileria, Australia

A Systematic Review of Ticks and Tick-borne Pathogens of Australian Wildlife

Kabir Brar, Abdul Ghafar, Abdul Jabbar

Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Werribee 3030, VIC, Australia

Ticks and tick-borne pathogens (TTBPs) can cause morbidity and mortality in humans, domestic animals, and wildlife. With the increasing prevalence of tick-borne disease in Australia, identifying potential reservoir hosts that maintain both the tick population and the pathogen within the environment is paramount. In this systematic review, 130 articles published from 1940 to 2024 were critically appraised to encompass the current knowledge on TTBPs pertaining to Australian wildlife. Tick fauna was found to comprise of 50 species mainly belonging to *Ixodes*, *Amblyomma*, and *Haemaphysalis* and predominately discovered using morphological methods. *Ixodes holocyclus* and *Ixodes tasmani* were found at greatest frequency on mammalian species, while *Ixodes hirsti* and *Ixodes holocyclus* were found at greatest frequency on avian species. *Rickettsia*, *Borrelia*, *Babesia*, and *Theileria* were prominent pathogens found in wildlife species. Most wildlife hosts' role in maintaining tick populations, and whether wildlife serve as amplifying or dilution hosts for pathogens is unclear. Furthermore, vector competence of ticks and whether any of the pathogens pose a threat to human, domestic animal, and wildlife remains unknown. Given the ever-changing ecosystem due to anthropogenic activity, a multidisciplinary approach is required to address these knowledge gaps to facilitate prevention of possible disease associated with TTBPs.

ID: 104 / CP18.1: 3

Contributed abstract

Keywords: Intestinal parasites, *Gazella marica*, *Gazella arabica*, *Gazella gazella arlangeri*, direct smear, flotation

Assessment of Intestinal Parasites Prevalence among Gazelle Species at King Khalid Wildlife Research Center (KKWRC) in Riyadh, Kingdom of Saudi Arabia"

Wafa Almegrin, Ghali Alawad, Hajar Alshehri, Shatha Alsalamah

Princess Nourah bint Abdulrahman University, Saudi Arabia

Gazelles are globally endangered animals, and the Kingdom of Saudi Arabia has

played its part in conserving them by establishing various reserves to increase their numbers and reintroduce them into their natural habitat. Gazelles can be affected by several intestinal parasites that can endanger their lives. This study aims to detect and investigate gastrointestinal parasites in some species of gazelles at the King Khalid Wildlife Research Center (KKWRC) in Riyadh city. Fecal samples were taken from three types of gazelles, *Gazella marica*, *Gazella arabica*, and *Gazella gazella arlangeri* and examined using direct smear and salt flotation methods. The overall prevalence of gastrointestinal parasites in some species of gazelles at King Khalid Wildlife Research Center was (61%, 69/113). The highest infection rate was observed in *Gazella arabica* (80.7%, 21/26), followed by *Gazella marica* (63.2%, 43/68), and the lowest in *Gazella gazella arlangeri* (26.3%, 5/19). The identified parasites were *Eimeria* sp. was the most prevalent (66.6%, 46/69), followed by *Nematodirus* sp. (37.6%, 26/69). The least prevalent parasites were *Strongyloides* sp. and *Enterobius* sp., each detected in 1.4% (1/69) of infected samples. Additionally, unidentified parasite species were found in 11.5% (8/69).

CP19: Cell & Molecular Biology 15 min talks

Time: Thursday, 03/July/2025: 1:30pm - 2:30pm · *Location:* Conference room 1

Session Chair: Claire Sayers, University of New South Wales

Session Chair: Colin Sutherland, LSHTM London

ID: 212 / CP19: 1

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology

Keywords: Apicomplexa, *Toxoplasma gondii*, Amino acid, Transporter

To make or to take: redundant amino acid acquisition pathways in *Toxoplasma* parasites.

Legian Zhao, Stephen J. Fairweather, Victor Makota, Capella Maguire, Giel G. van Dooren

Australian National University, Australia

The intracellular, apicomplexan parasite *Toxoplasma gondii* relies on both nutrient scavenging and biosynthetic pathways to acquire amino acids essential for survival. Plasma membrane-localised transporters belonging to the Apicomplexan Amino Acid Transporters (ApiATs) mediate uptake of many of these amino acids into *T. gondii* tachyzoites. The *T. gondii* genome also encodes synthesis pathways for a range of non-essential amino acids. We previously identified *TgApiAT2* as an ApiAT family member, although its physiological role in the parasite remained unclear. To gain insights into *TgApiAT2* function, we performed a CRISPR-based genetic screen, comparing gene essentiality in wild-type (WT) parasites to *TgApiAT2* knockouts. We found that synthesis pathways for the amino acids glutamine, serine, alanine, asparagine, and proline were dispensable in WT parasites, but became fitness-conferring in *TgApiAT2* knock outs, suggesting a role for *TgApiAT2* in the uptake of these amino acids. We demonstrate that *TgApiAT2* is indeed the primary glutamine transporter of *T. gondii* parasites. We also identified several other ApiATs as becoming more fitness conferring when *TgApiAT2* is knocked out, pointing to flexibility in the

uptake pathways of essential amino acids. Overall, our work identifies considerable redundancies in the ability of parasites to both make and take amino acids within their host.

ID: 133 / CP19: 2

Contributed abstract

Conference Topics: Cell Biology, Malaria, Microscopy, Molecular Biology, Other

Keywords: Malaria, vaccine, transmission-blocking, fertilisation, cryo-EM

A novel transmission blocking vaccine candidate for *Plasmodium falciparum*

Jill Chmielewski^{1,2}, Melanie H. Dietrich^{1,2}, Li-Jin Chan^{1,2}, Li Lynn Tan¹, Amy Adair¹, Frankie M. T. Lyons^{1,2}, Mikha Gabriela^{1,2}, Sash Lopaticki^{1,3}, Toby A. Dite^{1,2}, Laura F. Dagley^{1,2}, Lucia Pazzagli⁴, Priya Gupta⁴, Mohd Kamil⁴, Ashley M. Vaughan^{4,5}, Rattanaporn Rojrung⁶, Anju Abraham¹, Ramin Mazhari^{1,2}, Rhea J. Longley^{1,2,7}, Kathleen Zeglinski^{1,2}, Quentin Gouil^{1,2,8,9}, Ivo Mueller^{1,2,10}, Stewart A. Fabb¹¹, Rekha ShandreMugan¹¹, Colin W. Pouton¹¹, Alisa Glukhova^{1,2,12,13}, Shabih Shakeel^{1,2,12,14}, Wai-Hong Tham^{1,2,15}

¹The Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria 3052, Australia; ²Department of Medical Biology, The University of Melbourne, Melbourne, Victoria 3010, Australia; ³Department of Infectious Diseases, Doherty Institute, University of Melbourne, Parkville, Victoria 3010, Australia; ⁴Seattle Children's Research Institute, Seattle, WA, USA; ⁵Department of Pediatrics, University of Washington, Seattle, WA, USA; ⁶Division of Malaria Research, Proteo-Science Center, Ehime University, Japan; ⁷Faculty of Tropical Medicine, Mahidol University, Bangkok 10400, Thailand; ⁸Olivia Newton-John Cancer Research Institute, Heidelberg, Victoria 3084, Australia; ⁹School of Cancer Medicine, La Trobe University, Bundoora, Victoria 3086, Australia; ¹⁰School of Global Health, Shanghai Jiao Tong University, Shanghai 200025, China; ¹¹Monash Institute of Pharmaceutical Sciences, Monash University, Parkville, Victoria 3052, Australia; ¹²Department of Biochemistry and Pharmacology, The University of Melbourne, Melbourne, Victoria 3010, Australia; ¹³Drug Discovery Biology, Monash Institute of Pharmaceutical Sciences, Monash University, 381 Royal Parade, Parkville, Victoria 3052, Australia; ¹⁴ARC Centre for Cryo-electron Microscopy of Membrane Proteins, Monash Institute of Pharmaceutical Sciences, Monash University, 381 Royal Parade, Parkville, Victoria 3052, Australia; ¹⁵Research School of Biology, The Australian National University, Canberra, ACT 2600, Australia

Plasmodium falciparum is a major parasitic pathogen resulting in over 600,000 deaths annually. Within the mosquito host, the parasite undergoes fertilisation and sexual reproduction. Preventing parasite fertilisation in the mosquito midgut can halt malaria transmission. Two leading transmission-blocking vaccine candidates are Pfs230 and Pfs48/45, which are expressed as a complex on the surface of sexual stage parasites and are essential for male gamete fertility. We present a 3.36 Å resolution cryo-EM structure of the endogenous Pfs230-Pfs48/45 complex from late-stage sexual stage malaria parasites. This structure identified Pfs230 domains 13 and 14 as the site of interaction with Pfs48/45. We used transgenic parasites with a deletion of these domains to show they are crucial for localisation of Pfs230 on gamete surfaces, and their absence greatly reduces parasite transmission within the female *Anopheles* mosquito. Nanobodies against domains 13 and 14 inhibit Pfs230-Pfs48/45 complex formation, reduce transmission and structural analyses reveal their binding epitopes. Furthermore, domains 13 and 14 are targets of naturally acquired immunity. We also show that mRNA-LNP vaccination of mice with Pfs230 domains 13 and 14 elicits transmission-reducing antibodies. This work shows that Pfs230 domains 13 and 14 are new vaccine candidates for blocking the transmission of this deadly parasite.

ID: 129 / CP19: 3

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Malaria, Molecular Biology, Protozoa, Proteomics

Keywords: gene regulation, protein regulation, protein interactions, gene-editing, *Plasmodium*.

Characterisation of the Apicomplexan specific regulatory protein Alba4 and its putative interacting partners in the malaria parasite *Plasmodium falciparum*.

Meghan Zadow^{1,2}, Ghizal Siddiqui³, Joshua Morrow³, Darren Creek³, Christopher MacRaid³, Danny Wilson^{1,2,4}

¹Research Centre for Infectious Diseases, School of Biological Sciences, University of Adelaide, Adelaide 5005, South Australia.; ²Institute for Photonics and Advanced Sensing (IPAS), University of Adelaide, 5005, South Australia.; ³Drug Delivery, Disposition and Dynamics, Monash Institute of Pharmaceutical Sciences, Monash University, Parkville, Victoria, 3052, Australia; ⁴Burnet Institute, Melbourne 3004, Victoria, Australia.

Plasmodium spp. malaria parasites have distinct lifecycle stages requiring strict gene and protein regulation for stage transition and survival. Surprisingly, malaria and related parasites appear to have a reduced complement of transcriptional regulators, indicating that expanded families of post-transcriptional regulators such as the Alba (Acetylation lowers binding affinity) are key elements of regulatory processes. Here, we tagged *Plasmodium falciparum* Alba4 with FKBP-GFP and demonstrate that expression peaks during pre- and mid-replicative stages of blood stage development. Functional knock-down (mislocalisation) of PfAlba4 from the cytosol to the parasite nucleus resulted in up to a 40% reduction in replication. Taking advantage of the flexibility of the FKBP-GFP tag, Mass-Spectrometry identification of proximity labelled potential interacting partners yielded a dataset of >50 high-confidence interactors enriched for likely regulatory pathway proteins with 'negative regulation of translation' and 'RNA/mRNA binding' as the most enriched molecular functions. Around 23% of the high-confidence hits were conserved *Plasmodium* proteins of unknown function that might contain novel regulators of parasite growth. The gene-edited lines and high-quality preliminary interactome developed here will enable characterisation of PfAlba4's essential cell-regulatory function and potentially identify additional key regulatory proteins for antimalarial development.

ID: 237 / CP19: 4

Contributed abstract

Conference Topics: Bioinformatics, Cell Biology, Genomics, Molecular Biology, Proteomics

Keywords: *Giardia duodenalis*, RNA binding proteins, Post-transcriptional Regulation, CRISPR, RNA interactome capture, Encystation

Eukaryotic-innovative post-transcriptional regulatory networks emerged at the base of eukaryogenesis.

Balu Balan¹, Esther Bandala Sanchez¹, Waruni Abeysekera¹, Samantha J. Emery-Corbin⁷, Jarrod J. Sandow¹, David Zhu¹, Ahmad Wardak¹, Swapnil Tichkule⁸, Myo Naung^{1,3,5}, Amrita Vijay^{1,3}, Sachin Khurana^{1,3}, Jacob Munro^{1,3}, Pradip Roy^{1,2}, Brendan Robert E. Ansell^{1,3}, Olivia Rissland⁶, Staffan G. Svärd⁴, Peter Czabotar^{1,3}, Andrew I. Webb^{1,3}, Marija Dramcanin^{1,3}, Gordon K Smyth^{1,3}, Aaron R. Jex^{1,2,3}

¹Walter and Eliza Hall Institute, Australia; ²Faculty of Sciences, University of Melbourne, Melbourne, Victoria, Australia; ³Department of Medical Biology, University of Melbourne, Parkville, Victoria, Australia; ⁴Department of Cell and Molecular Biology, Uppsala University, Uppsala, Sweden; ⁵Disease Elimination and Maternal and Child Health, Burnet Institute, Melbourne, Victoria, Australia; ⁶Biochemistry and Molecular Genetics, University of Colorado Anschutz Medical Campus, USA; ⁷Monash Proteomics and Metabolomics Platform, Monash University, Victoria, Australia; ⁸Icahn School of Medicine at Mount Sinai, USA

RNA binding proteins (RBPs) serve as crucial post-transcriptional regulators in eukaryotes, orchestrating transcription, RNA transport, splicing, degradation, translation, and translational repression, thereby influencing cell fate, pluripotency, and differentiation. Remarkably, the eukaryotic RBPome remains largely unchanged from yeast to humans, suggesting that many novel RBPs emerged in basal eukaryotes—a largely unstudied phenomenon. Our phylogenomic atlas reveals that the eukaryotic RBPome is shaped by ancestral bacterial and archaeal RBP systems in addition to the emergence and expansion of innovative RBP families. We characterized the RBPome of *Giardia duodenalis*, an early-branching single-celled eukaryote predating yeast by a billion years, uncovering the origins of key eukaryotic PTR innovations. By integrating in silico modelling, transcriptomics, proteomics, and interactome capture, we revealed a diverse repertoire of both canonical and non-canonical (moonlighting) RBPs. Functional genetics, RNA-network capture, and phase-separation assays of critical RBPs—including early Pumilio homologs (PUF, PUM), helicases such as DDX3x and EIF4A, and moonlighting factors like Phosphoglycerate Kinase, indicate that *Giardia*'s RBP regulation exhibits complexity comparable to that in higher eukaryotes. These findings suggest that sophisticated RBP regulation emerged early in eukaryotic evolution, pivotal in eukaryotic emergence and diversification. Overall, our study significantly highlights the evolutionary conservation and early innovation of RBP regulation.

CP20: Diagnostics 15 min talks sponsored Abacus dx

Time: Thursday, 03/July/2025: 1:30pm - 2:45pm · *Location:* Conference room 2

Session Chair: Kelly Ly, Abacusdx

ID: 277 / CP20: 1

Contributed abstract

Conference Topics: Diagnostics, Molecular Biology, Protozoa

Keywords: Nanopore sequencing, Leishmania, heat shock protein 70 gene

Field validation of a novel nanopore sequencing approach reveals new distributions of *Leishmania* parasites in animals and humans in endemic regions

Thi Thuy Nguyen¹, Lucas Huggins¹, Gad Baneth², Andreia Fernandes Brilhante³, Panagiota Ligda⁴, Smaragda Sotiraki⁴, Kanok Preativatanyou⁵, Carla Maia⁶, Robin B. Gasser¹, Vito Colella¹

¹The University of Melbourne, Australia; ²The Hebrew University of Jerusalem, Israel; ³Federal University of Acre, Brazil;

⁴Hellenic Agricultural Organization, Greece; ⁵Chulalongkorn University, Thailand; ⁶Universidade NOVA de Lisboa, Portugal

Leishmaniases is a group of neglected tropical diseases caused by a diverse range of protozoa in the genus *Leishmania* that pose significant public health challenges. The epidemiology of each *Leishmania* species involves a wide spectrum of specific reservoir hosts and vectors that make transmission dynamics complex. Therefore, accurate species-level identification is central for effective diagnosis and control. However, many diagnostic approaches, e.g. microscopy and conventional PCRs, cannot distinguish between closely related species, particularly those coexisting in sympatry. We developed a nanopore sequencing assay targeting the heat shock protein 70 (HSP70) gene and optimised it using a range of *Leishmania* species. The assay was validated against a previously established HSP70-based real-time PCR assay using DNA from dog and human samples collected in endemic areas of the Mediterranean basin, Brazil, and Thailand and showed strong diagnostic agreement. Importantly, the nanopore assay detected previously unreported *Leishmania* species from dogs in Portugal and Brazil and was also able to characterise co-infections of between two-to-four species in humans from Brazil. These results demonstrate the assay's potential to capture the full diversity of *Leishmania* species in complex endemic settings, offering a promising approach for parasite surveillance and improved control of leishmaniases globally.

ID: 281 / CP20: 2

Contributed abstract

Conference Topics: Diagnostics, Strongyloides, Zoonoses

Keywords: recombinase polymerase amplifications, lateral flow, hookworms, Strongyloides, point-of-care

A novel lateral flow recombinase polymerase amplification assay combined with a simple faecal extraction method for the diagnosis of Strongyloidiasis and Hookworm infections in humans

Sze Fui Hii¹, Dinh Ng Nguyen², John Kaldor³, Susana Vaz Nery³, Rebecca Traub⁴, Vito Colella¹

¹Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Australia; ²Faculty of Animal Sciences and Veterinary Medicine, Tay Nguyen University, Vietnam; ³Kirby Institute, University of New South Wales, Australia; ⁴Department of Infectious Diseases and Public Health, City University of Hong Kong, Hong Kong SAR, China

It is estimated that over 1.5 billion people are infected with soil-transmitted helminths (STHs), with resource poor communities being the most affected. Traditional parasitological and coproscopic methods often lack sensitivity and specificity. While molecular techniques such as PCR and qPCR offer high sensitivity and specificity, they require advanced laboratory infrastructure and technical expertise, limiting their use as point-of-care (POC) diagnostics. Recombinase polymerase amplification (RPA) is a sensitive and rapid isothermal molecular method that can be interpreted via fluorescence or lateral flow strips, making it suitable for POC applications.

This study aimed to develop a lateral flow RPA (LF-RPA) assay alongside a simple and cost-effective faecal DNA extraction method for the rapid detection of *Necator americanus*, *Ancylostoma ceylanicum*, and *Strongyloides stercoralis*. The LF-RPA

assays demonstrated high sensitivity (>90%) and 100% specificity when compared to multiplex STH qPCRs. Moreover, the novel faecal extraction method, when used with LF-RPA, showed substantially higher sensitivity than traditional coproscopic methods.

These findings highlight the potential of LF-RPA assays, combined with the novel extraction method, as practical and efficient POC diagnostic tools for STH infections.

ID: 251 / CP20: 3

Contributed abstract

Conference Topics: Diagnostics, Epidemiology, Helminthology, Strongyloides

Keywords: Strongyloidiasis, Hookworms, qPCR, Molecular epidemiology

Assessing the burden of hookworm species and *Strongyloides stercoralis* in Vanuatu: the role of molecular diagnostics

Paolo Bareng¹, Adam Bartlett¹, Sze Fui Hii², Stephanie Tabe³, Garae Mackline³, Fasiah Taleo⁴, Vito Colella², Prudence Rymill³, Susana Nery¹

¹The Kirby Institute, UNSW Sydney, New South Wales, Australia; ²Faculty of Veterinary and Agricultural Sciences, University of Melbourne, Melbourne, Victoria, Australia; ³Ministry of Health, Port Vila, Shefa, Vanuatu; ⁴World Health Organisation, Port Vila, Shefa, Vanuatu

The Vanuatu Ministry of Health recently delivered two rounds of mass drug administration (MDA) to accelerate control of multiple NTDs, including soil-transmitted helminths (STHs). Cluster surveys were conducted in approximately 30 villages in Tafea (2021 and 2023), Sanma (2022) and Shefa (2023 and 2024). Stools were collected from 137 to 932 participants per survey, and quantitative PCR was used to estimate cluster adjusted prevalence of the different hookworm species and *S. stercoralis*.

Tafea had the highest hookworm prevalence: 33.6% (range:19.8-50.8%) pre-MDA and 35.5% (range: 25.4-47.1%) post-MDA. In Shefa, the pre-MDA hookworm prevalence was 22.8% (range:14.2-34.6%), and 11.5% post-MDA (range:7.2-17.8%). In Sanma, hookworm prevalence was 19.1% (range:12.8-27.4%). *Necator americanus* was the predominant hookworm in Tafea (pre-MDA: prevalence 31.4%, range: 17.8-49.2%; post-MDA: 34.9%, range: 24.6-46.8%). Notably, *Ancylostoma ceylanicum* was the most prevalent hookworm in Sanma (prevalence 12.1%, range:2.8–24.4%) and similar to *N. americanus* in Shefa (prevalence 11.9%, range: 9.1-15.5% pre-MDA; 6.3%, range:3.7-10.7% post-MDA). Most area councils had *S. stercoralis* prevalence <5%, although several areas in Shefa exceeded 10%.

Many area councils in the three provinces remain at risk for hookworm infection, including the zoonotic hookworm. PCR identified priority areas for interventions targeting zoonotic reservoirs and the use of ivermectin for strongyloidiasis.

ID: 233 / CP20: 4

Contributed abstract

Conference Topics: Epidemiology, Malaria

Keywords: Plasmodium knowlesi, IgG antibody response, classification algorithm, serological surveillance

Lactate Dehydrogenase antigen detection tool for symptomatic and cryptic infections of *Plasmodium vivax*

Anju Abraham^{1,2}, Vijaiashree Rajesh^{1,2}, Ivo Mueller^{1,2}, Lauren Smith^{1,2}, Rhea Longley^{1,2}

¹Immunity & Global Health Division, Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria, Australia;

²Department of Medical Biology, University of Melbourne, Melbourne, Victoria, Australia

Plasmodium vivax presents a unique challenge to malaria elimination due to its dormant liver stage and high prevalence of asymptomatic, low-density infections. These features hinder accurate diagnosis and surveillance, particularly in the Asia-Pacific region, where the *P. vivax* burden remains high. We have developed and validated *P. vivax*-specific antibodies to serve as serological exposure markers (SEMs) capable of identifying individuals with recent exposure to *P. vivax*. However, these SEMs misclassify some individuals with current blood-stage infections due to the delay in peak antibody responses. To address this gap, we established a *P. vivax*-specific lactate dehydrogenase (PvLDH) antigen detection assay to complement the existing SEMs. In cohort studies, the PvLDH assay identified 78.5% and 71.8% of PCR-positive infections in Thailand and Ethiopia, respectively. While the assay performs well for active infections, its sensitivity is limited in detecting low-density, asymptomatic cases, making it currently unsuitable as a standalone surveillance tool. However, detection of PvLDH in two individuals at PCR-negative timepoints suggests potential utility in uncovering hidden parasite dynamics. This assay represents a promising diagnostic addition and a valuable research tool for understanding *P. vivax* biology. Ongoing optimisation could enhance its role in integrated surveillance strategies to accelerate malaria elimination efforts.

ID: 230 / CP20: 5

Contributed abstract

Conference Topics: Epidemiology, Malaria

Keywords: Knowlesi malaria, Zoonotic Malaria, Serological surveillance, Antigen validation, IgG antibodies

Integrating *Plasmodium knowlesi* Serology into Multiplex Panels to Strengthen Malaria Surveillance

Zi Kang Ooi¹, Anju Abraham¹, Caitlin Bourke¹, Giri S. Rajahram², Timothy William², Benjamin A. Seager¹, Stephen W. Scally¹, Pailene S. Lim¹, Jetsumon Sattabongkot³, Kevin Tetteh⁴, Gavin J. Wright⁵, Chris Drakeley⁶, Nicholas Anstey⁷, Matthew Grigg⁷, Rhea J. Longley¹

¹Walter and Eliza Hall Institute of Medical Research, Parkville, VIC, Australia; ²Infectious Diseases Society of Kota Kinabalu, Sabah, Malaysia; ³Mahidol Vivax Research Unit, Faculty of Tropical Medicine, Mahidol University, Bangkok, Thailand; ⁴FIND Global Alliance for Diagnostics, Geneva, Switzerland; ⁵Department of Biology, Hull York Medical School, York Biomedical Research Institute, University of York, York, United Kingdom; ⁶Department of Infection Biology, London School of Hygiene & Tropical Medicine, London, United Kingdom; ⁷Global and Tropical Health Division, Menzies School of Health Research, Charles Darwin University, Darwin, Northern Territory, Australia

Malaria elimination in Southeast-Asia is threatened by *Plasmodium knowlesi*, with clinical cases underestimated despite potential severity. Limited molecular surveillance outside of Malaysia necessitates alternative tools to monitor transmission. Serological surveillance shows promise for monitoring falciparum and vivax malaria transmission. Integrating *P. knowlesi*-specific antigens into *Plasmodium* multiplex panels requires validating specificity against cross-reactivity species. Twelve *P. knowlesi* antigens were selected, including some designed to minimise *P. vivax* cross-reactivity. To assess immunogenicity and specificity, *P. knowlesi* antigens were coupled to MagPlex beads and tested in patients with confirmed infections (33 *P. knowlesi*, 31 *P. falciparum*, 31 *P. vivax*, and 21 *P. malariae*) at Day 0, 7, and 28 post-treatment, plus 27 non-endemic and 90 healthy control samples. Samples were analysed using Luminex MAGPIX Multiplexing. Three *P. knowlesi* antigens showed significantly higher antibody responses at Day 7 in *P. knowlesi* vs. *P. vivax* patients ($p < 0.05$, Mann-Whitney Test). IgG antibodies against PkMSP10, PkSSP2, and Pk8 increased >4.5-fold at Day 7 vs. Day 0. Other antigens showed low immunogenicity or high cross-reactivity in *P. vivax*. These three antigens should be integrated into multiplex panels alongside a *P. knowlesi*-specific machine-learning classification algorithm. This study provides robust tools to assess *P. knowlesi* exposure and inform large-scale serosurveillance.

CP21: Livestock Parasites 15 min talks

Time: Thursday, 03/July/2025: 1:30pm - 3:00pm · *Location:* Conference room 3

Session Chair: Emily Francis, The University of Sydney

Session Chair: Johann Schroder, Gemini R&D Services

ID: 178 / CP21: 1

Contributed abstract

Conference Topics: Bioinformatics, Diagnostics, Genomics, Livestock Parasites, Protozoa, Veterinary Parasitology

Keywords: Trichomonas foetus, bovine trichomonosis, long-read sequencing, Oxford Nanopore Technologies, metagenomics

Detection of *Trichomonas foetus* in Bulls Using Long-Read Sequencing

Zhetao Zhang¹, Ala E. Tabor¹, Gry B. Boe-Hansen^{1,2}, Hannah V. Siddle¹, Chian Teng Ong¹

¹The University of Queensland, Queensland Alliance for Agriculture & Food Innovation, Centre for Animal Science, St Lucia 4072, Queensland, Australia; ²The University of Queensland, School of Veterinary Science, Gatton, Queensland 4343, Australia

Bovine trichomonosis, caused by the protozoan parasite *Trichomonas foetus*, is a venereal disease of significant economic concern for cattle industries globally. While asymptomatic in bulls, the parasite causes subfertility and early embryonic loss in cows. In Australia, where no licensed vaccine exists, control measures rely heavily on accurate diagnosis and culling of infected bulls. Current diagnostic approaches - culture and qPCR - are hindered by the complex microbial environment of the bull prepuce. These methods often result in false negatives, due to the failure to revive *T. foetus* or degraded DNA quality, and false positives, stemming from co-cultured or cross-reactive trichomonads. To address these limitations, we explored the utility of long-read sequencing (Oxford Nanopore Technologies, ONT) combined with a metagenomics approach for improved detection of *T. foetus*. Using mock samples spiked with *T. foetus* DNA and field samples from bulls previously confirmed positive via qPCR, we demonstrate that ONT sequencing has great potential to offer enhanced sensitivity and specificity for *T. foetus* detection. This approach would enable direct detection of *T. foetus* within the microbial community, minimising diagnostic ambiguity, becoming a robust diagnostic alternative for bovine trichomonosis, strengthening disease surveillance, and safeguarding the Australian cattle industry.

ID: 235 / CP21: 2

Contributed abstract

Conference Topics: Epidemiology, Veterinary Parasitology

Keywords: Dairy goats, causal inference, Eimeria, prevalence, risk factors

Epidemiological insights into *Eimeria* species in Australian dairy goats using causal inference model estimates

Endris Ali¹, Abdul Ghafar¹, Mark A. Stevenson¹, Sandra Baxendell², Ian Beveridge¹, Abdul Jabbar¹

¹Melbourne Veterinary School, The University of Melbourne, Melbourne, Victoria, Australia; ²Goat Veterinary Consultancies-goatvetoz, Brisbane, Queensland, Australia

Coccidiosis, a parasitic disease caused by *Eimeria* species, is a significant health and production constraint for goats worldwide. Very little is known about coccidiosis in Australian goats. This study investigated the prevalence and risk factors associated with *Eimeria* spp. in Australian dairy goats. Between November 2023 and July 2024, 1,188 individual faecal samples were collected across 71 dairy goat farms and analysed using the modified McMaster technique to quantify oocysts per gram (OPG) in faeces. Seventeen risk factors for infection with *Eimeria* spp. were assessed through multivariable mixed-effects logistic regression analysis, guided by a directed acyclic graph. The overall prevalence of *Eimeria* spp. was 68.5%. Kids exhibited the highest prevalence (76.5%) followed by weaners (75.1%) and adults (65.0%). The mean OPG of *Eimeria* spp. was also significantly higher in kids (14,437) than in weaners (3,741) and adults (242). Age, sex, climatic zone, stocking density, bedding type and the frequency of changing bedding significantly influenced the prevalence of *Eimeria* spp. This study provides the first report on the prevalence of *Eimeria* spp. and associated risk factors in Australian dairy goats. Knowledge of key risk factors could be used to inform targeted management strategies to control coccidiosis in Australian dairy goat farms.

ID: 270 / CP21: 3

Contributed abstract

Conference Topics: Ectoparasites, Host-parasite interactions, Livestock Parasites, Veterinary Parasitology

Keywords: Blowfly, Host immune response, Sheep, Proteomics

Proteomic analysis to understand host immune response in the early stage of flystrike in sheep.

Sugandhika Welikadage¹, Ying Ting Yang², Shilpa Kapoor¹, Clare Anstead¹, Jean-Pierre Scheerlinck¹, Trent Perry², Vern Bowles¹

¹Melbourne Veterinary School, The University of Melbourne; ²School of Biosciences, Faculty of science, The University of Melbourne

This study investigated the host response in the skin during the early stages of flystrike with the aim of identifying key proteins involved at this time. Fourteen 3-year-old Merino sheep were experimentally challenged with blowfly (*Lucilia cuprina*) eggs and the protein profiles of larval-challenged skin sites were compared to mock-treated control sites. Proteomic analysis identified 526 sheep proteins with 132 significantly different between challenged and control sites. In addition, 105 proteins showing increased abundance in response to larval infestation. Among the 105 proteins, 23% exhibited protease inhibitor activity, 14% had anticoagulant properties, 21% were associated with the innate immune response including antibacterial defense proteins and 13% were involved in iron-binding. This study highlights the role of protease inhibitors, inflammatory proteins and wound-healing factors in the early response to a flystrike infection.

ID: 186 / CP21: 4

Contributed abstract

Conference Topics: Helminthology, Livestock Parasites, Veterinary Parasitology

Keywords: Haemonchus contortus, Pteridium esculentum, anthelmintic, Ethnoveterinary medicine

INHIBITION OF HAEMONCHUS CONTORTUS LARVAL DEVELOPMENT FROM PTERIDIUM ESCULENTUM EXTRACTS

Elouise Bliss

Federation University, Australia

Haemonchus contortus is a blood-feeding helminth residing in the abomasum (fourth stomach) of small ruminants, including sheep, goats and alpacas, and is the most pathogenic of ruminant parasitic infections.

The primary treatment is oral application of chemical anthelmintic drugs and extensive use has led to increased drench in Australia and overseas. Therefore, novel anthelmintic options need to be explored.

Ethnoveterinary medicine takes into account the traditional knowledge and practices of Indigenous plants in the treatment of veterinary diseases and applying such knowledge may help discover novel anthelmintic compounds.

The Bracken family (*Pteridium spp.*) is well documented as a toxic plant to livestock when grazed in large quantities however Indigenous application of the Southern Hemisphere species (*Pteridium esculentum*) suggests its use as a 'hard time' food source and a Bush Medicine for the treatment of digestive discomfort such as cramps and diarrhoea. Currently, no research has been conducted on the possible anthelmintic effects of *Pteridium esculentum*.

The methanol extracts of fronds and roots have an IC50 of 29.10ug/mL and 19.49ug/mL respectively. The larval development assays on the hydrosol and tea are still being conducted as well as toxicity analysis.

ID: 220 / CP21: 5

Contributed abstract

Conference Topics: Ectoparasites, Host-parasite interactions, Immunology, Livestock Parasites, Vaccines

Keywords: Bovine, cattle ticks, immune response, vaccine

Evaluating the bovine immune response to a novel tick vaccine

Mikayla Crouch¹, Hannah V Siddle², Ala Tabor^{1,2}

¹The University of Queensland, School of Chemistry & Molecular Biosciences, St Lucia 4072, Queensland, Australia; ²The University of Queensland, Queensland Alliance for Agriculture & Food Innovation, Centre for Animal Science, St Lucia 4072, Queensland, Australia

The ability of a vaccine to elicit a strong immune response is key to its success. Poor longevity of previous commercialised tick vaccines inhibited industry uptake. Thus, it is a priority to better understand the bovine immune response. The humoral, or B cell, arm of the adaptive immune system is well reported in tick vaccine trials as IgG titres from ELISA's. This project is part of a proof-of-concept trial for a novel tick vaccine and hopes to expand understanding of the cellular (T cell) component of bovine immune responses through major histocompatibility complex (MHC) genotyping.

MHC II molecules bind and present antigens to T-cells. Given the high polymorphism of MHC II genes, it is hypothesised that variation in MHC II alleles will influence antigen presentation. Furthermore, cattle with an MHC II allele that exhibits the highest affinity for tick protein antigens, will be expected to produce the greatest amount of antibodies. Variation in vaccine efficacy could thus be explained in part by the relationship between MHC II alleles and IgG production. For commercial application, it would be favourable that the tick antigens used in this novel vaccine can effectively bind a range of MHC II alleles.

ID: 224 / CP21: 6

Contributed abstract

Conference Topics: Ectoparasites, Molecular Biology, Veterinary Parasitology

Keywords: Australian sheep blowfly, flystrike, bacteria, 16s RNA sequencing

Comparative Analysis of the Microbial Communities of Rural and Urban *Lucilia cuprina* Subspecies Using 16S rRNA Gene Sequencing

Shilpa Kapoor^{1,2,3}, Ying Ting Yang³, Aaron R. Jex^{1,2}, Philip Batterham³, Robin B. Gasser¹, Vernon M. Bowles¹, Trent Perry³, Clare A. Anstead¹

¹Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Parkville, VIC 3010, Australia; ²Population Health and Immunity, Walter and Eliza Hall Institute of Medical Research, Parkville, VIC 3052, Australia; ³Bio21 Molecular Science and Biotechnology Institute, The University of Melbourne, Parkville, VIC 3010, Australia

Blowflies of the genus *Lucilia* (Calliphoridae) hold significant importance in forensic science, medical research, and agriculture, due to their role as facultative ectoparasites of domesticated sheep. The Australian sheep blowfly, *L. cuprina dorsalis*, is a primary causative agent of cutaneous myiasis, causing substantial economic losses in the wool industry. In contrast, the closely related subspecies *L. c. cuprina*, does not induce myiasis in sheep but is a necrophagous species in urban regions of Australia. The bacterial communities of these blowflies play crucial roles in their physiology, behavior, and host interactions; however, our understanding of these communities is limited. This study characterized the microbiomes of *L. c. cuprina* (urban) and *L. c. dorsalis* (rural) using 16S rRNA gene sequencing. The findings indicate that while both subspecies harbor a broadly similar microbial composition, their respective urban and rural environments influence the prevalence of specific bacterial genera. For example, the genus *Ignatzschineria* is highly abundant in *L. c. dorsalis*, whereas *Pseudomonas* is more prevalent in *L. c. cuprina*. These findings provide fundamental insights into the microbial dynamics of *Lucilia* species, contributing to a broader understanding of their ecological and biological roles, with potential applications in forensic entomology, medical research, and agricultural pest management.

CP19.1: Cell & Molecular Biology 5 min talks

Time: Thursday, 03/July/2025: 2:30pm - 3:00pm · *Location:* Conference room 1

Session Chair: Claire Sayers, University of New South Wales

Session Chair: Colin Sutherland, LSHTM London

ID: 125 / CP19.1: 1

Contributed abstract

Conference Topics: Malaria, Molecular Biology

Keywords: malaria, transmission, population dynamics, barcoding

Cellular barcoding of malaria parasites to define the population dynamics of transmission

Claire Sayers, Jake Baum

School of Biomedical Sciences, University of New South Wales, Sydney

Malaria parasites must transmit from vertebrate hosts to mosquito vectors to spread disease. The parasites experience several population bottlenecks throughout their lifecycle. Most notably, the population can drop from millions of parasites in the host blood to single parasites in the mosquito midgut. Another significant bottleneck occurs when an infected mosquito injects salivary gland sporozoites into the dermis of a new host, where only a small proportion of parasites will successfully infect the liver. These population dynamics are critical to the success of current malaria control strategies, but are also vital for research into new ways of preventing and eradicating malaria. Despite this, population bottlenecks of malaria parasites have never been quantitatively investigated. In this pilot, we introduced 10 unique barcodes into a dispensable region of a rodent malaria parasite genome to create a population of barcoded parasites. The barcoded parasites were transmitted to mosquitoes, and back to mice, and genomic DNA samples were taken for barcode sequencing. Our results show that the barcodes are successfully transmitted, so we can scale-up to using thousands of barcodes to define population bottlenecks. Future work will include testing different malaria parasite infection routes and determining how malaria drugs and vaccines affect population dynamics.

ID: 219 / CP19.1: 2

Contributed abstract

Conference Topics: Apicomplexa Biology, Biochemistry, Cell Biology, Molecular Biology

Keywords: Nutrient uptake, RNA modification, Translational Control, Apicomplexa, Toxoplasma

Elucidating mechanisms behind the regulation of arginine uptake in the intracellular parasite

Toxoplasma gondii

F. Victor Makota, Giel van Dooren

Research School of Biology, The Australian National University, Canberra, ACT, Australia

Arginine is an essential nutrient for the proliferation of *Toxoplasma gondii* parasites. We have shown previously that parasites encounter different arginine concentrations as they infect different organs of their host, and that parasites respond by tightly controlling arginine uptake. They do this through regulating the expression of the major plasma membrane-localised arginine transporter, *TgApiAT1*. In arginine-replete conditions, *TgApiAT1* expression is decreased, whereas its abundance increases when arginine concentrations are low. *TgApiAT1* regulation is mediated post-transcriptionally by an upstream open reading frame in the *TgApiAT1* transcript. To better understand how parasites sense and respond to arginine availability in their environment, we have developed an arginine-sensitive fluorescence-based reporter strain of *T. gondii*. Using this strain, we have undertaken a flow cytometry-based positive selection genome-wide CRISPR/Cas9 screen to identify genes important for arginine-dependent *TgApiAT1* regulation. This screen identified a candidate nucleolar RNA methyltransferase as being important for repressing *TgApiAT1* expression when arginine is abundant, and we are now developing approaches to understand the molecular mechanisms of how it functions in mediating arginine-dependent expression of *TgApiAT1*. Overall, our findings highlight ways in which a model intracellular pathogen responds to nutrient availability in their environment.

ID: 265 / CP19.1: 3

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Malaria, Microscopy

Keywords: malaria, plasmodium, EdU, DNADetect, probes

Thymidine kinase-independent click chemistry DNADetect™ probes for assessment of DNA proliferation in malaria parasites and mammalian cells

Jacinta Macdonald¹, David Hilko¹, Gillian Fisher¹, Tina Skinner-Adams^{1,2}, Sally-Ann Poulsen^{1,2}, Katherine Andrews^{1,2}

¹Institute for Biomedicine and Glycomics, Griffith University, Nathan, Brisbane, Queensland 4111, Australia; ²School of Environment and Science, Griffith University, Nathan, Brisbane, Queensland 4111, Australia

The alkyne modified thymidine analogue 5-ethynyl-2'-deoxyuridine (EdU) is a gold standard chemical probe for DNA synthesis and proliferation detection in mammalian cells. EdU is incorporated into nuclear DNA via the thymidine salvage pathway and can be detected using copper catalysed azide-alkyne cycloaddition (CuAAC) with a fluorescent azide. However, as *Plasmodium malariae* parasites lack the thymidine kinase enzyme that allows metabolism, EdU and similar probes (e.g., BrdU) cannot be utilised. While *P. falciparum* engineered to express thymidine kinase from *Herpes simplex* virus can overcome this limitation and enable DNA labelling studies using BrdU and EdU, this approach may not be feasible for the analysis of different *Plasmodium* species, multiple laboratory lines and field isolates. To overcome this, we previously reported novel thymidine-based probes that bypass thymidine kinase and that are membrane-permeable due to their masked-monophosphate 'pronucleotide' design¹, showing that these DNADetect™ probes robustly label replicating asexual intraerythrocytic stage *P. falciparum* parasites. To expand the utility of these DNADetect™ probes, data will be presented on the labelling efficiency of these probes compared to EdU in mammalian cells using flow cytometry and microscopy, as well as analysis of parasites species for which these probes have applicability.

ID: 187 / CP19.1: 4

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Malaria, Microscopy, Molecular Biology

Keywords: Cytostome, Kelch-13, Gametocyte, Microscopy, Organelle

Investigating the role of Kelch-13 during sexual development in *P. Falciparum*

Sophie Collier, Haowen Deng, Long Huynh, Stuart Ralph

The University of Melbourne, Australia

In *P. Falciparum*, resistance to the frontline antimalarial artemisinin is predominantly caused by mutations in the Kelch-13 (K13) protein. During asexual development, K13 plays an important role in the formation, stability and function of a double-membraned invagination called the cytotome, whose role is to facilitate uptake of haemoglobin from the host red blood cell into the parasite. This provides the parasite with essential amino acids required for growth and simultaneously releases haem-iron as a bioproduct which is crucial for artemisinin activation. Whilst K13 and its role in haemoglobin uptake is well studied during asexual development, its role during sexual blood, mosquito and liver stages are yet to be investigated.

Using fluorescence microscopy, we show that K13-GFP displays a dynamic localisation pattern throughout gametocytogenesis, with several K13-GFP puncta often converging to form a unique rod-shaped structure in late-stage gametocytes. Similarly, we are using lattice light-sheet microscopy to track the fate of K13-GFP and cytotomes during gametogenesis. Using a knock-sideways approach, we will study the biological consequences of K13 mislocalisation on cytotome formation and parasite growth. This work helps to elucidate the biology of cytotomes and determine whether they serve a function other than haemoglobin uptake during other life cycle stages.

ID: 180 / CP19.1: 5

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Drugs, Host-parasite interactions, Malaria, Molecular Biology, Proteomics, Vaccines

Keywords: Plasmodium falciparum, rhoptry, proteomics, TurboID proximity labelling, cell biology

Functional Characterisation of Plasmodium Rhoptry Proteins

Bernard Nathaniel, Christina Dizdarevic, Joyanta Modak, Natalie Counihan, Tania de Koning-Ward

Institute for Mental and Physical Health and Clinical Translation (IMPACT), School of Medicine, Deakin University, Waurn Ponds, Victoria, Australia

Malaria, caused by *Plasmodium* parasites, has been health and economic burdens for decades. In the human host, *Plasmodium* invade and remodel red blood cells (RBCs) to facilitate growth and replication. Rhoptries, one of apical organelles, are involved in invasion, parasite establishment within the RBCs, and host cell remodelling, rendering rhoptry proteins promising therapeutic targets. However, mechanistic insight into rhoptry biology (e.g., protein trafficking, secretion, and function) remains rudimentary, with only 45 *Plasmodium* rhoptry proteins identified to date. The upregulation of hundreds of genes prior to invasion suggests many more rhoptry proteins await discovery.

Rhoptry proteins have been previously identified by *ad hoc* approaches. Here, advanced proximity labelling techniques (i.e. TurboID) to identify rhoptry-localised proteins are being utilised. By fusing TurboID to known rhoptry proteins in various regions (neck, bulb, and cytoplasmic face), this study aims to decipher the complete rhoptry proteome. Using this approach, 129 proteins, including 15 known rhoptry proteins, were significantly enriched in CERL1-TurboID-expressing parasites, a rhoptry cytoplasmic face protein. Ongoing work tagging other rhoptry proteins and functionally characterising novel proteins will delineate the complete rhoptry proteome and provide insights into parasites invasion and survival within host cells, from which potential drug and vaccine targets can be evaluated.

CP20.1: Diagnostics 5 min talks sponsored Abacus dx

Time: Thursday, 03/July/2025: 2:45pm - 3:00pm · Location: Conference room 2
Session Chair: Kelly Ly, Abacusdx

ID: 218 / CP20.1: 1

Contributed abstract

Conference Topics: Diagnostics, Malaria

Keywords: Malaria hrp2/3 gene deletion

Gene Deletions in Malaria Parasite Trigger Diagnostic Policy Reforms in Ethiopia

Sindew Mekasha Feleke^{1,2}, Bacha Mekonen², Geremew Tasew², Jonathan B. Parr³, Hiwot Solomon⁴

¹La Trobe University, Australia; ²Ethiopian Public Health Institute (EPHI); ³University of North Carolina at Chapel Hill (UNC), USA; ⁴Federal Ministry of Health, Ethiopia

Plasmodium falciparum parasites with deletions in the histidine-rich protein 2 and 3 (pfrp2/3) genes pose a significant challenge to malaria elimination efforts in Africa. To assess the prevalence of these deletions in Ethiopia, we conducted a large-scale study in 2019 using the WHO's pfrp2/3 deletion surveillance protocol combined with deep sequencing. The study enrolled 12,572 malaria-suspected cases from health facilities in Amhara, Tigray, and Gambella regions, using two WHO pre-qualified RDTs. Of the 2,704 confirmed *P. falciparum* infections, 350 (13%) were flagged as potential pfrp2/3 deletions (PfHRP2-, Pf-pLDH+). Molecular testing of 176 discordant samples revealed that 113 (64%) were confirmed to have pfrp2/3 deletions. In addition, the second phase of the 2021 nationwide survey, enrolling 22,500 subjects, found 7.1% of *P. falciparum* cases (1458 subjects) had PCR-confirmed pfrp2 deletions. This large-scale surveillance highlights the widespread presence of pfrp2/3 gene deletions in Ethiopia and has led to policy revisions by the Ministry of Health, shifting from HRP2-detection RDTs to non-HRP2-detection RDTs, in accordance with WHO guidelines.

ID: 113 / CP20.1: 2

Contributed abstract

Conference Topics: Bioinformatics, Diagnostics, Ectoparasites, Host-parasite interactions, Molecular Biology, One Health

Keywords: MicroRNA, Biomarkers, Vector-borne diseases, Systematic review, Disease vectors

MicroRNAs as Emerging Potential Biomarkers for Parasitic Vector-Borne Diseases: A Systematic Review

Tilini K. De Silva¹, John Stenos², Stephen Graves², Tarka Bhatta², Nathan Unsworth³, Cameron Stewart⁴, Ryan Farr⁴, Simon Firestone¹

¹Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Parkville, VIC 3052, Australia.; ²Australian Rickettsial Reference Laboratory, Barwon Health, University Hospital Geelong, Geelong, VIC 3220, Australia.; ³Sensors and Effectors Division, Defence Science and Technology Group, 506 Lorimer Street, Fisherman's Bend, Melbourne, VIC 3207, Australia.; ⁴CSIRO Health & Biosecurity, Australian Centre for Disease Preparedness, Geelong, VIC 3220, Australia.

Vector-borne diseases (VBDs) pose substantial public health challenges due to their multifaceted transmission dynamics, underscoring the necessity for precise diagnostic strategies for disease management. MicroRNAs (miRNAs), a class of small non-coding RNAs, have recently emerged as promising biomarkers for various diseases. This systematic review aimed to analyse the current research on miRNAs in humans and arthropod vectors that could serve as potential biomarkers for the diagnosis of VBDs. A search of four electronic databases resulted in the identification of 76 qualifying articles. Many parasitic VBDs including babesiosis, filariasis, leishmaniasis, malaria, schistosomiasis, and trypanosomiasis were analysed. Overlaps of differentially expressed miRNAs were reported by *in-vivo* human studies including miR-208a and miR-21-5p in trypanosomiasis patients. Specific miRNA panels were identified as potential biomarkers, including hsa-miR-150-5p and hsa-miR-15b-5p for detecting *P. vivax* malaria, and sma-miR-bantam and sma-miR-2c-3p for detecting schistosomiasis. The analyses of miRNAs in ectoparasitic vectors including ticks, revealed diverse expression patterns, contributing to the understanding of life stage-specific, species-specific, and organ-specific miRNAs. This systematic review highlights the gaps and opportunities in the application of miRNA biomarkers for VBDs. While miRNAs hold potential, further studies are needed to demonstrate proof of concept, establish standardized protocols, and validate their use in clinical settings.

ID: 114 / CP20.1: 3

Contributed abstract

Conference Topics: Diagnostics, Molecular Biology, Protozoa

Keywords: CRISPR, trichomonads, periodontitis, trichomoniasis, point-of-care

Developing point-of-care assays for *Trichomonas* spp. using CRISPR.

Joshua Slattery^{1,2}, Martin Pal^{1,2}, Bernd Kalinna¹

¹Rural Health Research Institute, Charles Sturt University, Orange NSW; ²School of Dentistry and Medical Sciences, Charles Sturt University, Wagga Wagga NSW

The diagnosis of trichomonad infections still heavily relies on low-sensitivity microscopy. Whilst PCR-based point-of-care (POC) methods are advancing, turn-around-times currently do not meet patient needs in remote settings. Unfortunately, in Australia those living in remote communities are the most affected by these infections. *T. vaginalis* infection causes an increased risk of cancers. Meanwhile, *T. tenax* is potentially causative in periodontal disease, though, low-sensitivity methods have hampered investigations. CRISPR-based diagnostics, however, provide a highly specific and sensitive method, practical in a point-of-care setting.

Two diagnostic assays are being developed for *T. vaginalis* and *T. tenax*. These assays will utilise the CRISPR-associated protein Cas12a, guided by species-specific RNA guide sequences to cleave target DNA. By virtue of a secondary nuclease activity, Cas12a will simultaneously cleave a ssDNA reporter, producing a detectable signal.

So far, DNA extracted from cultured *T. tenax* has been detected by a plate-based fluorescence assay. Further work is underway to incorporate isothermal amplification, and re-package the assay into a POC-capable lateral-flow-based assay.

Additionally, the *T. vaginalis* genome is being assessed by bioinformatic methods for suitable DNA targets to which RNA guides may be designed.

These assays will detect trichomonads in patients at the point-of-care, facilitating rapid diagnosis and treatment.

CP22: Omics 15 min talks

Time: Thursday, 03/July/2025: 3:30pm - 4:30pm · Location: Conference room 1

Session Chair: Shilpa Kapoor, The University of Melbourne

Session Chair: Aaron Jex, WEHI

ID: 172 / CP22: 1

Contributed abstract

Conference Topics: Bioinformatics, Drugs, Protozoa

Keywords: Kinase, Drug discovery, Giardia, Protein annotation

Annotation of the *Giardia duodenalis* Kinome: Perspectives for Kinase Drug-Targets

Alex Lam^{1,2}, Isabelle Lucet^{2,3}, Aaron Jex^{1,2,4}, Samantha Emery-Corbin⁵

¹Infection and Global Health Division, WEHI, Melbourne, Australia; ²Department of Medical Biology, University of Melbourne, Melbourne, Australia; ³Chemical Biology Division, WEHI, Melbourne, Australia; ⁴Faculty of Science, University of Melbourne, Melbourne, Australia; ⁵Monash Proteomics & Metabolomics Platform, Monash Biomedicine Discovery Institute & Department of Biochemistry and Molecular Biology, Monash University, Melbourne, Australia

The gastrointestinal parasite *Giardia duodenalis* causes ~200 million symptomatic infections annually, with chemotherapeutic interventions limited to nitroheterocyclic antibiotics such as metronidazole. However, drug-resistant treatment failures occur in up to 20% of cases, highlighting the urgency of novel chemotherapeutics. Kinases are attractive drug-targets, with demonstrated success as chemotherapeutics in human diseases. This has extended into anti-parasitic research, however the first and only annotation of the *G. duodenalis* kinome was built upon a fragmented genome. Recent advancements in protein structure prediction and the publication of the accurate chromosomal-scale assembly *G. duodenalis* genome necessitates a reannotation of this kinome. We utilise extensive HMM-based sequence profiling and AlphaFold-supported structural comparisons on this renewed *G. duodenalis* kinome, conservatively identifying 264 *G. duodenalis* kinase domains. We identified 177 kinases of ambiguous phylogeny, some to the Never-in-Mitosis A kinase (Nek) -HMMs and the bacterial protein kinase B (PknBs) -HMMs. With sequence motifs differing to the canonical kinase domain observed throughout evolution, we propose adapting a different nomenclature for these kinases based on likely functions and protein-protein interactions, we broadly term these non-core protein kinases (ncPKs). We provide insights which may serve as first-steps in targeting these likely *Giardia*-specific ncPKs, to developing safe, selective and efficacious anti-giardials.

ID: 183 / CP22: 2

Contributed abstract

Conference Topics: Drugs, Malaria

Keywords: Malaria, Drug discovery, Metabolomics, Proteomics

Chemoproteomics and metabolomics-based discovery of drug targets for antimalarial candidates

Carlo Giannangelo, Abbey McCorquodale, Chris Taylor, Jennifer Le, Darren Creek

Drug Delivery, Disposition and Dynamics, Monash Institute of Pharmaceutical Sciences, Monash University, Parkville, 3052, Australia

Phenotypic screening has successfully identified thousands of molecules that kill malaria parasites. However, most of these hits act by unknown mechanisms, making further development challenging. To address this, we have developed a collection of omics-based strategies to elucidate drug mechanisms and molecular targets on a system-wide scale.

We applied these strategies to a phenotypically active cyclopropyl amide series with slow asexual activity kinetics, multistage antimalarial activity and unknown function. Untargeted metabolomics initially revealed that two exemplars, MMV1804508 and MMV1804743, specifically perturb parasite pyrimidine biosynthesis. Solvent proteome profiling coupled to quantitative proteomics analysis identified parasite cytochrome *bc1* (*cytbc1*) as the target from an unprecedented ~4,500 proteins detected from a parasite lysate. Verifying *cytbc1* as the target, proguanil reversed MMV1804508 resistance in *P. falciparum* SB1-A6 parasites that are pan-resistant to mitochondrial electron transport chain inhibitors and MMV1804508 inhibited *cytbc1* activity from a parasite mitochondrial extract. Surprisingly, cross-resistance studies against several *cytb* mutants revealed these compounds are not susceptible to the same resistance mutations that thwart atovaquone and other investigational *cytb* inhibitors.

Combined, cyclopropyl amides deprive the parasite of essential pyrimidine nucleotides by inhibiting *cytbc1*. Our study demonstrates the power of multi-omics to reveal complex drug mechanisms and facilitate drug discovery for malaria.

ID: 151 / CP22: 3

Contributed abstract

Conference Topics: Apicomplexa Biology, Biochemistry, Malaria, Proteomics

Keywords: systems biology, protein interactions, DNA replication

Generating a comprehensive map of protein-protein interactions in the malaria parasite

Christopher A. MacRaid¹, Ghizal Siddiqui¹, Meghan E. Zadow², Danny W. Wilson², Darren J. Creek¹

¹Drug Delivery, Disposition and Dynamics, Monash Institute of Pharmaceutical Sciences, Monash University, Parkville, Victoria, 3052, Australia; ²Research Centre for Infectious Diseases, School of Biological Sciences, University of Adelaide, Adelaide 5005, South Australia,

The Plasmodium parasites that cause malaria have evolved a highly specialised and divergent biology to accommodate a complex lifecycle spanning multiple insect and vertebrate host cells; much of this biology remains poorly understood. To

address this lack of knowledge, we are applying new proteomic approaches including Protein Correlation Profiling (PCP) and Cross-Linking Mass Spectrometry (XLMS) to *P. falciparum*, to establish a comprehensive map of protein-protein interactions. PCP and XLMS are complementary approaches to the study of protein interactions at proteome scale, but they have not been widely applied together, or outside of model organisms. We have developed new machine-learning protocols to integrate our PCP and XLMS datasets with existing data, resulting in a complexome covering almost half of the observable *P. falciparum* asexual blood-stage proteome. This significantly enhances existing datasets, comprising 3500 high-confidence interactions involving 919 proteins. Identified protein complexes illuminate diverse aspects of parasite biology including host cell remodelling, nutrient uptake and translational control. To illustrate the power of this dataset, we present a case study of the atypical DNA replication in schizogony. We identify highly divergent components of the parasite replisome, new and previously misannotated histone chaperones and illuminate the architecture of the parasite kinetochore.

ID: 189 / CP22: 4

Contributed abstract

Conference Topics: Drugs, Malaria, Proteomics

Keywords: Antimalarial, drug mechanism, proteomics, drug target

Elucidating the Mechanism of 2-Aminobenzimidazole Antimalarials Through Proteomics-Based Target Deconvolution

Darren Creek, Yunyang {Eileen} Zhou, Matthew Challis, Ghizal Siddiqui

Monash University, Australia

There is an urgent imperative for the discovery of new antimalarials with novel modes of action. A newly discovered class of antimalarial candidates, 2-aminobenzimidazoles (ABIs), exhibit remarkable potency against the erythrocytic stage of *Plasmodium falciparum*. To elucidate the molecular targets of ABIs, we employed proteomics-based target deconvolution approaches, specifically Solvent Profiling Proteomics (SPP) and Limited Proteolysis–Mass Spectrometry (LiP-MS).

SPP identified 10 proteins that were consistently stabilised in the presence of ABI compounds compared to controls. These were primarily proteins linked to translation and protein folding, including multiple 60S ribosomal proteins and protein disulfide isomerase (PF3D7_0827900). Complementary LiP-MS analysis identified 29 protein targets including the 60S acidic ribosomal protein P2 (PF3D7_0309600) and protein disulfide isomerase (PF3D7_0827900). These results suggest that ABI compounds interact with the parasite's protein translation and folding machinery.

These findings are further supported by untargeted proteomics performed on ABI-treated parasite cultures, which identified 100 significantly altered proteins, including 60S ribosomal proteins L12 (PF3D7_0517000) and L28 (PF3D7_1142500) previously implicated in the SPP experiments. Future research will focus on validating the impact of ABIs on protein translation, with the ultimate goal of designing novel antimalarials that act via novel mechanisms of action.

CP23: One Health 15 min talks

Time: Thursday, 03/July/2025: 3:30pm - 4:45pm · *Location:* Conference room 2

Session Chair: Breanna Knight, Murdoch University

Session Chair: Harsha Sheorey, St Vincent's Hospital, Melbourne

ID: 190 / CP23: 1

Contributed abstract

Conference Topics: Strongyloides

Keywords: strongyloidiasis, elimination, One Health

Setting the Platform for Strongyloidiasis Elimination in Australia

Darren Gray

QIMR Berghofer, Australia

Strongyloidiasis is a disease of poverty caused by parasitic nematodes and is considered to be the most neglected of the neglected tropical diseases. In Australia, *S. stercoralis* is the infecting species that disproportionately affects remote Indigenous communities, with the prevalence ranging from 10- 60%, although the true extent is unknown. Infection ranges from asymptomatic to fatal. The lifecycle and transmission are complex with both a parasitic cycle and free-living cycle. This is further complicated by autoinfection and the possibility of zoonotic transmission. These factors coupled with less than ideal diagnostics, and treatment options where there is evidence of treatment failures, the spectre of emerging resistance and the inability to prevent reinfection, all have implications for control and elimination efforts. The complexity of transmission, underlying causes and deficiencies in knowledge, treatment and diagnostics means there is no “quick fix” for strongyloidiasis in Australia (and the world). Multiple disciplines and sectors spanning the community, human and veterinary health, education and the environment will be required to come together to defeat this disease of the poor that should not be in Australia. Therefore, a One Health approach is required for its elimination.

ID: 289 / CP23: 2

Contributed abstract

Conference Topics: Helminthology, One Health, Strongyloides

Keywords: Strongyloides stercoralis, soil-transmitted helminth, dogs, one health

A Transmission Model for Understanding *Strongyloides stercoralis* Transmission Dynamics among Humans, Dogs, and the Environment

Mackrina Winslow¹, Vito Colella², Juan Pablo Villanueva-Cabezas^{1,3}, Patricia Therese Campbell¹

¹Department of Infectious Diseases, The University of Melbourne, at the Peter Doherty Institute for Infection and Immunity, Melbourne, Victoria, 3000, Australia; ²Melbourne Veterinary School, Department of Veterinary Biosciences, Faculty of Science,

The University of Melbourne, Victoria, 3000, Australia; ³The Nossal Institute for Global Health, The University of Melbourne, Melbourne, Victoria, 3000, Australia

Strongyloides stercoralis is a soil-transmitted helminth that affects over 600 million people and 6% of dogs worldwide. Its life cycle includes parthenogenesis, auto-infection, and environmental sexual reproduction. Molecular phylogenetic studies based on a short fragment of *S. stercoralis* genome have identified two genetic lineages: one that infects both humans and dogs and another found exclusively in dogs. However, the role of dogs as source of *S. stercoralis* infection to humans is still matter of debate. We developed a novel *S. stercoralis* compartmental transmission model exploring on the potential role of dogs in human infection. The model incorporates two environmental reservoirs, dogs, and humans stratified by age. We used Cambodian data to calibrate the model and explored scenarios with varying lineage prevalence in dogs. Parameter combinations that reproduced these scenarios were generated using Latin Hypercube Sampling and simulated to project the likely impact of interventions targeting humans and/or dogs. Treating children and adults annually for three years reduced human prevalence by 85%, compared to 5% with children-only treatment. MDA needed to be restarted after five years of community-wide MDA due to rebound. Future work will explore different coverage and efficacy levels in both humans and dogs to further refine control strategies.

ID: 280 / CP23: 3

Contributed abstract

Conference Topics: Epidemiology, Helminthology, Molecular Biology, One Health, Parasites of companion animals, Strongyloides, Zoonoses

Keywords: Strongyloides, control, nanopore, soil-transmitted heminth

From Tails to Toes: A next-generation sequencing approach to assess the genetic variability of *Strongyloides* spp. infections in children, dogs and long-tail macaques in Kampong Chhnang, Cambodia

Patsy A. Zendejas-Heredia¹, Molyden Vann², Lucas G. Huggins¹, Shannon M. Hedtke³, Richard Bradbury⁴, Warwick N. Grant³, Robin B. Gasser¹, Rekol Huy², Martin Walker^{5,6}, Rebecca J. Traub¹, Virak Khieu², Vito Colella¹

¹Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Parkville, Victoria, Australia; ²National Centre for Parasitology, Entomology and Malaria Control, Ministry of Health, Phnom Penh, Cambodia; ³Department of Environment and Genetics, School of Agriculture, Biomedicine and Environment, La Trobe University, Bundoora, Victoria, Australia; ⁴Australian Institute of Tropical Health and Medicine, James Cook University, Queensland Australia.; ⁵Department of Pathobiology and Population Sciences, Royal Veterinary College, University of London, United Kingdom.; ⁶London Centre for Neglected Tropical Disease Research, Department of Infectious Disease Epidemiology, Imperial College London, London, United Kingdom.

Strongyloidiasis affects an estimated 600 million people globally; yet, many aspects of its epidemiology remain unclear. Despite *S. stercoralis* being highly endemic in Asia, the role of animals as reservoir for zoonotic transmission has not yet been elucidated. We used nanopore sequencing to assess genetic variability of *Strongyloides* spp. infecting children, dogs, and long-tail macaques in Kampong Chhnang, Cambodia. We report shared *S. stercoralis* haplotypes in humans and dogs from the same villages and a high diversity of unique haplotypes in school-aged children, potentially suggesting a dominance of human-human transmission over zoonotic. Additionally, we provide the first report of *S. fuelleborni* infections in non-human primates in Cambodia and a high genetic diversity across geographical regions and primate hosts across Asia and Africa. These findings emphasise the usefulness of deep sequencing in determining species identity, shedding light on the transmission of *Strongyloides* species in humans and animals.

ID: 272 / CP23: 4

Contributed abstract

Conference Topics: One Health, Parasites of dogs, Zoonoses

Keywords: Lymphatic filariasis, dogs, one health, Brugia, zoonosis

Dogs as a reservoir host for a novel zoonotic *Brugia* in Sri Lanka

Ushani Atapattu¹, Lucas Huggins¹, Indeewarie Gunaratna², Vito Colella¹

¹Melbourne Veterinary School, University of Melbourne, VIC 3010, Australia; ²Anti-filariasis Campaign, Ministry of Health, Colombo 5, Sri Lanka

Lymphatic filariasis (LF) is a neglected tropical disease affecting ~120 million people globally, caused mainly by the parasitic filarial nematodes *Wuchereria bancrofti* and *Brugia malayi*. After several rounds of mass-drug administration, Sri Lanka was officially declared to have eliminated LF as a public health problem in 2016. However, *Brugia* infections were unexpectedly re-detected during human post-validation surveillance phase (PVSP) in the mid-2000s. These re-emergent parasites showed subperiodic periodicity unlike the nocturnally periodic strains previously observed, suggestive of zoonotic transmission. Subsequent surveys have identified a high incidence of *Brugia* microfilariae in dogs implicating them as potential reservoirs. We compared DNA sequences from human (n=12) and dog (n=16) samples that tested positive for *Brugia* microfilariae by microscopy, using partial *cox1* and *rRNA* gene regions through a new next-generation sequencing method. Phylogenetic and sequence type analyses demonstrated that *Brugia* sequences from dogs were identical to those from human infections in Sri Lanka and distinct from *B. malayi* strains in Southeast Asia. These findings provide molecular evidence that dogs are reservoirs of a novel zoonotic *Brugia* species infecting humans in Sri Lanka. This has critical implications for LF surveillance and control strategies during the PVSP in Sri Lanka.

ID: 287 / CP23: 5

Contributed abstract

Conference Topics: Parasites of dogs, Veterinary Parasitology, Zoonoses

Keywords: canine heartworm, dogs, zoonotic, dirofilaria, morphological, molecular, nematodes

A morphological and molecular framework for defining a new zoonotic species in the Asia-Pacific: *Dirofilaria asiatica* sp. nov.

Vito Colella, Neil D. Young, Ralph Manzanell, Ushani Atapattu, Sunita B. Sumanam, Lucas G. Huggins, Anson V. Koehler, Robin B. Gasser

Department of Veterinary Biosciences, Faculty of Science, The University of Melbourne, Parkville, VIC 3010, Australia

Parasitic nematodes of the family Onchocercidae (superfamily Filarioidea) have evolved alongside vertebrate hosts for millions of years. While morphological methods have played a central role in identifying species, many taxa remain cryptic and are challenging to differentiate, complicating diagnosis and disease control. Despite advances in our knowledge of the canine heartworm (*Dirofilaria immitis*), substantial gaps remain regarding other *Dirofilaria* spp., and their impact on both animal and human health. We conducted the first known comprehensive morphological and molecular characterisation of a novel *Dirofilaria* sp., *Dirofilaria asiatica* sp. nov., from dogs. This new species, genetically consistent with a previously identified genotype of *Dirofilaria*, initially found in humans in Hong Kong, was described using a combined morphological-molecular approach. The findings reveal that *D. asiatica* sp. nov. differs significantly from other known species, including *D. repens* and *D. immitis*, and might be responsible for the majority of zoonotic *Dirofilaria* infections in southern and southeastern Asia. This combined morphological and molecular approach establishes a framework for future investigations of filarioid nematodes, with implications for improving diagnosis and understanding of their epidemiology in both veterinary and medical contexts.

CP24: Fasciola 15 min talks

Time: Thursday, 03/July/2025: 3:30pm - 4:45pm · *Location:* Conference room 3

Session Chair: Tanapan Sukee, The University of Melbourne

Session Chair: Neil Young, The University Of Melbourne

ID: 166 / CP24: 1

Contributed abstract

Conference Topics: Drugs, Fasciolosis/Liver fluke, Helminthology, Livestock Parasites, Veterinary Parasitology

Keywords: Anthelmintic resistance, ELISA, Triclabendazole, Albendazole, livestock

Beyond Suspicion: Confirming the prevalence and occurrence of drug-resistant *Fasciola hepatica* in sheep, goats and cattle in the Southern Tablelands of NSW, Australia

Chelsie Uthayakumar¹, Hayley DeCristi¹, Emily Kate Francis¹, Roger Willoughby², Shannon Taylor³, Nichola Eliza Davies Calvani¹

¹Sydney School of Veterinary Science, The University of Sydney, Australia; ²Gunning Water & Ag Solutions, Gunning, Australia;

³The University of Sydney, Sydney, Australia

Fasciola hepatica (liver fluke) is the 13th most important cause of losses to the Australian sheep meat industry. First detected in Australia in 1995, resistance to the frontline drug, Triclabendazole (TBZ) is now present worldwide. In 2023, livestock producers from the NSW Southern Tablelands raised concerns over a 230% increase in liver fluke due to suspected drug resistance. To confirm or deny these suspicions as the cause of drug failure, we evaluated the prevalence and drug susceptibility of *F. hepatica* on eight farms. Nine mobs (seven sheep, one goat, one cattle) were split into three treatment groups of 15 animals/group and were administered either TBZ, AVOMEC DUEL/Albendazole (ABZ) (sheep/goats), or water. Treatments were administered according to individual animal weights, with faecal samples, animal weight, and body condition score collected on Day 0 and 21.

Our results showed evidence of TBZ resistance (89%-92% efficacy) on one sheep property. Interestingly, TBZ susceptibility (97%-98% efficacy) but ABZ resistance (77%-79% efficacy) was detected on the goat property, marking the first report of ABZ resistance in goats. Our results reinforce the increasing threat of drug resistance, highlighting the need for ongoing surveillance and development of alternative control options.

ID: 167 / CP24: 2

Contributed abstract

Conference Topics: Fasciolosis/Liver fluke, Livestock Parasites, Veterinary Parasitology

Keywords: Clustering, Diagnostics, Liver Fluke, Livestock, Surveillance

A contemporary map of *Fasciola hepatica* distribution in sheep and cattle in New South Wales

Simran N. Vyas¹, Janina McKay-Demeler², Michael Ward¹, Nichola Eliza Davies Calvani¹

¹Sydney School of Veterinary Science, The University of Sydney, Australia; ²Elizabeth Macarthur Agricultural Institute, NSW

Fasciola hepatica is a global threat to livestock production, human health, and food security. In New South Wales (NSW), Australia, data on the distribution of *F. hepatica* are more than 50 years out of date and lack species-specific insights for cattle and sheep. Accurate, up-to-date distribution data are essential for livestock producers to implement targeted control programs, for veterinarians to provide timely and effective treatment recommendations, and for researchers to identify emerging trends, such as those influenced by climate change. This study addresses this knowledge gap by using diagnostic samples submitted to the Elizabeth MacArthur Agricultural Institute (EMAI) between 2019-2023 to update the distribution of *F. hepatica* in cattle and sheep in NSW. Diagnostic records were extracted, cleaned, analysed and geospatially mapped at the postcode level to reveal temporal and spatial trends by livestock species. Our findings suggest that *F. hepatica* hotspots in sheep are concentrated in the southeastern regions of NSW, whereas in cattle, hotspots extend along the coast. These results reinforce the existing dogma of *F. hepatica* distribution in NSW, providing evidence-based insights that are key to improved surveillance, refining precision parasite management, and mitigating the ongoing impacts of *F. hepatica* on animal health and production in NSW.

ID: 194 / CP24: 3

Contributed abstract

Conference Topics: Fasciolosis/Liver fluke

Keywords: livestock producers, questionnaire, liver fluke, management practices, Australia

Survey on liver fluke (*Fasciola hepatica*) control practices used by Australian livestock farmers

Rana Muhammad Athar Ali¹, Neil David Young¹, Nichola Eliza Davies Calvani², Leah Tyrell³, Mark A. Stevenson¹, Grant Rawlin⁴, Travis Beddoe⁵, Terry Spithill⁵, Abdul Jabbar¹

¹Melbourne Veterinary School, The University of Melbourne, Werribee, Victoria 3030, Australia; ²Sydney School of Veterinary Science, The University of Sydney, NSW 2006, Australia; ³The Mackinnon Project, University of Melbourne, 250 Princes Hwy, Werribee, Victoria 3030, Australia; ⁴Department of Energy, Environment and Climate Action, Agriculture, Victoria, Bundoora, Victoria; ⁵Department of Ecological, Plant and Animal Sciences, La Trobe University, Bundoora, Victoria, Australia

This study assessed liver fluke control practices used by cattle, sheep, goat and alpaca producers in Australia. The online questionnaire prepared using REDCap comprised of 58 questions about farm demography, knowledge of liver fluke, diagnosis, treatment, anthelmintic resistance, and management and husbandry practices. Following a pilot study, the survey was distributed to all registered livestock producers through various animal industries on 15th February 2025. It was also shared on social media platforms, including Facebook and Twitter. Three hundred and twenty-seven farmers responded to the survey, resulting in 145 complete and 182 incomplete responses. Most responses were from cattle producers followed by sheep, goat and alpaca producers. Whilst participants were from all major livestock producing regions in Australia except Western Australia, the highest number of responses were received from New South Wales and Victoria. Descriptive statistics revealed that interval-based treatment, limited adoption of diagnostic tests, and infrequent rotation of flukicidal drugs are the main reasons for the continued prevalence and increased liver fluke resistance. This study provides insights into farmers' perceptions and management practices across various production systems, providing real-time perspectives to improve effective and strategic control of liver fluke in Australia.

ID: 135 / CP24: 4

Contributed abstract

Conference Topics: Epidemiology, Fasciolosis/Liver fluke, Helminthology, Host-parasite interactions, Invasive Species, Livestock Parasites

Keywords: Lymnaeid Snail, Quadrat Sampling, Seasonality, Invasive Species, Integrated Parasite Management

"First frost, last frost" Updating current knowledge on the seasonality of *Fasciola hepatica* in NSW's Southern Tablelands

Priscilla Huynh¹, Roger A. Willoughby², Neil D. Young³, Tanapan Sukee³, Emily K. Francis¹, Nichola E. D. Calvani¹

¹Sydney School of Veterinary Science, Faculty of Science, The University of Sydney, Camperdown, NSW, Australia; ²Gunning Ag & Water Solutions, Gunning, NSW, Australia; ³Melbourne Veterinary School, Faculty of Veterinary and Agricultural Sciences, The University of Melbourne, Vic, Australia

In Australia, liver fluke (*Fasciola hepatica*) is the 13th most important endemic disease in the sheep meat industry due to impacts on growth rate, body condition score and reproduction. *F. hepatica* larval (miracidia) development and infection of intermediate *Lymnaeid* snail hosts require average daily temperatures >10°C. In the NSW Southern Tablelands, climatic conditions between May to September are considered unsuitable for snail survival and parasite development. Producers leverage this seasonal break to guide chemical treatments, adhering to the dogma of 'first frost, last frost'. However, while conducting a drug resistance trial in July 2024, we observed numerous active liver fluke snails in this region when temperatures were <10°C.

To investigate the impacts of snail activity outside historical temperature ranges on liver fluke seasonality, we are conducting a longitudinal survey. Two spring-fed sites on six *F. hepatica*-infected sheep properties in the region will be quadrat sampled for snails bi-monthly. Collected snails will be speciated using morphological and molecular methods to assess abundance and diversity throughout the year, in correlation with local climate data. To preserve the remaining efficacy of limited chemical controls, the preliminary findings will help redefine current risk periods, which are crucial to informing sustainable, integrated parasite management strategies.

ID: 209 / CP24: 5

Contributed abstract

Conference Topics: Fasciolosis/Liver fluke

Keywords: Fasciola, behaviour, host-parasite interaction

The effect of intermediate host species on the host-finding and host-attachment abilities of *Fasciola hepatica* miracidia.

Natasha Sharma¹, Tanapan Sukee¹, Scott Cummins², Bonnie Webster³, Winston Ponder⁴, Anson V Koehler¹, Neil D Young¹

¹The University of Melbourne, Australia; ²The University of Sunshine Coast; ³Natural History Museum London; ⁴Australian Museum

Fasciola hepatica, the causative agent of fascioliasis in Australian sheep and cattle, requires a permissive molluscan host to complete its lifecycle. The larval stage of this parasite is well-adapted to host-finding, with chemotactic abilities that can sense potential host biomolecules. This study investigated whether *F. hepatica* miracidia have host-specificity in the pre-attachment phase, and the ability of miracidia to attach and penetrate both permissive and non-permissive and native and non-native hosts was also examined. This was achieved through a quantitative and qualitative comparison of miracidial behavioural change post-exposure to snail-conditioned water (SCW) of four key snail species, both known permissive and non-permissive hosts in the Australian landscape. *F. hepatica* miracidia were also exposed to live snails of the representative species to ascertain whether host permissiveness correlates with miracidial host-attachment ability. *F. hepatica* miracidia showed clear shifts in kinetic profiles post-exposure to SCW, with no differences observed between snail species. When exposed to live snails, *F. hepatica* miracidia had higher attachment success to both permissive and non-permissive native snails compared to invasive snails, and higher success in invasive permissive hosts over invasive non-permissive hosts. These results provide an important foundation for further exploration into intermediate host-parasite interactions within an Australian context.

CP22.1: Omics 5 min talks

Time: Thursday, 03/July/2025: 4:30pm - 5:00pm · Location: Conference room 1

Session Chair: Shilpa Kapoor, The University of Melbourne

Session Chair: Aaron Jex, WEHI

ID: 275 / CP22.1: 1

Contributed abstract

Conference Topics: Bioinformatics, Genomics, Malaria

Keywords: RNA modifications, Epitranscriptomics, m⁶A, Nanopore, RNA-seq

Detecting and characterising base modifications in *Plasmodium falciparum* with Nanopore direct RNA sequencing

Joshua Levendis, Lakvin Fernando, Amy Distiller, Emma McHugh, Stuart Ralph

Department of Biochemistry and Pharmacology, The University of Melbourne, Parkville, Victoria 3052, Australia

Harnessing RNA modifications in therapeutics has enabled breakthroughs in vaccines. Despite their importance in RNA stability and translation, the mechanisms of RNA modifications are poorly understood. N⁶-methyladenosine (m⁶A) is the most abundant RNA modification in eukaryotes and changes translational efficiency or reduces mRNA stability. We studied this modification in *Plasmodium falciparum* by disrupting the methyltransferase that makes m⁶A with a knock-sideways mislocalisation system. Recent developments in sequencing by Oxford Nanopore Technologies (ONT) have enabled the direct detection of m⁶A in RNA. We therefore disrupted the methyltransferase and used Nanopore direct RNA-sequencing to study differential methylation at multiple points during the *P. falciparum* lifecycle. We were able to detect differentially methylated transcripts after mislocalising the methyltransferase, confirming the utility of both the knock-sideways system and Nanopore RNA-sequencing in studying m⁶A in *P. falciparum*. We detected differentially expressed genes after disrupting the methyltransferase, and found removing m⁶A affected the 3' end position of transcripts. Our work shows Nanopore RNA-seq can be used to detect m⁶A abundance and location in *P. falciparum*, proving it a valuable technique for studying the impact of RNA modifications on parasite biology.

ID: 211 / CP22.1: 2

Contributed abstract

Conference Topics: Drugs, Malaria, Proteomics

Keywords: antimalarial, mechanism of action, drug target identification, TPP, CETSA, Lip-MS

"Too Hot to Handle: Stability Proteomics Reveals Antimalarial Drug Targets"

Jerzy Dziekan¹, Sachin Khurana¹, Arne Alder², Sofia Mortensen³, Holger Sondermann³, Tim Gilberger², Alan Cowman¹

¹The Walter and Eliza Hall Institute of Medical Research, Parkville, VIC, Australia; ²Bernhard Nocht Institute for Tropical Medicine, Hamburg, Germany; ³Center for Structural Systems Biology, Hamburg, Germany

Identifying drug targets for novel small-molecule therapeutics discovered through phenotypic screening remains a major challenge in antimalarial drug development. *Stability Proteomics* methodologies—untargeted and unbiased approaches for identifying drug-binding proteins in an organism—offer a powerful solution to this knowledge gap. These methods exploit the fundamental principle that ligand binding stabilizes proteins, reducing their susceptibility to denaturation. By leveraging this concept, several orthogonal techniques have been introduced, which interrogate the proteome and detect drug-induced change in stability based on target protein's altered susceptibility to denaturation by temperature, organic solvents, or proteolytic cleavage.

To advance antimalarial drug discovery, we developed next-generation experimental and analytical workflows for three complementary and orthogonal *Stability Proteomics* approaches: Thermal Proteome Integral Solubility Alteration Assay (Thermal PISA), Solvent PISA, and Limited Proteolysis Mass Spectrometry (Lip-MS). We applied these methods to characterize target engagement and downstream mechanism-of-action (MoA) effectors for a diverse library of antimalarial compounds, including clinically approved drugs, tool compounds, and leading candidates in clinical trials.

Our findings demonstrate the universality, sensitivity, and specificity of these methodologies in identifying drug-target interactions, and highlighting *Stability Proteomics* as a transformative approach for accelerating target deconvolution in drug discovery, with potential applications extending well beyond antimalarials.

ID: 239 / CP22.1: 3

Contributed abstract

Conference Topics: Biochemistry, Drugs, Genomics, Molecular Biology, Protozoa

Keywords: Neglected tropical diseases, *Trypanosoma brucei*, DNA secondary structures, drug targets, genomics.

Genomics and Drug Discovery: Advancing the Fight Against Neglected Tropical Diseases

Ludovica Monti^{1,2}, Joana R. Correia Faria³, John M. Kelly⁴, Genta Firth¹, Lingxiao Long¹, Gem Flint^{2,5}, Silvia Galli², Thomas E. Maher^{2,5}, Marco Di Antonio^{2,5,6}

¹School of Chemistry and the Bio21 Molecular Science and Biotechnology Institute, The University of Melbourne, Parkville, Victoria, Australia; ²Department of Chemistry, Molecular Sciences Research Hub, Imperial College London, London, UK; ³Department of Biology, University of York, York, UK; ⁴London School of Hygiene and Tropical Medicine, London, UK; ⁵Institute of Chemical Biology, Molecular Sciences Research Hub, Imperial College London, London, UK; ⁶The Francis Crick Institute, London, UK.

Parasitic pathogens responsible for neglected tropical diseases continue to disproportionately affect some of the world's most vulnerable populations facing economic challenges, and contributing significantly to both mortality and long-term morbidity. Despite decades of research, available treatments remain limited, and are often associated with severe adverse effects. Moreover, the risk of possible emergence of drug resistance remains a concern. There is, therefore, the need to develop new alternative strategies for therapeutic intervention.

In this presentation, we will focus on human African trypanosomiasis, a devastating parasitic disease caused by *Trypanosoma brucei*, and explore the use of high-throughput sequencing technologies and genomics to identify and characterise novel

druggable targets. A central aspect of this work involves investigating the role of unique DNA secondary structures, known as G-quadruplexes, in parasite biology and survival. These structures represent an emerging class of targets with potential for therapeutic exploitation. The presentation will explore how genomic-based methodologies can be integrated into drug discovery pipelines to develop next-generation therapeutics for parasitic diseases.

ID: 158 / CP22.1: 4

Contributed abstract

Conference Topics: Biochemistry, Bioinformatics, Malaria

Keywords: Metabolomics, SIL, Plasmodium

Metabolic Tracing in *P. falciparum* Using a Stable Isotope Labelling Strategy

Junwei Tang, Darren Creek, Christopher MacRaid

Monash Institute of Pharmaceutical Sciences, Monash University, Parkville, Vic, Australia

Plasmodium falciparum, the causative agent of malaria, undergoes complex metabolic transformations across its life cycle stages, presenting opportunities for targeted therapeutic interventions. This study employs stable isotope labeling (SIL) with ¹³C₆-glucose to investigate metabolic fluxes in infected red blood cells (iRBCs) under different developmental conditions. High-resolution mass spectrometry and bioinformatic analysis enable the identification of both native and labeled metabolites, offering insights into metabolic pathway activities and potential novel metabolite discovery. The approach captures dynamic metabolic changes across various stages, contributing to a comprehensive understanding of *Plasmodium* metabolism and supporting efforts to develop novel antimalarial strategies.

ID: 254 / CP22.1: 5

Contributed abstract

Conference Topics: Apicomplexa Biology, Cell Biology, Malaria, Molecular Biology, Proteomics

Keywords: Plasmodium, translation regulations

Understanding the translational repression landscape in gametocytogenesis.

Niva Jayakrishnan^{1,2}, Balu Balan², Katrina Larcher^{1,2}, Sash Lopaticki^{1,2}, Aaron Jex², James McCarthy^{1,2}, Danushka. S. Marapana², Matthew W.A. Dixon^{1,2}

¹Department of Infectious Diseases, The Peter Doherty Institute, University of Melbourne, Victoria.; ²Infection and Global Health Division, Walter and Eliza Hall Institute, Victoria.

The malaria-causing parasite *Plasmodium falciparum* has a complex lifecycle transitioning through diverse cellular niches within the human host and the Anopheles mosquito vector. Transmission of the parasite from the human to the mosquito requires the dramatic differentiation (cell fate change) from an asexually dividing short-lived cell (48 hrs) to a long-lived (12 days) sexual stage gametocyte capable of forming gametes in the mosquito midgut. While gametocyte development is a significant lifecycle bottleneck that could be exploited in transmission blocking interventions, a fundamental understanding of the cell fate decisions driving the development of gametocytes is unknown. *Plasmodium* parasites lack sex chromosomes and have a reduced repertoire of transcription factors, making them heavily reliant on post-transcriptional mechanisms for gene regulation. One such mechanism is translation repression, a process where RNA-binding proteins (RBPs) regulate stage-specific gene expression, through the selective binding of transcripts stopping them from being translated. The stage specific release of these held transcripts allows for rapid protein translation and stage switching. In this work we will use a combined genetic approach targeting both canonical and novel RBPs, with transcriptomics and proteomics analysis to define the landscape of translational repression driving gametocytogenesis.

ID: 263 / CP22.1: 6

Contributed abstract

Conference Topics: Apicomplexa Biology, Malaria, Protozoa, Proteomics

Keywords: Malaria, mRNA Base Modifications, m6A, Proteomics

N⁶-methyladenosine-dependent changes in the Proteome of *Plasmodium falciparum* during Asexual Replication

Lakvin Fernando^{1,2}, Amy Distiller^{1,2}, Joshua Levendis^{1,2}, Emma McHugh^{1,2}, Stuart Ralph^{1,2}

¹Department of Biochemistry and Pharmacology, University of Melbourne; ²Bio21 Molecular Science & Biotechnology Institute

The rapid, synchronous asexual reproduction of *Plasmodium falciparum* during the intraerythrocytic developmental cycle (IDC) is a key determinant of pathogenesis. *P. falciparum* uses many mechanisms to regulate gene expression during the IDC including mRNA base modifications such as N⁶-methyladenosine (m6A). In other eukaryotes m6A can regulate pre-mRNA maturation, mRNA export, cytosolic stability and translational efficiency.

The methylation of adenosine to m6A in *Plasmodium falciparum* is catalysed by the protein complex known as the m6A writer. Three core proteins that form this complex are conserved in eukaryotic organisms. The methyltransferase (*PfMT-A70*) is related to the human METTL3 and is the enzymatic component of the m6A writer.

In this study, we use knock sideways to anchor *PfMT-A70* to the parasite membrane, away from its usual site of action, the nucleus. Using direct RNA sequencing we have demonstrated that knock sideways reduces the amount of m6A on typically methylated mRNA adenosines. Any m6A-mediated changes in mRNA function may affect the abundance of the protein that each mRNA encodes. Therefore, we aimed to study the proteome of *P. falciparum* following *PfMT-A70* knock sideways.

CP23.1: One Health 5 min talks

Time: Thursday, 03/July/2025: 4:45pm - 5:00pm · Location: Conference room 2

Session Chair: Breanna Knight, Murdoch University

Session Chair: Harsha Sheorey, St Vincent's Hospital, Melbourne

ID: 193 / CP23.1: 1

Contributed abstract

Conference Topics: Education/Outreach, Epidemiology, One Health, Parasites of cats, Parasites of companion animals, Parasites of dogs, Strongyloides, Veterinary Parasitology, Zoonoses

Keywords: Strongyloides stercoralis, mass drug administration, one health elimination, community participation

Companion Animals in Strongyloides Elimination: A Promising Solution with Practical Challenges

Bonny Cumming¹, Jessica Hoopes¹, Tamara Riley², Kirstin Ross³, Michael Spry⁴

¹Animal Management in Rural and Remote Indigenous Communities Ltd (AMRRIC), Larrakia Country, NT, Australia; ²Yardhura Walani, National Centre for Epidemiology and Population Health, The Australian National University, Canberra, Ngunnawal Country, ACT, Australia; ³College of Science and Engineering, Flinders University, Adelaide, Kurna Country, SA, Australia; ⁴Environmental Health Strategy, Public Health Division, Northern Territory Government, Larrakia Country, NT, Australia

Strongyloidiasis is a neglected tropical disease causing chronic illness and potentially fatal infection, particularly in immunocompromised individuals. *Strongyloides stercoralis* is prevalent among marginalised populations in tropical and subtropical areas, with the highest rates in rural and remote northern Australia. Mass drug administration (MDA) is seen as a promising strategy for reducing infection rates and preventing long-term health impacts in these communities.

Recent findings of a shared *S. stercoralis* haplotype in humans, dogs, and cats support a One Health approach that includes treating companion animals as part of strongyloidiasis control efforts. While this integrated approach may improve both human and animal health, its implementation in remote Indigenous communities presents practical challenges.

Developing effective animal-targeted MDA programs requires meaningful collaboration that reflects community priorities and draws on local knowledge. Success depends on factors such as limited evidence on the efficacy of available (often off-label) treatments, and difficulties in monitoring parasite shedding among free-roaming dogs and cats. Environmental risks, including drug resistance and contamination, also require careful management.

To ensure success, MDA programs must involve genuine community participation and respect the cultural significance of companion animals in Aboriginal and Torres Strait Islander communities, promoting sustainable, ethical, and culturally appropriate health strategies.

ID: 213 / CP23.1: 2

Contributed abstract

Conference Topics: Parasites of cats, Parasites of companion animals, Veterinary Parasitology, Wildlife parasitology, Zoonoses

Keywords: Toxocara, Toxoplasma, Feral Cat, Zoonosis

Zoonotic parasites in the Empire of the Feral Cat

Ryan O'Handley, Kaitlyn Alder, Lily Martin, Charles Caraguel

Adelaide University, Australia

The population density of feral cats on Kangaroo Island, South Australia is 10 times that of the mainland. The impact of feral cats on native wildlife is well established, but their impact as reservoirs for zoonotic parasites is not fully understood. Since 2022, blood and faecal samples have been collected from more than 300 feral cats on KI that were euthanised as part of the cat eradication program. We examined faecal samples from a subset of 70 cats for gastrointestinal parasites and used the MAT to test blood samples for the presence of *Toxoplasma gondii* antibodies. Antibodies to *T. gondii* were demonstrated in 72.1% of the cats, and patent *Toxocara cati* infections were confirmed in 87% of cats. *Ancylostoma tubaeformae* eggs were found in 36% of cats while 11% of cats were excreting *Spirometra* spp eggs in their faeces. Interestingly, *T. gondii* oocysts were not identified in the faeces of any cats, suggesting cats are first exposed to the parasite at a very young age. The results indicate feral cats on KI pose a major risk to human and environmental health due to the high prevalence of zoonotic parasites. The high prevalence of *Toxocara cati* is of particular importance.

ID: 296 / CP23.1: 3

Contributed abstract

Conference Topics: Helminthology, One Health, Zoonoses

Keywords: Soil-transmitted helminths, Vanuatu

Genetic diversity and transmission of soil-transmitted helminths in humans and dogs from Vanuatu

Molly Waldron¹, Lucas Huggins¹, Patsy Zendejas-Heredia¹, Sze Fui Hii¹, Ian Douglas², Clare Anstead¹, Prudence Rymill³, Fasihah Taleo⁴, Susana Vaz Nery³, Vito Colella¹

¹Faculty of Science, Veterinary Preclinical Sciences Building, University of Melbourne, Parkville, Victoria 3052, Australia; ²Vets Beyond Borders, Molong, New South Wales 2866, Australia; ³Ministry of Health, Port Vila, Shefa, Vanuatu; ⁴World Health Organisation, Port Vila, Shefa, Vanuatu

Soil-transmitted helminths (STHs) are a group of parasitic nematodes responsible for chronic infections in both humans and animals, causing some of the most important and prevalent neglected tropical diseases worldwide. Current efforts to control STHs in humans are based on treatment of at-risk populations with benzimidazoles. Multiple factors complicate STH control, including inaccurate diagnostic tools to assess the effectiveness of interventions. In addition, in the Asia Pacific, animals may be reservoirs of zoonotic STHs for humans, further hindering control efforts. Despite this, our understanding of STH diversity across hosts and the extent of animal contributions to human infections remains limited, largely due to the scarcity of studies using robust molecular methods. This research project will explore the genetic diversity of nematodes, including STHs, in dogs and humans from Vanuatu using a recently developed long-read NGS (MinION) assay. Further, we will perform whole mitochondrial genome sequencing of *Strongyloides* spp. and *Ancylostoma* spp. isolates from humans and dogs. Both species have been indicated as potentially zoonotic in low- and middle-income countries.

This study will provide fundamental data on potential inter-species transmission of zoonotic STHs in Vanuatu, informing the need for novel strategies for STH control.

CP24.1: Fasciola 5 min talks

Time: Thursday, 03/July/2025: 4:45pm - 5:00pm · *Location:* Conference room 3

Session Chair: Tanapan Sukee, The University of Melbourne

Session Chair: Neil Young, The University Of Melbourne

ID: 285 / CP24.1: 1

Contributed abstract

Conference Topics: Fasciolosis/Liver fluke

Keywords: snail vectors, *Fasciola hepatica*, mitochondrial genome, rRNA

Molecular tools for assessing the genetic diversity of introduced freshwater snails as trematode vectors in Australia

Tanapan Sukee¹, Anson V. Koehler¹, Jane Hodgkinson², Neil D. Young¹

¹Melbourne Veterinary School, Faculty of Sciences, The University of Melbourne, Parkville, Victoria, Australia; ²Institute of Infection, Veterinary & Ecological Sciences, Faculty of Health and Life Sciences, The University of Liverpool, Leahurst, Liverpool, The United Kingdom

Freshwater snails act as intermediate hosts for trematode parasites that affect humans, livestock, and wildlife. In Australia, *Fasciola hepatica* significantly impacts the livestock industry, with transmission primarily involving native snail species. Although, introduced lymnaeid snails such as *Pseudosuccinea columella* and *Orientogalba viridis* may expand the geographic range and transmission dynamics of fasciolosis. However, the genetic identity and diversity of these introduced snails remain uncharacterised in Australia.

This study uses Oxford Nanopore long-read sequencing to generate complete mitochondrial genomes and full nuclear ribosomal RNA genes from introduced freshwater snails. By combining mitochondrial and ribosomal data, we aim to address the lack of reference sequences of key vectors of trematode parasites and improve phylogenetic resolution.

These genetic references will enable accurate identification of potential trematode vectors and help clarify their evolutionary relationships with known host species globally. The data generated will also support the development of diagnostic tools and strengthen biosecurity efforts. Ultimately, this work contributes to understanding and managing the spread of invasive snails and the trematode parasites they may carry.

ID: 257 / CP24.1: 2

Contributed abstract

Conference Topics: Fasciolosis/Liver fluke

Keywords: *Fasciola hepatica*, Anthelmintic Resistance, Triclabendazole, Genotype, Phenotype

Exploring anthelmintic resistance in *Fasciola hepatica* in endemic regions of Australia

Mengwei Zheng¹, Aya C. Taki¹, Tanapan Sukee¹, Jane Hodgkinson², Robin B. Gasser¹, Neil D. Young¹

¹Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Veterinary and Agricultural Sciences, The University of Melbourne, Parkville, VIC 3010, Australia; ²Department of Infection and Microbiome, Institute of Infection, Veterinary and Ecological Sciences, Leahurst Campus, University of Liverpool, Neston, CH64 7TE, UK

Fasciolosis, a disease caused by the liver fluke *Fasciola hepatica*, is a common endoparasitic disease affecting ruminants worldwide. Current control strategies rely heavily on the anthelmintic triclabendazole (TBZ), which is active against both immature and adult stages of this parasite. However, the emergence of TBZ resistance since 1995 has emphasised the need to investigate the characteristics of TBZ resistant and susceptible populations of *F. hepatica*. In this study, we will use a range of genotypic and phenotypic methods to comprehensively characterize such *F. hepatica* populations in Australia. First, double digestion restriction site-associated DNA sequencing (ddRADseq) will be used to explore the parasite's population genetic structure in key areas of Australia. Then, whole genome sequencing (WGS) will study isolates of *F. hepatica* exhibiting varying levels of susceptibility to TBZ in infected animals from distinct geographical locations and define/identify genetic markers that associate with resistance. Subsequently, TBZ resistance/susceptibility will be assessed in vitro using a novel phenotypic (whole parasite) assay.

As this project progresses, we expect to obtain comprehensive genotypic and phenotypic profiles for various *F. hepatica* populations, to understand TBZ resistance in this country and, using these profiles, guide future control strategies.

ID: 157 / CP24.1: 3

Contributed abstract

Conference Topics: Ecology, Epidemiology, Fasciolosis/Liver fluke, Host-parasite interactions, Livestock Parasites, Veterinary Parasitology, Wildlife parasitology

Keywords: *Fasciola hepatica*, kangaroos, wallabies, non-chemical control, integrated parasite management

Fluke fate: Unravelling the role of macropods in liver fluke transmission on shared pastures

Chloe Burden, Nichola Eliza Davies Calvani

Sydney School of Veterinary Science, The University of Sydney, Australia

Fasciola hepatica contributes an estimated \$25 million in production losses to Australia's sheep meat industry. The parasite's indirect lifecycle and broad mammalian host specificity enables persistent infections on farms via infection of accidental wildlife hosts in regions endemic to Lymnaeid snails. Traditionally, control of *F. hepatica* in livestock relies on a narrow repertoire of anthelmintic drugs, now facing reduced efficacy due to evolved resistance. As chemical control becomes increasingly challenging, sustainable evidence-based, non-chemical strategies focused on reducing pasture contamination and infection rates in both livestock and wildlife hosts are of renewed importance in shared grazing environments.

During a parasitology information session hosted by Sydney University in 2023, graziers in the NSW Southern Tablelands raised concerns on the value of exclusion fencing to mitigate livestock infections in regions co-habited by large numbers of Eastern grey kangaroos, red-necked and swamp wallabies. Only four studies have been conducted on *F. hepatica* infection in macropods to date, none of which examined their relative capacity to contaminate pasture. Using animals culled during routine pest management, we aim to evaluate the true contribution of Eastern grey kangaroos and wallabies to pasture contamination to provide evidence for the use of exclusion fencing in high-risk areas.

2025 Annual Conference of the Australian Society for Parasitology Inc.

30 June – 3 July, 2025 Melbourne Connect, Melbourne, Vic, Australia

Delegates

Name	Organisation
Zainab Umar Abdullahi	University of Melbourne
Anju Abraham	Walter and Eliza Hall Institute
Liisa Ahlstrom	Elanco
Elizabeth Aitken	Peter Doherty Institute, University of Melbourne
Rana Muhammad Athar Ali	University of Melbourne, Australia
Endris Ali	University of Melbourne
Wafa Almegrin	Princess Nourah bint Abdulrahman University
Kathy Andrews	Griffith University
Fiona Angrisano	Burnet Institute
Clare Anstead	University of Melbourne
Anjaleena Anthony	Promega Corporation
Dionne Argyropoulos	Walter and Eliza Hall Institute
Leonhard Satrio Arinanto	University of Melbourne
Amanda Ash	Murdoch University
Ushani Atapattu	University of Melbourne
Peter Atkinson	University of Adelaide
Kyle Awalt	Walter & Eliza Hall Institute of Medical Research
Balu Balan	Walter and Eliza Hall Institute
Paolo Bareng	Kirby Institute, UNSW
Xavier Barton	Murdoch University
Di Barton	Charles Sturt University
Jake Baum	UNSW Sydney
James Beeson	Burnet Institute
Jhobert Bernal	WEHI
Ian Beveridge	University of Melbourne
Elouise Bliss	Federation University
Felicia Bongiovanni	The Walter and Eliza Hall Institute of Medical Research
Nathan Bott	RMIT University
Vern Bowles	The University of Melbourne
Kabir Brar	University of Melbourne
Keira Brown	Charles Sturt University
Diana Bulla Castaneda	RMIT
Hayley Bullen	Burnet Institute
Sugandika Bulumulla	Murdoch University

Chloe Burden	The University of Sydney
Connor Bury	The University of Adelaide
Nichola Calvani	The University of Sydney
Melissa Carabott	RMIT University
Li Jin Chan	The Walter and Eliza Hall Institute
Rainbow Chan	WEHI
Jeff Chan	Southern Cross Diagnostics
Jill Chmielewski	WEHI
Mrittika Chowdury	Deakin University
Vito Colella	The University of Melbourne
Sophie Collier	The University of Melbourne
Brian M Cooke	James Cook University
Haylee Crawford-Weaver	DCCEEW
Darren Creek	Monash University
Mikayla Crouch	University of Queensland
Bonny Cumming	AMRRIC
Brenna Daily	Institute for molecular Biosciences UQ
Madeline Dans	Walter and Eliza Hall Institute
Karen Day	University of Melbourne
Tania de Koning-Ward	Deakin University
Tilini De Silva	The University of Melbourne
Haowen Deng	University of Melbourne
Matthew Dixon	University of Melbourne
Denise Doolan	University of Queensland
Michael Duffy	University of Melbourne
Vincent Duru	University of Melbourne
Narelle Dybing	Murdoch University
Jerzy Dziekan	The Walter and Eliza Hall Institute of Medical Research
Jackson Elms	Walter and Eliza Hall Institute of Medical Research
David Emery	University of Sydney
Samantha Emery-Corbin	Monash University
Rebecca Farnell	Federation University Australia
Sarah Farrell	University of Melbourne
Paola Favuzza	Walter and Eliza Hall Institute of Medical Research
Sindew Mekasha Feleke	La Trobe University
Lakvin Fernando	University of Melbourne
Joana Ferreira Costa	University of Melbourne
Joanne Ford	Thermo Fisher Scientific
Freya Fowkes	University of Melbourne
Emily Francis	The University of Sydney
Robin Gasser	UniMelb
Charles Gauci	University of Melbourne
Teha Shumbej Gebi	Monash University

Carlo Giannangelo	Monash University
Gemma Godsell	Abacus Dx
Christopher Goodman	University of Melbourne
Warwick Grant	La Trobe University
Darren Gray	QIMR Berghofer
Michaela Grima	Deakin University
Luke Hall	University of Technology Sydney
Alexander Harris	Burnet Institute
Shannon Hedtke	La Trobe University
Ashleigh Heng-Chin	Burnet Institute
Sasha Herbert	The University of Melbourne
Sze Fui Hii	The University of Melbourne
Chee Ho H'ng	VectorBuilder
Jessica Home	University of Melbourne
Jemma Hudson	RMIT
Lucas Huggins	University Of Melbourne
Kate Hutson	Cawthron Institute
Priscilla Huynh	University of Sydney
Long Huynh	The University of Melbourne
Alessia Hysa	Burnet Institute / The University of Melbourne
Abdul Jabbar	University of Melbourne
Niva Jayakrishnan	WEHI/UoM
Natalie Jefferson	Charles Sturt University
Aaron Jex	WEHI
Lisa Jones	ASP
Bernd Kalinna	Charles Sturt University
Shilpa Kapoor	The University of Melbourne
Janani Karunaratne	University of Melbourne
Ashton Kelly	University of Queensland
Olivia Kelly	University of Sydney
Nutpakal Ketprasit	Bio21 Institute, The University of Melbourne
Sachin Khurana	The Walter and Eliza Hall Institute
Tanya King	Federation University
Breanna Knight	Murdoch University
Anson Koehler	University of Melbourne
Vivin Kokuhenadige	University of Melbourne Peter Doherty Institute
Andrew Kotze	University of Queensland
Vinoth Kumarasamy	National University of Malaysia
Liriye (Lydia) Kurtovic	Burnet Institute
Chanelle (Keng Heng) Lai	The University of Adelaide
Alex Lam	WEHI
Elena Lantero Escolar	The Walter and Eliza Hall Institute of Medical Research
Katrina Larcher	The University of Melbourne
Andrew Larkins	Murdoch University

Jennifer Le	Monash University
Adele Lehane	Australian National University
Rachel Leonard	Australian National University
Zhaochun Li	Monash University
Ruijia Liang	Australian National University
Marshall Lightowlers	University of Melbourne
Pailene Lim	WEHI
Qingqing Lin	WEHI
Elysia Ling	The University of Melbourne
Dawson Ling	Walter and Eliza Hall Institute of Medical Research
Alysha Literski	Burnet Institute
Tharaka Liyanage	The University of Melbourne
Rhea Longley	WEHI
Alex Loukas	James Cook University
Kelly Ly	Abacus dx
Alan Lymbery	Murdoch University
Frankie Lyons	WEHI
Jacinta Macdonald	Griffith University
Quinn Mackie	University of Adelaide
Chris MacRaid	Monash Institute of Pharmaceutical Sciences
Sonakshi Madan	Deakin University
Mahta Mansouri	Walter and Eliza Hall Institute
Danushka Marapana	WEHI
Storm Martin	Murdoch University
Ramin Mazhari	WEHI
Kirsty Mccann	Deakin University
Malcolm McConville	University of Melbourne
Geoff McFadden	University of Melbourne
Connor McHugh	James Cook University
Ludovica Monti	The University of Melbourne
Elizabeth Mullens	Federation University Ballarat
Bahar E Mustafa	The University of Melbourne
Maddie Nam	The University of Melbourne
Bernard Nathaniel	Deakin University
Myo Naung	Burnet Institute
Anne Nguyen	University of New South Wales
William Nguyen	WEHI
Hanh Nguyen	Zip Diagnostics
Khoi Nguyen	Burnet Institute
Suji O'Connor	The Australian National University
Katherine O'Flaherty	The University of Melbourne
Ryan O'Handley	Adelaide University
Akachukwu Onwuka	University of Melbourne

Zi Kang Ooi	Walter and Eliza Hall Institute of Medical Research
Millicent Opoku	La Trobe University
Charlotte Oskam	Murdoch University
Unaiza Parkar	Thermo Fisher Scientific
Nisha Paudel	Deakin University
Gilson Paul	Burnet Institute
Matthew Paxman	ZiP Diagnostics
Ashleigh Peck	Murdoch University
Kaitlin Pekin	Burnet Institute
Michelle Power	Macquarie University
Cecilia Power	RMIT University
Rosemary Presburger	University of Otago
Sarah Preston	Federation University Australia
Chunyi (Bessie) Qian	Walter and Eliza Hall Institute of Medical Research
Nancy Quashie	Noguchi Memorial Institute for Medical Research, University of Ghana
Zaynab Radih	Australian National University
Stuart Ralph	The university of Melbourne
Grace Reeves	University of Queensland
Nic Ristevski	Walter and Eliza Hall Institute for Medical Research
Florian Roeber	Invetus Pty Ltd
Ornella Romeo	The University of Adelaide
Praidp Roy	Universty of Melbourne
Dayna Sais	University of Technology Sydney
Kevin Saliba	Australian National University
Harvey Santos	The University of Queensland
Eti Sarkar	James Cook University
Claire Sayers	University of New South Wales
Stephen Scally	WEHI
Johann Schröder	Gemini R&D Services
Dale Seaton	Elsevier
Fleur Sernee	The University of Melbourne
Shokoofeh Shamsi	Charles Sturt University
Natasha Sharma	The University of Melbourne
Harsha Sheorey	St Vincent's Hospital, Melbourne
Jiaqi Shi	Burnet Institue
Mohini A Shibu	The University of Melbourne
Ghizal Siddiqui	Monash Univeristy
Hannah Siddle	University of Queensland
Suchandan Sikder	James Cook University
Amber Simonpietri	Walter and Eliza Hall Institute
Karan Singh	The Walter and Eliza Hall Institute of Medical Research

Shamit Singla	The University of Adelaide
Neha Sirwani	La Trobe University
Jan Slapeta	University of Sydney
Joshua Slattery	Charles Sturt University
Brad Sleebs	Walter and Eliza Hall Institute
Simone Sleep	Pathocell Research and Diagnostics
Nick Smith	ASP
Emily Smith	The University of Queensland
Edith Spiers	University of Melbourne
Terry Spithill	La Trobe University
Katelyn Stanhope	Burnet Institute
Senna Steen	Burnet Institute
Don Strazzeri	Vetoquinol Australia Pty Ltd
Tanapan Sukee	The University of Melbourne
Colin Sutherland	LSHTM London
Kahlia Szabo	Deakin University
Ala Tabor	UQ
Mun Hua Tan	The University of Melbourne
Junwei Tang	Monash University
Sara Taylor	QIMR Berghofer
Wai-Hong Tham	Walter and Eliza Hall Institute
Leann Tilley	University of Melbourne
Jarryd Tiu	The University of Adelaide
Chris Tonkin	The Walter and Eliza Hall Institute
Nham Tran	University of Technology Sydney
Katharine Trenholme	QIMRB
Cristian Trisca	RMIT University
Patrick Tumwebaze	Griffith University, Nathan Campus, GRIDD
Alexander Tynan	Rural Health Research Institute
Alex Uboldi	The Walter and Eliza Hall Institute of Medical Research
Giel van Dooren	Australian National University
Amrita Vijay	WEHI
Molly Waldron	The University of Melbourne
Deonne Walther	James Cook University
Susie Wang	WEHI
Sugandhika Welikadage	University of Melbourne
Amanda Wernicke	Southern Cross Diagnostics Pty Ltd
Maree Widdicombe	RMIT University
Mary-Louise Wilde	University of Melbourne
Danny Wilson	The University of Adelaide
Mackrina Winslow	The University of Melbourne, at the Peter Doherty Institute for Infection and Immunity
Grace Wright	Burnet Institute
Xuexin Xia	University of Melbourne

Xiao Xiao	The Walter and Eliza Hall Insitute of Medical Research
Jianuo Xu	University of Melbourne
Lee Yeoh	Burnet Institute
Rachael Yong	Deakin University
Neil Young	The University Of Melbourne
Meghan Zadow	University of Adelaide
Patsy Zendejas-Heredia	The University of Melbourne
Zhetao Zhang	The Queensland Alliance for Agriculture and Food Innovation (QAAFI)
Jingjing Zhang	University of Melbourne
Leqian Zhao	Australian National University
Yuanting Zheng	the University of Melbourne
Mengwei Zheng	The University of Melbourne
Minghui Zhu	University of Melbourne

2025 Annual Conference of the Australian Society for Parasitology Inc.

30 June – 3 July, 2025 Melbourne Connect, Melbourne, Vic, Australia

Author List

Author Name	Paper IDs	Conference Sessions
Abbas, Ghazanfar	242	CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3)
Abdullah, Swaid	155, 144	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Abeysekera, Waruni	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Abraham, Anju	241, 133, 230, 233	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Absalon, Sabrina	255	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Abullah, Swaid	181	CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2)
Adair, Amy	128, 154, 133	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Agius, Paul	174	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Ahmady, Farah	117	CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Aitken, Elizabeth	204, 258	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Alawad, Ghali	104	CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3)
Alder, Arne	211	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Alder, Kaitlyn	213	CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Alemu, Agersew	258	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Ali, Endris	242, 235	CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)

Ali, Endris Aman	168	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Ali, Rana Muhammad Athar	194	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Almegrin, Wafa	104	CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3)
Alsalamah, Shatha	104	CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3)
Alshehri, Hajar	104	CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3)
Anderson, Bethany M	276	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Andrews, Katherine	265	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Andrews, Kathy	201	CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1)
Ang, Ching-Seng	204	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Angrisano*, Fiona	116, 120	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Ansah, Patrick	111	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Anstead, Clare	270, 296, 266, 216, 248, 224	CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3), CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Anstey, Nicholas	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Anzenhofer, Christian	201	CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1)
Apicella, Riannon	292	S1 (Tue, 2025/7/1 11:00-11:30; Conference room 3)
Argyropoulos, Dionne	241	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Arinanto, Leonhard Satrio	286	CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)
Ash, Amanda	175, 138, 164, 170, 249	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2), CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3), CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3), CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Ashby, Sara	288	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)

Ashton, Trent	116	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Atapattu, Ushani	273, 150, 288, 272, 287	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3), CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Atkinson, Peter	124	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3)
Awalt, Jon Kyle	141	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Awalt, Kyle	171, 132	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1)
Awandare, Gordon A.	274	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Ayisi, Franklin	173	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Azizan, Suffian	118	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Bahlo, Christiane	191	CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3)
Baker, Louise	205, 248, 266, 216	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Balan, Balu	205, 248, 266, 216, 231, 237, 254	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Bandala Sanchez, Esther	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Baneth, Gad	277	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Bangre, Oscar	111	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Barbosa, Amanda.D	175	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)

Bareng, Paolo	222, 251	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Barnes, Claudia	116, 148, 127	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Barry, Alyssa	222, 127, 259, 247, 253, 274	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Bartlett, Adam	251	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Barton, Di	298	P4 (Thu, 2025/7/3 9:45-10:30; Conference plenary room)
Barton, Diane	203	CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3)
Barton, Xavier	271	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Batterham, Philip	224	CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Baum, Jake	131, 125	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2), CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Baxendell, Sandra	242, 235	CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Beddoe, Travis	194	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Beeson, James	295, 160, 217, 228, 222, 145, 197, 227, 253, 176, 137	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)

Beugnet, Frédéric	288	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Beveridge, Ian	299, 242, 181, 115, 235	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2), CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Bhatta, Tarka	113	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Bischof, Rob	112, 117	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Blagborough, Andrew	116, 120	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Blazejak, Katrin	292	S1 (Tue, 2025/7/1 11:00-11:30; Conference room 3)
Bliss, Elouise	186	CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Boakye, Daniel A.	173	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Boddey, Justin A.	278	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Boe-Hansen, Gry	121, 178	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Bongiovanni, Felicia	110	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Boonroumkaew, Patcharaporn	290	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Bosch, Jürgen	227	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Bott, Nathan	223, 161, 196	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Boulet, Coralie	141, 127, 122	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP16.1 (Thu, 2025/7/3 12:15-12:30; Conference room 1)
Bouphakaly, Oula	249	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Bourke, Caitlin	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Bowles, Vern	270	CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)

Bowles, Vernon M.	248, 224	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Boyd, Davina	249	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Bradbury, Richard	293, 280	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Brand, Stephen	141, 132	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1)
Brar, Kabir	299, 182	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3)
Braun, Anne-Sophie C.	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Brilhante, Andreia Fernandes	277	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Brindley, Paul	290, 291	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Brown, Keira	203	CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3)
Brustenga, Leonardo	299	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Buchanan, Hayley	295, 199	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
Bullen, Hayley	148, 116, 120	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Bulumulla, Sugandika	175	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Burden, Chloe	157	CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3)
Bury, Connor	250, 198, 206	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Cabezas-Cruz, Alejandro	181	CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2)
Cafiso, Alessandra	266	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Callery, James	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)

Calvani, Nichola E. D.	135	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Calvani, Nichola Eliza Davies	194	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Campbell, Bronwyn	223, 161	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Campbell, Patricia Therese	289	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Cantacessi, Cinzia	161	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Carabott, Melissa	161	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Carabott, Melissa J.	196	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Caraguel, Charles	124, 213	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3), CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Carias, Lenore L.	227	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Cauci, Charles	115	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Challis, Matthew	189	CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Chambers, Michael	292	S1 (Tue, 2025/7/1 11:00-11:30; Conference room 3)
Chan, Li Jin	128, 154, 133	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Chandramohan, Daniel	160	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Chang, Bill	268, 269	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)
Chaumeau, Victor	174	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Chen, Tian	149	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Chishimba, Sandra	176	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Chisholm, Scott	118	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Chitnis, Chetan E.	119	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Chittavong, Malavanh	249	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)

Chmielewski, Jill	153, 128, 146, 154, 145, 133	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Chng, Lena	252	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Chowdhury, Sumaiya	226, 195, 225	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Clinical trial team, NCT04319380	160	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Cobbold, Rowland	155	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3)
Cobbold, Simon	264, 283	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
Coff, Lachlan	223	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Colella, Vito	273, 150, 288, 251, 277, 281, 272, 280, 287, 289, 296	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3), CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2), CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Collier, Sophie	187	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Constantinoiu, Constantin	267	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)
Conway, Eamon	241, 110	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Copedo, Joanna	140	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Correia Faria, Joana R.	239	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)

Cotton, Steve	191	CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3)
Counihan, Natalie	127, 180, 276	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Cowman, Alan	141, 252, 211, 126, 278	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1), CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Cozijnsen, Anton	156, 118	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Crabb, Brendan	127	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Creek, Darren	116, 245, 244, 188, 129, 183, 189, 158, 276, 151	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1), CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Crouch, Mikayla	221, 220	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Cumming, Bonny	192, 193	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Cummins, Scott	209	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Currie, Bart	165	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Cwilinski, Krystina	225	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Czabotar, Peter	266, 216, 237	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)

Dagley, Laura F.	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Dalton, John	225	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Dans, Madeline	141, 171, 132, 127, 122	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP16.1 (Thu, 2025/7/3 12:15-12:30; Conference room 1)
Dawood, Wisam	201	CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1)
Dawson, Aurelie T	126	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1)
Day, Karen	111, 118	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Day, Nicholas	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
De Koning-Ward, Tania	118, 127, 180, 276	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
De Paoli, Amanda	276	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
De Silva, Tilini K.	113	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Dean, Emily	214	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3)
DeCristi, Hayley	166	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Deed, Samantha	111	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Delisle, Lizenn	140	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Deng, Haowen	187	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Derseh, Habtamu	112	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Deshmukh, Arunaditya	119	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Dhorda, Mehul	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Di Antonio, Marco	239	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Dicko, Alassane	160	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)

Dietrich, Melanie	234, 128, 154, 227, 133	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Dillon, Annaliese	188	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Distiller, Amy	263, 275	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Dite, Toby A.	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Dixon, Matthew	261, 199, 254	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Dizdarevic, Christina	180	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Dobaño, Carlota	137	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Dodean, Rozalia A.	156	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Dogovski, Con	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Dondorp, Arjen	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Donnelly, Sheila	226, 225, 195	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Douglas, Ian	296	CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Drago, Chiara L.	227	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Drakeley, Chris	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Dramcanin, Marija	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Duffy, Michael	295, 118	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Duru, Vincent C.	179	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Dziekán, Jerzy	252, 211	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)

Eden, Carl	288	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Edgington-Mitchell, Laura	276	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Egan, Siobhon	271, 138	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2)
Eliza Davies Calvani, Nichola	166, 167, 157	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3), CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3), CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3)
Elliot, Crystal	243	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Ellis, John	260	CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Emery-Corbin, Samantha	297, 172	P3 (Thu, 2025/7/3 9:00-9:45; Conference plenary room), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Evelyn, Cindy	141, 126, 199	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
Fabb, Stewart A.	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Farnell, Rebecca	191	CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3)
Farr, Ryan	113	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Farrell, Sarah N.	156	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Feleke, Sindew	284, 240	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Feleke, Sindew M.	173	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Feleke, Sindew Mekasha	215, 218	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Felgner, Philip	103	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Felgner, Philip L	290	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Feng, Gaoqian	145	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Fernando, Deepani D	207, 208	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2)

Fernando, Lakvin	263, 275	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Ferreira Costa, Joana	246	CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)
Fidock, David	141	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Field, Matt	101	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Firestone, Simon	113, 149	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2), CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Firth, Genta	239	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Fischer, Katja	207, 208	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2)
Fisher, Gillian	265	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Flint, Gem	239	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Flint, Liliith	159	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Fontaine, Joseph	271	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Ford, Ameila	120, 116	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Forsyth, Maureen	288	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Fowkes, Freya	174, 228	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Fowkes, Freya J. I.	197	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Francis, Emily	243, 162, 135	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Francis, Josh	165	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Frempong, Kwadwo	240, 173	CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Frolich, Sonja	169	CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
G Richesb, Andrew	245	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1)

G. Svärd, Staffan	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
G. van Dooren, Giel	212	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Gabriela, Mikha	128, 154, 133	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Gaiarsa, Stefano	266, 216	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Galli, Silvia	239	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Gancheva, Maria	116, 250, 198, 206, 146	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)
Gardner, Brett R.	149	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Gasser, Robin	268, 269, 293, 202	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3), CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Gasser, Robin B.	288, 277, 224, 280, 287, 257	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2), CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3)
Gauci, Charles	168	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Gauci, Charles G.	299, 242, 181, 149	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2), CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Gbariel, Sarah	164	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Gebi, Teha Shumbej	244	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)

Geoghegan, Niall	141, 199, 255, 126	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1)
Ghafar, Abdul	242, 179, 181, 182, 235	CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2), CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Giacomin, Paul	101, 103, 185, 291	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Giannangelo, Carlo	276, 245, 188, 183	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1), CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Gibson, Justine	155	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3)
Gilberger, Tim	211	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Gilson, Paul	116, 141, 118, 148, 120, 127, 122	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP16.1 (Thu, 2025/7/3 12:15-12:30; Conference room 1)
Glukhova, Alisa	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Gofton, Alexander	266, 216	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Gonelli, Christopher	204	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)

Goodman, Christopher	118, 156, 142	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), P2 (Wed, 2025/7/2 9:45-10:30; Conference plenary room)
Goodman, Dean	295	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Gouil, Quentin	266, 216, 128, 133	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Govind, Suresh Kumar	106, 107	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1), CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1)
Graffeo, Bridget	299	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Grant, Warwick	215, 173, 284, 240, 267, 280	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Graves, Patricia M	267	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)
Graves, Stephen	113	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Gray, Darren	190	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Greenhill, Andrew	191	CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3)
Greenwood, Brian	160	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Griffin, Michael D.W.	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Grigg, Matthew	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Grotz, Myriam	118	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Gunaratna, Indeewarie	272	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Gupta, Priya	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
H. Ryanb, John	245	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1)
Hall, Luke	260	CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)

Harris, Alexander	160	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Hasang, Wina	204, 258	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Hawe, Mei	197	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Haydon, Shane	202	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Hayward, Douglas	144	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
He, Qixin	274	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Hedtke, Shannon	215, 173, 284, 240, 267, 280	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Hendrickson, Emily	215, 284, 240, 173	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Heng-Chin, Ashleigh S.	197	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Henshall, Isabelle	153, 145	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Hii, Sze Fui	251, 281, 296	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Hilko, David	265, 184	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Hodgkinson, Jane	257, 285	CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3), CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3)
Hofferek, Vinzenz	159	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Holt, Deborah	165	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Home, Jessica	295	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)

Hoopes, Jessica	192, 193	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Horner, Leni	112	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Hudson, Jemma	223, 161, 196	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Hufschmid, Jasmin	299, 149	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Huggins, Lucas	150, 277, 272, 296, 280	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2), CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Huggins, Lucas G.	287	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Hutson, Kate	140, 143	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Huy, Rekol	280	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Huynh, Long	187, 255	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Huynh, Priscilla	135	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Hysa, Alessia	137	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
I. McFadden, Geoff	246	CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)
I. Webb, Andrew	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Insisiengmay, Bounnaloth	170, 249	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3), CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Intapan, Pewpan	290	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Investigators, Australian Centre of Research Excellence in Malaria Elimination	197	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
J. Emery-Corbin, Samantha	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)

J. Fairweather, Stephen	212	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
J. Sandow, Jarrod	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Jabbar, Abdul	168, 299, 242, 179, 181, 115, 149, 182, 235, 194	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2), CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3), CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3), CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3), CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Jackson, Paul	141, 132	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1)
Jackson, Philomina	173	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Jaensch, Susan	144	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Jarman, Kate	141, 252	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Jayakrishnan, Niva	254	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Jayawickrama, Amalie	283	CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
Jaywickrama, Amalie	264	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Jefferson, Natalie	232	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Jex, Aaron	205, 216, 231, 172, 254, 266, 248, 224	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Johnson, Michael	131	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)

Jones, Malcolm K	144	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Jung, Nicolai C.	227	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
K Smyth, Gordon	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
K.L.D., Tharaka D. Liyanage	149	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Kaldor, John	281	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Kalinna, Bernd	130, 114	CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2), CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Kamil, Mohd	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Kancharla, Papireddy	156	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Kang, Hyungsuk	144	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Kapoor, Shilpa	248, 216, 224, 270	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Karbanowicz, Thomas	216	CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Karl, Stephan	247	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Kate Francis, Emily	166	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Katris, Nicholas	264, 282	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
Kazura, James W.	247	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Kearney, Ellen	174, 197	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Keatley, Sarah	170	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Kelly, Jane X.	156	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Kelly, John M.	239	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Kelly, Olivia	243, 162	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3)

Keokhamphavanh, Boulay	170	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Ketprasit, Nutpakal	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Khandokar, Yogesh	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Khieu, Virak	150, 280	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Khoshmanesh, Khashayar	205	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)
Khurana, Sachin	264, 237, 211	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
King, Christopher L.	227	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
King, Tanya	112	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Kiniboro, Benson	247, 222	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Kittiphanakul, Praphan	174	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Knight, Breanna	170	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Koehler, Anson	202, 209	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3), CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Koehler, Anson V.	287, 285	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2), CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3)
Kohler, Anson	293	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Kokuhennadige, Vivin	204	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Komander, David	264	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Kombut, Benishar	204, 145	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Kotze, Andrew C	144	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Kumar, Manoharan	101	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Kumar Sharma, Vivek	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)

Kumarasamy, Vinoth	106, 107	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1), CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1)
Kurtovic, Liriye	160, 176, 137, 145	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Kurtovic, Liriye {Lydia}	217	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Kurz, Thomas	201	CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1)
Lacerda, Marcus	241	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Lai, Keng Heng	153	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1)
Lam, Alex	172	CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Laman, Moses	259, 247, 176, 222, 145	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Lamont, Macie	241	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Lang, Ruijia	256	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Lantero-Escolar, Elena	278	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Larcher, Katrina	199, 254	CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Larkins, Andrew	164, 170, 249	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3), CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3), CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Lau, Colleen L	267	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)
Lautu-Gumal, Dulcie	259, 247	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Le, Jennifer	188, 183	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)

Lee, Rogan	152	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Lee, Wei	165	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Leeming, Michael G.	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Lehane, Adele	141, 147, 127, 256	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Lekkala, Sai	264	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Leonard, Rachel A	256	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Levendis, Joshua	263, 275	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Liang, LI	103	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Liang, Ruijia	147	CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1)
Liffner, Ben	255	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Ligda, Panagiota	277	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Lightowlers, Marshall	102	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Lim, Pailene	241, 119, 230	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Lin, Janise	241	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Lin, Qingqing	261	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1)
Ling, Dawson	255, 126	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1)
Ling, Elysia	168, 242	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3)
Literski, Alysha	148	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Liu, Joy	205	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)

Liyanage, Tharaka	299	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Lo, Nathan	266, 216	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Long, Lingxiao	239	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Longley, Rhea	241, 228, 222, 233, 119, 133, 230	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Looker, Oliver	148	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Lopaticki, Sash	261, 199, 133, 254	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Loukas, Alex	101, 103, 185, 290, 291	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Lowes, Kym	141, 252	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Lucet, Isabelle	172	CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Luque, Daniel	131	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Lymber, Alan	138, 238	CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2), CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Lymbery, Samuel	238	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Lyons, Frankie	128, 154	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)

Lyons, Frankie M. T.	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
M Fisher, Gillian	245	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1)
Macdonald, Jacinta	201, 265	CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1), CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
MacFadden, Geoffrey	118	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Mackie, Quinn	250	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Mackline, Garae	251	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
MacRaid, Christopher	244, 129, 158, 151	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Madan, Sonakshi	247	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Madi Salloum, Priscila	108	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Maguire, Capella	212, 262	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1)
Maher, Thomas E.	239	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Maia, Carla	277	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Maier, Alexander	141	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Makota, F. Victor	262, 219	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Makota, Victor	212	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Malachin, Alyssa N.	227	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Maleewong, Wanchai	290	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Manickam, Yogavel	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Manning, Laurens	176	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Mansouri, Mahta	123	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Mantila, Daisy	145	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)

Manzanell, Ralph	287	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Mao, Emma	250	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Marapana, Danushka S	126, 254	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Marshall, Alexis	161	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Martin, Lily	213	CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Martin, Storm	229	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Martinez, Francisco J.	119	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Massetti, Luca	273	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3)
Mayfield, Helen J	267	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)
Mayxay, Mayfong	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Mazhari, Ramin	241, 222, 133	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
McAllister, Milton	206	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
McCann, Kirsty	247, 127, 259, 253	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
McCarthy, James	261, 199, 254	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
McCauley, John A.	278	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
McCluskey, Adam	250	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
McConville, Malcolm	159, 264, 282	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
McCorquodale, Abbey	183	CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
McCosker, Kieren	121	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)

McDonagh, Phillip	273	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3)
McDonald, Analise	243	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
McFadden, Geoff	261	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1)
McFadden, Geoffrey	295	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
McFadden, Geoffrey I.	156, 142	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), P2 (Wed, 2025/7/2 9:45-10:30; Conference plenary room)
McGowan, Michael	121	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
McGowan, Sheena	123	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Mchuge, Connor	103, 101, 185	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
McHugh, Emma	263, 275	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
McKay-Demeler, Janina	243, 167, 162	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3)
McVernon, Jodie	110	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Mehra, Somya	259, 222	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Mekonen, Bacha	218	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Mencke, Norbert	292	S1 (Tue, 2025/7/1 11:00-11:30; Conference room 3)
Mengi, Alice	204	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Meumann, Ella	165	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Michon, Pascal	222	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Miles, Kim	101, 185	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Mitreva, Makedonka	291	CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)

Modak, Joyanta	127, 180	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Mollard, Vanessa	156, 246	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)
Monti, Ludovica	239	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Morrow, Joshua	245, 169, 129, 244	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Mortensen, Sofia	211	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Morton, Craig J.	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Mueller, Ivo	241, 259, 110, 247, 228, 119, 176, 222, 145, 133, 233	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Mullens, Elizabeth	117	CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Munro, Jacob	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Mustafa, Bahar E	181	CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2)
N. Vyas, Simran	167	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Nagaraja, Haleagrahara	101	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Nakajima, Rie	290	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Nam, Madisson	288	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Namgyel, Ugyen	150	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)

Nammunige, Nirupama A	207	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Naniogue, Sedou	173	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Narh, Charles	274	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Nate, Elma	259	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Nathaniel, Bernard	180	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Naung, Myo	222, 237, 253	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Nery, Susana	251, 281	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Nery, Susana Vaz	296	CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Newton, Paul	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Ngo, Anna	141, 252	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Nguon, Chea	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Nguyen, Anne	131	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Nguyen, Dinh Ng	281	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Nguyen, Hanh	165, 118	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Nguyen, Loan	121	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Nguyen, Phuong Thao	226, 225	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Nguyen, Thi Thuy	277	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Nguyen, Trung Duc	225	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Nguyen, Tuan Anh	225	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)

		CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP16.1 (Thu, 2025/7/3 12:15-12:30; Conference room 1)
Nguyen, Will	116, 141, 120, 127, 122	
Nicholson, Eleanor	246	CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)
Nielsen, Torben	124	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3)
Nijhof, Ard M.	179, 181	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2)
Nosten, Francois	174	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Nowak, Barbara	223, 161, 196	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
O'Handley, Ryan	206	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
O'Connor, Suji	134	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
O'Flaherty, Katherine	174, 197, 228	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
O'Handley, Ryan	124, 214, 250, 198, 213	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3), CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3), CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Olsen, David	252, 278	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Ome-Kaius, Maria	259, 247, 222, 145	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Ong, Chian Teng	178	CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)

Onwuka, Akachukwu	258	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Ooi, Zi Kang	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Opi, D. Herbert	227, 217, 228, 176, 137, 145	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Opoku, Millicent	267, 215, 173, 284, 240	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
O'Rourke, Denise	149	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Osei, Joseph H. N.	173	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Oskam, Charlotte	271	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Otoo, Sampson K.	173	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Ouedraogo, Jean-Bosco	160	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Page, Stephen	250	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Pal, Martin	114	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Panjikar, Santosh	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Papenfuss, Anthony	266, 141, 216	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Pareek, Kapil	118	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Parkyn Schneider, Molly	127	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Parr, Jonathan B.	218	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Pascual, Mercedes	111	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Pathirana, Nuwandi	238	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)

Paxman, Matthew	165	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Pazzagli, Lucia	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Peck, Ashleigh	138	CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2)
Pekin, Kaitlin	176, 145	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Penington, Jocelyn	141	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Perry, Trent	224, 270	CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Peto, Thomas	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Petter, Michaela	118	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Phommasone, Koukeo	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Pi-Bansa, Sellase	173	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Pickering, Darren	101, 185	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Piedrafita, David	112, 191, 117	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Piontek, Michael	137	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Plantard, Olivier	266	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Ponder, Winston	209	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Poulin, Robert	108	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Poulsen, Sally-Ann	184, 265	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Pouton, Colin W.	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Power, Cecilia	196	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Pradeepkumar, P.I.	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Preativatanyou, Kanok	277	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)

Presburger, Rosemary	108	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Preston, Sarah	112, 191, 117	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Price, David	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Qian, Chunyi	234	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1)
Qiu, Deyun	256, 147, 127	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1), CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Quashie, Nancy	294	P1 (Tue, 2025/7/1 9:15-10:30; Conference plenary room)
R Macdonald, Jacinta	245	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1)
R. Jex, Aaron	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Radih, Zaynab	177	CP16.1 (Thu, 2025/7/3 12:15-12:30; Conference room 1)
Radmann, Christy-Amber	236	CP16.1 (Thu, 2025/7/3 12:15-12:30; Conference room 1)
Rae, Louise	273	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3)
Rajahram, Giri S.	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Rajesh, Vijaishree	233	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Ralph, Stuart	187, 263, 275, 255	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1), CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Ralton, Julie	159	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Ramachandran, Nimitha	221	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Ramsland, Paul	223, 161, 196	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Rasyidi, Fathia	111	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Rawlin, Grant	194	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Raza, Ali	121	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)

Razook, Zahra	259, 247	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Razzok, Zahra	127	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Reeves, Grace	155	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3)
Rego, Ryan O.M.	179	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Rendall, Anthony	203	CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3)
Richards, Jack	165	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Riemer, Jan	266, 216	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Riley, Tamara	193	CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Rios-Teran, Cecilia	111	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Rissland, Olivia	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Ristevski, Nic	231	CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)
Rivory, Phoebe	152	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Robert E. Ansell, Brendan	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Robinson, Leanne	259, 222, 145	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Robinson, Leanne J.	247	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Roby, Justin	130	CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Rodpai, Rutchanee	290	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Roe, Merryn	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Roeber, Florian	292	S1 (Tue, 2025/7/1 11:00-11:30; Conference room 3)
Rogers, Kelly	141, 199, 126	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1)

Rogerson, Stephen	204, 258, 176	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Rohackova, Helena	179	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Rojrung, Rattaporn	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Romeo, Ornella	198, 206	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Ross, Kirstin	193	CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Roy, Pradip	205, 266, 216, 237	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Ruparel, Ushma	264	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Ruscher, Roland	101, 185	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Ruybal-Pesantez, Shazia	259, 247	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Ryan, Rachael	101	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Ryan, Stephanie	185	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Ryan, Una	175	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Rymill, Prudence	251, 296	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
S Skinner-Adams, Tina	245	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1)
Sadaow, Lakkhana	290	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Sais, Dayna	226, 225, 195	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)

Saliba, Kevin J	177, 236	CP16.1 (Thu, 2025/7/3 12:15-12:30; Conference room 1), CP16.1 (Thu, 2025/7/3 12:15-12:30; Conference room 1)
Samarawickrama, Gangi R	207	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Santos, Harvey	121	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Sarkar, Eti	103	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Sassera, Davide	266, 216	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Sattabongkot, Jetsummon	241, 230	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Saunders, Eleanor	159	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Saunders, Richard	143	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Sawasdichai, Sunisa	174	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Sayasone, Somphou	164	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Sayers, Claire	125	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Scally, Stephen W.	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Scammells, Peter	123	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Scaralal, Jahit	137	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Scheerlinck, Jean-Pierre	270	CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Schneider, Molly	116	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Schoffer, Kael	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Schulz, Danae	118	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Scott, Ian	144	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Scott, Nicholas	264	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Seager, Benjamin A.	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Seal, Alexa	232	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Seizova, Simona	252	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)

Sernee, Fleur	159	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Shakeel, Shabih	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Shami, Gerald J.	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Shamsi, Shokoofeh	232, 130, 203	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3), CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2), CP18.1 (Thu, 2025/7/3 12:15-12:30; Conference room 3)
ShandreMugan, Rekha	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Sharma, Amit	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Sharma, Natasha	209	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Sheorey, Harsha	293	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Shibazaki, Yuri	199	CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
Shibu, Mohini	261	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1)
Shrestha, Himal	284	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Siba, Peter M.	247	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Siddharam, Bagale	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Siddiqui, Ghizal	116, 276, 188, 169, 129, 151, 189	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Siddiquid, Ghizal	245	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1)
Siddle, Hannah	121, 221, 220, 178	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Sikder, Suchandan	101, 103, 185	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)

Simonpietri, Amber	264, 282	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
Simpson, Julie	174, 228	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Singh, Karan	264, 283	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
Singla, Shamit	169	CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
Sirwani, Neha	267, 173, 240	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Skinner-Adams, Tina	265	CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1)
Slapeta, Jan	243, 162, 144, 152	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Slattery, Joshua	114	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Sleebs, Brad	116, 141, 171, 132, 120, 127, 122	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1), CP16.1 (Thu, 2025/7/3 12:15-12:30; Conference room 1)
Smith, Lauren	241, 233	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Smith, Maxine	185	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Smith, Zelda	127	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Smout, Michael	290, 291	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)

Solomon, Hiwot	218	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Sondermann, Holger	211	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Song, Jiangning	268, 269	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)
Sotiraki, Smaragda	277	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Spithill, Terry	194	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Spry, Michael	193	CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Stack, Colin	232	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Stanhope, Katelyn M.	253	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Stark, Damien	260	CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Stavru, Fabrizia	266, 216	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1)
Steel, Senna	120	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Steen, Senna	116	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Stenos, John	113	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Stevenson, Mark	124, 242, 235, 194	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3), CP6.1 (Tue, 2025/7/1 14:45-15:00; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3), CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Stewart, Cameron	113	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Stocker, Thomas	144	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Strazzeri, Don	292	S1 (Tue, 2025/7/1 11:00-11:30; Conference room 3)
Stupple, Paul	188	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Su, Wenyin	141	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Sukee, Tanapan	135, 209, 257, 285	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3), CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3), CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3), CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3)

Sumanam, Sunita	223	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Sumanam, Sunita B.	287	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Sutherland, Colin	105	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
T. Andrews, Katherine	245	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1)
Tabe, Stephanie	251	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Tabor, Ala	121, 221, 266, 216, 220, 178	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Taggart, Kelly	170	CP12 (Wed, 2025/7/2 11:00-12:30; Conference room 3)
Tai, Chia-Wei	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Takashima, Eizo	228, 222	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Taki, Aya C.	257	CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3)
Taleo, Fasahah	251, 296	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Tan, Li Lynn	234, 133	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Tan, Mun Hua	111	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Tang, Jingyi	118	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Tang, Junwei	158	CP22.1 (Thu, 2025/7/3 16:30-17:00; Conference room 1)
Tasew, Geremew	218	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Taylor, Chris	183	CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Taylor, Sara	207, 208	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2)
Taylor, Shannon	166	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)

Tesine, Paula	204	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Tetteh, Kevin	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Tham, Wai-Hong	234, 128, 227, 154, 133	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Thomas, Adam	217, 176	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Thurgood, Peter	205	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)
Tichkule, Swapnil	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Tiedje, Kathryn	111	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Tilley, Leann	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Tiu, Jarryd	146	CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)
Tobe, Shanan	271	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2)
Tolpinrud, Anita	149	CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Tomsen, Robert	267	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)
Tong, Joshua	128	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Tonkin, Chris	264	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Tonkin, Christopher	282, 283, 252	CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Tortorelli, Giada	246	CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)
Tran, Nham	226, 225, 195	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Traub, Rebecca	273, 288, 281, 280	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2), CP23 (Thu,

		2025/7/3 15:30-16:45; Conference room 2)
Tripura, Rupam	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Tsuboi, Takafumi	228, 222	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2), CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Tumwebaze, Patrick	245	CP1.1 (Tue, 2025/7/1 12:15-12:30; Conference room 1)
Tynan, Alexander	130	CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Tyrell, Leah	194	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Uboldi, Alessandro	282, 283	CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1)
Uboldi, Alex	264	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Ugbobuaku-Roys, Annie	276	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Unger, Holger	204	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Unsworth, Nathan	113	CP20.1 (Thu, 2025/7/3 14:45-15:00; Conference room 2)
Uthayakumar, Chelsie	166	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Vahi, Ventis	241	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
van Dooren, Giel	141, 219, 262, 256	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP19.1 (Thu, 2025/7/3 14:30-15:00; Conference room 1), CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Vann, Molyden	280	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Vaughan, Ashley M.	133	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Vaughan, David	143	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Veitch, Maggie	185	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Vibin, Jessy	259	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)

Vijay, Amrita	205, 248, 266, 216, 237	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP7.1 (Tue, 2025/7/1 16:45-17:00; Conference room 1), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Villanueva-Cabezas, Juan Pablo	289	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
von Seidlein, Lorenz	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
Waldron, Molly	296	CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Walker, Martin	280	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Walker, Nick	241	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
Walther, Deonne	291	CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Wang, Susie	252	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Wang, Tao	268, 202	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP18 (Thu, 2025/7/3 11:00-12:15; Conference room 3)
Wang, Yusheng	256	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Ward, Michael	167, 144, 152	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3)
Wardak, Ahmad	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Webster, Bonnie	209	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Welikadage, Sugandhika	270	CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Westman, Mark	243	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Wetzel, David	137	CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2)
Wheatley, Adam	204	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
White, Michael	241	CP5 (Tue, 2025/7/1 13:30-14:30; Conference room 2)
White, Nicholas	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)
White, Oliver	161	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)

Widdicombe, Maree	161, 196	CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3), CP9 (Tue, 2025/7/1 15:30-17:00; Conference room 3)
Wiethoelter, Anke	273	CP3 (Tue, 2025/7/1 11:30-12:30; Conference room 3)
Wilde, Mary-Lou	261, 142, 246	CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), P2 (Wed, 2025/7/2 9:45-10:30; Conference plenary room), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1)
Wilhelm, Olivia	204	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
William, Timothy	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)
Williams, Oscar	204	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Willoughby, Roger	166, 135	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3), CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Wilson, Danny	116, 250, 153, 169, 198, 206, 146, 145, 129, 151	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3), CP4.1 (Tue, 2025/7/1 14:30-15:00; Conference room 1), CP13 (Wed, 2025/7/2 13:30-14:30; Conference room 1), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP13.1 (Wed, 2025/7/2 14:30-15:00; Conference room 1), CP17.1 (Thu, 2025/7/3 12:15-12:30; Conference room 2), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Wines, Laura	248	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Winnicki, Anna C.	227	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Winslow, Mackrina	289	CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Wiradiputri, Kharizta	252	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Wittlin, Sergio	141	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Wong, Wilson	222	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Wong, Yide	103, 185	CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2), CP14 (Wed, 2025/7/2 13:30-14:45; Conference room 2)
Wright, Gavin J.	230	CP20 (Thu, 2025/7/3 13:30-14:45; Conference room 2)

Wright, Grace	176	CP17 (Thu, 2025/7/3 11:00-12:15; Conference room 2)
Xie, Stanley C.	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Yambo, Phantica	204	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Yang, Ying Ting	224, 270	CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3), CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Ye, Xi	184	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Yeoh, Lee	295	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Yeoh, Lee M.	118, 227, 253	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2)
Yong, Rachael	274	CP10 (Wed, 2025/7/2 11:00-12:15; Conference room 1)
Young, Kelly	250	CP6 (Tue, 2025/7/1 13:30-14:45; Conference room 3)
Young, Neil	268, 269, 223, 209	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2), CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Young, Neil D.	288, 287, 135, 257, 285	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2), CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3), CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3), CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3)
Young, Neil David	194	CP24 (Thu, 2025/7/3 15:30-16:45; Conference room 3)
Yu, Chunhao	118	CP7 (Tue, 2025/7/1 15:30-16:45; Conference room 1)
Yuxin Mao, Emma	116	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1)
Zadow, Meghan	129, 151	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Zakrzewski, Martha	207, 208	CP8 (Tue, 2025/7/1 15:30-16:45; Conference room 2), CP8.1 (Tue, 2025/7/1 16:45-17:00; Conference room 2)
Zaloumis, Sophie	228	CP11.1 (Wed, 2025/7/2 12:15-12:30; Conference room 2)

Zeglinski, Kathleen	128, 133	CP11 (Wed, 2025/7/2 11:00-12:15; Conference room 2), CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Zemen, Endalew	244	CP4 (Tue, 2025/7/1 13:30-14:30; Conference room 1)
Zendejas-Heredia, Patsy	296	CP23.1 (Thu, 2025/7/3 16:45-17:00; Conference room 2)
Zendejas-Heredia, Patsy A.	288, 280	CP15 (Wed, 2025/7/2 13:30-15:00; Conference room 3), CP23 (Thu, 2025/7/3 15:30-16:45; Conference room 2)
Zerna, Gemma	117	CP14.1 (Wed, 2025/7/2 14:45-15:00; Conference room 2)
Zhan, Qi	111	CP5.1 (Tue, 2025/7/1 14:30-15:00; Conference room 2)
Zhang, Jingjing	168	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Zhang, Ying	252	CP16 (Thu, 2025/7/3 11:00-12:15; Conference room 1)
Zhang, Zhetao	178	CP21 (Thu, 2025/7/3 13:30-15:00; Conference room 3)
Zhao, Huan	168	CP2.1 (Tue, 2025/7/1 12:15-12:30; Conference room 2)
Zhao, Leqian	212	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)
Zheng, Mengwei	257	CP24.1 (Thu, 2025/7/3 16:45-17:00; Conference room 3)
Zheng, Yuanting	268, 269	CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2), CP2 (Tue, 2025/7/1 11:00-12:15; Conference room 2)
Zhou, Qingmiao	132	CP10.1 (Wed, 2025/7/2 12:15-12:30; Conference room 1)
Zhou, Yunyang	276, 189	CP1 (Tue, 2025/7/1 11:00-12:15; Conference room 1), CP22 (Thu, 2025/7/3 15:30-16:30; Conference room 1)
Zhu, David	237	CP19 (Thu, 2025/7/3 13:30-14:30; Conference room 1)

2025 Annual Conference of the Australian Society for Parasitology Inc.

30 June – 3 July, 2025 Melbourne Connect, Melbourne, Vic, Australia

Conference Organisation

Hayley Bullen	Burnet Institute	Conference Co-Chair
Aaron Jex	WEHI	Conference Co-Chair
Elizabeth Aitken	Peter Doherty Institute, University of Melbourne	Conference Committee Member
Lucas Huggins	University Of Melbourne	Conference Committee Member
Sarah Preston	Federation University Australia	Conference Committee Member
Ghizal Siddiqui	Monash University	Conference Committee Member
Nick Smith	ASP	Conference Committee Member
Lisa Jones	ASP	Conference Coordinator

Amazing Conference Volunteers

Prerna Prashanth	Bio21 Institute/University of Melbourne
Rebecca Farnell	Federation University Australia
Michaela Grima	Deakin University
Tanya King	Federation University
Vivin Kokuhennadige	University of Melbourne Peter Doherty Institute
Alex Lam	Walter and Eliza Hall Institute for Medical Research
Jennifer Le	Monash University
Alysha Literski	Burnet Institute
Connor McHugh	James Cook University
Elizabeth Mullens	Federation University Ballarat
Khoi Nguyen	Burnet Institute
Millicent Opoku	La Trobe University
Nic Ristevski	Walter and Eliza Hall Institute for Medical Research
Praidep Roy	University of Melbourne
Natasha Sharma	The University of Melbourne
Senna Steen	Burnet Institute
Kahlia Szabo	Deakin University
Amrita Vijay	Walter and Eliza Hall Institute for Medical Research
Rachael Yong	Deakin University

FLEAS & TICKS SUCK

STOP SUCKING WITH



The longest lasting protection against fleas and ticks*

*In a high-tech collar. Read product leaflet for full instructions. ©2024 Elanco or its affiliates. Elanco Australasia Pty Ltd (ABN 64 076 745 198), Level 3, 7 Eden Park Drive, Macquarie Park NSW 2113. All trademarks are the property of their respective owners. For further information contact: 1800 995 709 from anywhere in Australia Monday to Friday or email productsupport@elancoah.com. ELAN0590. PM-AU-24-0329

Elanco

The *real* parasite threat putting cat lives at risk

New Australian research into feline parasites clarifies that the risk of feline heartworm is low to non-existent, and the real parasite threats are lungworm and tapeworm. The challenge for vets is: are management strategies aligned with this new understanding?

Cats are not the natural host for Heartworm and will mount an immune response if the parasite is encountered, and many regions of Australia experience temperatures that are too low to support the development of larvae.

Heartworm (*Dirofilaria immitis*)

Recent studies confirm an extremely low prevalence of heartworm in Australian cats, suggesting no need for a blanket approach to Heartworm prevention in cats. Data on the prevalence of Heartworm and Tapeworm in Australian cats is limited. New research by Roeber et al. (2024) sought to address this by investigating the prevalence of Heartworm (*Dirofilaria immitis*) and Tapeworm (*Dipylidium caninum* and *Taenia spp.*). The study of shelter and pound cats in Eastern Australia found no positive cases of Heartworm¹. This aligns with earlier findings by Adagra et al. in 2021, suggesting an extremely low prevalence of cat Heartworm². Consequently, a blanket approach to Heartworm prevention for all cats is not necessary. These studies dispelled myths about Heartworm in cats and reinforced the need to protect cats from the relevant parasitic threats – Lungworm and Tapeworm.

“This study highlights the low risk of heartworm in cats across Eastern Australia, advocating for a tailored prevention approach rather than routine year-round treatment,” says Dr Florian Roeber, Business Unit Leader, Wongaburra Research Centre. DVM, PhD., Grad. Dip. Bus. Mgt.

Dr Ryan O’Handley from the University of Adelaide has been involved in developing an open-access Heartworm dashboard to enable Australia’s Heartworm risk landscape to be mapped and examined in real-time. This tool allows veterinarians to confidentially shift from blanket year-round treatment to a benefit-risk assessment model. In addition to these innate factors that prevent Australian cats from contracting Heartworm, providing up-to-date prevalence data can also assist veterinarians in making informed decisions.

“This study highlights the low risk of heartworm in cats across Eastern Australia, advocating for a tailored prevention approach rather than routine year-round treatment.”

Dr Florian Roeber, Business Unit Leader, Wongaburra Research Centre
DVM, PhD., Grad. Dip. Bus. Mgt.

Lungworm (*Aelurostrongylus abstrusus*)

Unlike Heartworm and Tapeworm, Lungworm can cause severe lung damage, often without obvious clinical signs³. Historical data from a 2009 study revealed a 13.8% prevalence in peri-urban semi-feral cats in Victoria⁴. Subclinical infections and diagnostic challenges further complicate detection and management. Therefore, broad-spectrum parasiticides that include Lungworm are essential for all Australian cats to prevent this easily preventable parasite, including indoor cats.

Tapeworms (*Dipylidium caninum* and *Taenia spp.*)

The same study identified a 1.1% prevalence of *Taenia spp.* and no cases of *Dipylidium caninum*. While these results suggest a low prevalence, they highlight the importance of incorporating Tapeworm control in regular worming protocols. Quarterly worming that covers Tapeworms and other gastrointestinal parasites is essential for cats.

A Call to Action

Veterinarians must challenge their own misconceptions and prioritise cat parasite control compliance for Lungworm and Tapeworm. Australian vets suggest that 25% of cats they see have no protection against parasites^{5,6}. Is this a similar number for your clinic? By fostering awareness and implementing effective prevention strategies, we can safeguard the health

and well-being of our feline companions. Regular parasite control protocols must address the full spectrum of the prevalent Australian parasites, including emerging threats like Lungworm. Veterinarians play a critical role in educating pet owners about these risks and ensuring that preventive care reflects current research findings.

Easy-to-administer, long-lasting control that targets the major parasitic threats will enhance compliance. Cat owners should be offered products that fulfil these requirements. Does your Feline Wellness Program cover cats from Lungworm and Tapeworm and is it long-lasting?



Try Felpreva® for fuss-free protection



All-in-one parasite cover

Controls the main feline parasites, including paralysis ticks, lungworm, and tapeworm

13
weeks

Longest-lasting* single spot-on

Fewer treatments to keep them protected – only four times per year*



Low volume & quick absorption

With the new active ingredient, tigolaner



ISFM approved

Easy-to-give certified by the International Society of Feline Medicine

*Felpreva® is for cats and kittens from 10 weeks of age and greater than 1kg. It treats and prevents fleas and treats and controls paralysis ticks (*Ixodes holocyclus*) for 13 weeks.

REFERENCES:

1. Roeber, F., Apicella, R., Chambers, M., Strazzeri, D., Mencke, N. and Blazejak, K., 2024. Prevalence of *Dirofilaria immitis*, *Dipylidium caninum* and *Taenia spp.* in populations of cats from shelters and research colonies, in two endemic regions of eastern Australia. *Current Research in Parasitology & Vector-Borne Diseases*, 6, p.100226.
2. Adagra, C., Squires, R., Adagra, A., Elliman, J. and Constantinoiu, C., 2021. Prevalence of infection with *Dirofilaria immitis* in cats in Townsville, Australia. *Veterinary Parasitology: Regional Studies and Reports*, 24, p.100580.
3. Gerdin, J.A., Slater, M.R., Makolinski, K.V., Looney, A.L., Appel, L.D., Martin, N.M. and McDonough, S.P., 2011. Post-Mortem Findings in 54 Cases of Anesthetic Associated Death in Cats from Two Spay—Neuter Programs in New York State. *Journal of feline medicine and surgery*, 13(12), p.959-966.
4. Lacorcchia, L., Gasser, R.B., Anderson, G.A. and Beveridge, I., 2009. Comparison of bronchoalveolar lavage fluid examination and other diagnostic techniques with the Baermann technique for detection of naturally occurring *Aelurostrongylus abstrusus* infection in cats. *Journal of the American Veterinary Medical Association*, 235(1), p.43-49.
5. Sapio Market Research of Australian Veterinarians, 2021.
6. <https://animalmedicinesaustralia.org.au/report/pets-in-australia-a-national-survey-of-pets-and-people-2/>



Product spotlight

BORDIER
Strongyloides ratti ELISA
For the quantitative detection of IgG antibodies against *Strongyloides* nematodes in human serum
ARTG Listed: 274944



SCD IS PROUDLY AUSTRALIAN OWNED
Established in 2008, Southern Cross Diagnostics distributes innovative life science and diagnostic solutions to Australian medical laboratories



Product spotlight

BORDIER
Schistosoma mansoni IgG ELISA
For the diagnosis of human schistosomiasis.
ARTG Listed: 274944



BEST IN CLASS SERVICE
Supporting Australian laboratories throughout all stages of testing



SPECIALIST IN PARASITOLOGY
SCD focuses on providing high quality parasitology kits with technical support locally available



Product spotlight

ALETHIA MALARIA

TRUST IN YOUR MALARIA DIAGNOSIS

Detect all five Plasmodium Species
Simplify your Malaria workflow



Alethia Malaria Features:

- 100% Negative Predictive Value
- Simple, molecular method
- 2-3 minutes of hands on time
- 1-10 samples per run
- Results in under 45 minutes
- HRP2 protein deletion detected

Report with confidence

Alethia Malaria detects all five Plasmodium species down as low as 2 parasites per μ L. Once negative on Alethia, there is no need for thick and thin blood films.

The method is simple without need for specialised training or technical expertise.

Contact SCD for further information or to book a demonstration.
info@scdiagnostics.com.au



Empower Your Parasitology Research

Discover powerful tools that support every stage of your workflow

Genomic Analysis of Parasites

- ✓ High-yield and rapid automated DNA/RNA purification from blood, stool, vectors, or culture samples with Maxwell® RSC Systems.
- ✓ Streamlined protocols for difficult matrices.



Pathogen Detection and Quantification

- ✓ Highly sensitive GoTaq® qPCR and RT-qPCR reagents for accurate parasite load assessment.
- ✓ Multiplex amplification options for complex infections.

Host-Parasite Interaction Studies

- ✓ Cell viability, apoptosis, and stress assays for evaluating host responses.
- ✓ Customisable solutions for high-throughput analysis.



...and more!

Get in touch



Explore Solutions





Putting science first.

Post-Doctoral Fellowships at New England Biolabs®

Established in the mid 1970s, New England Biolabs, Inc. (NEB®) is the industry leader in the discovery and production of enzymes for molecular biology applications and now offers the largest selection of recombinant and native enzymes for genomic research. NEB continues to expand its research and development into areas related to DNA replication, programmable nucleases, epigenetics, molecular parasitology, sample preparation for next generation sequencing, synthetic biology, glycobiology and RNA analysis.

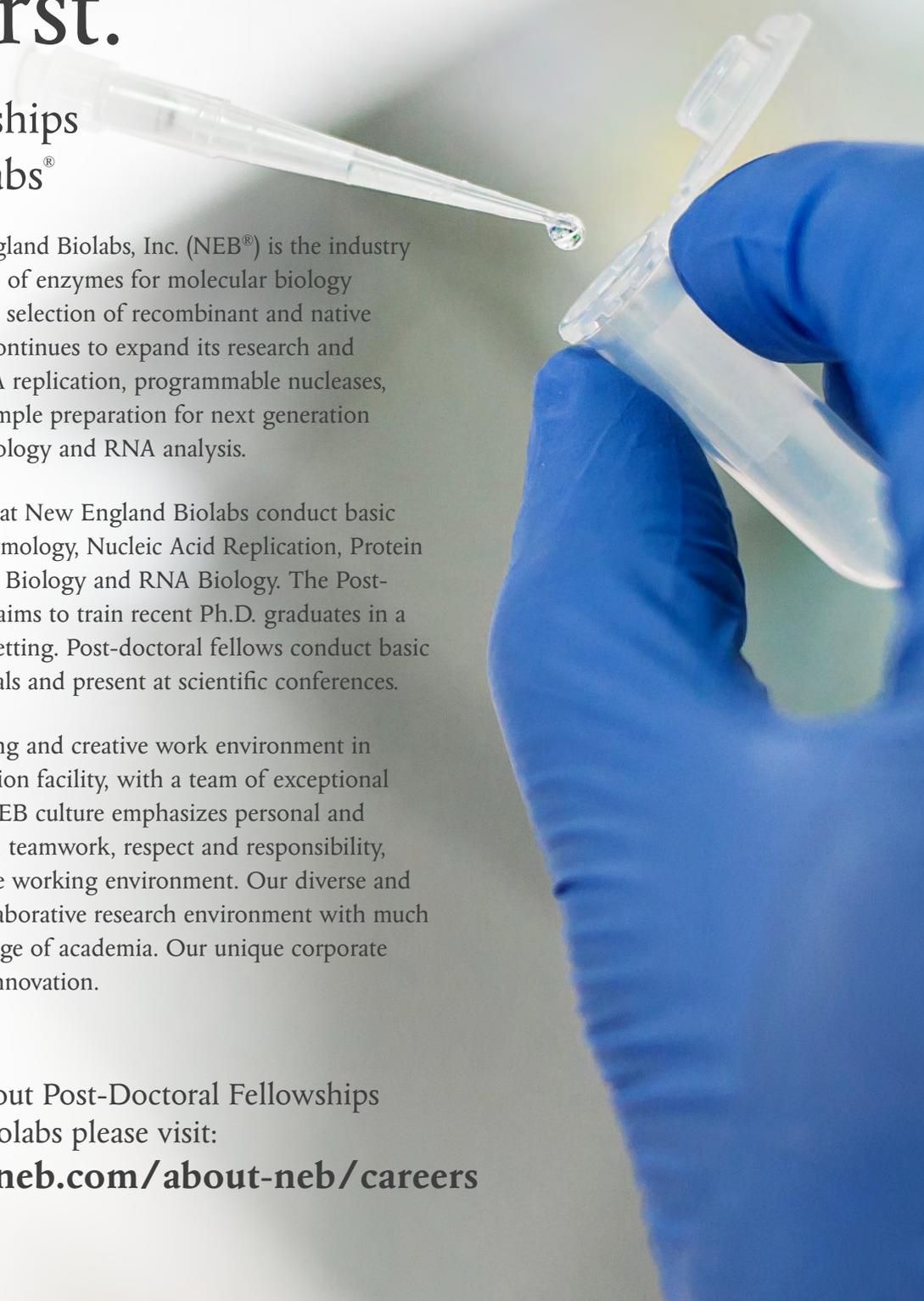
Scientists in the Research Department at New England Biolabs conduct basic research in the areas of Molecular Enzymology, Nucleic Acid Replication, Protein Expression and Modification, Genome Biology and RNA Biology. The Post-Doctoral Fellowship program at NEB aims to train recent Ph.D. graduates in a modern industrial molecular biology setting. Post-doctoral fellows conduct basic research, publish in high quality journals and present at scientific conferences.

Employment at NEB offers a stimulating and creative work environment in a state-of-the-art research and production facility, with a team of exceptional scientists and professional staff. The NEB culture emphasizes personal and professional growth through creativity, teamwork, respect and responsibility, while maintaining a casual campus-like working environment. Our diverse and talented team of scientists enjoy a collaborative research environment with much of the freedom and intellectual challenge of academia. Our unique corporate philosophy encourages dialogue and innovation.



For information about Post-Doctoral Fellowships at New England Biolabs please visit:

<https://www.neb.com/about-neb/careers>





We would like to acknowledge the generous support of
our 2025 ASP Annual Conference Sponsors

