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GOLD COAST 2026

Program & Abstracts

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2026 Annual Conference of the Australian Society for Parasitology Inc.

29 June – 2 July, 2026 Mantra on View, Surfers Paradise, Gold Coast Australia

Contents

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Welcome from the ASP President

Dear Colleague,

On behalf of the Australian Society for Parasitology (ASP) Council and the 2026 Conference Organising Committee, we extend a warm welcome to the 2026 ASP Annual Conference. The Conference will take place at Mantra on View Hotel, starting with the Welcome Reception on Monday June 29, from 6:00 – 8:00pm. The scientific program will run across three full days from 9:00am, Tuesday June 30. The 2026 ASP AGM will take place on Wednesday July 1, 2026 and the Conference will conclude with dinner at Sea World, Gold Coast, Pre- Dinner at Dolphin Beach (featuring the enchanting dolphin encounters) followed by Dinner in the Plaza with a DJ on the evening of Thursday July 2, 2026 from 6:30 – 10:00pm.

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 and One Health. The program covers all aspects of parasitology research and that includes basic research in all areas of life science. This year, just prior to the 2026 ASP Conference, on Monday 29, 2026, 9am-3:30pm Strongyloides Australia will run a one-day workshop and there will also be an Oral Parasite Workshop. We also pay tribute to three parasitologists who have sadly passed away during the Conference. There will be a Tribute to Dame Bridget Ogilvie AC delivered by Prof Karen Day, University of Melbourne; a Nick White Tribute Symposium on the first day of the conference, tribute delivered by Prof Colin Sutherland, LSHTM and a Tribute to Bob Sinden in the Cells, Molecules and Genes session, the tribute will be delivered by Prof Jake Baum, UNSW. On the first full day of the 2026 ASP Annual Conference, where we start with a breakfast event specifically for our research students and early career researchers. The theme for this Student & ECR workshop is “Choose your own adventure - career planning for parasitologists” with an interactive panel discussion about career paths and the choices and decisions we make that affect our careers with scientists who represent a range of career stages and experiences; Dr Catherine Gordon, Dr Liisa Ahlstrom, Christopher Dean Goodman, Dr. habil. Kristina Lehnert, Professor Jonathn Marchant and Dr Storm Martin.

We would like to acknowledge the generous support of our 2026 ASP Annual Conference sponsors, thanks to Elsevier and the International Journal for Parasitology (IJP), IJP DDR and IJP PAW, Elanco, QIMR Berghofer Medical Research Institute, Centre for Tropical Health & Emerging Diseases, Institute for Biomedicine and Glycomics, Griffith University, Southern Cross Diagnostics, New England Biolabs and VectorBuilder.

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

Professor Aaron Jex
President, ASP

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Program Overview

Date: Monday, 29/June/2026	
9:00am - 4:00pm	**OP: Oral Parasites Workshop**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Simone Sleep**, AICIM
9:00am - 4:00pm	**SW: Strongyloides Workshop**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Catherine Gordon**, QIMR Berghofer
2:00pm - 5:00pm	
Tea breaks, Registration and Sponsor space	**Registration: Registration** Location: **Tea breaks, Registration and Sponsor space**
6:00pm - 8:00pm	**Welcome: Welcome Reception**
Lobby & Restaurant	Location: **Lobby & Restaurant**
Date: Tuesday, 30/June/2026	
7:15am - 8:45am	**ECRBreakfast: Early Career Researcher breakfast event**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Jacinta Macdonald**, Griffith University
9:00am - 9:45am	**WelcometoCountry: Welcome to Country Cultural event and Introduction**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University Session Chair: **Swaid Abdullah**, The University of Queensland
9:45am - 10:30am	**BMM: The Bancroft-Mackerras Medal for Excellence Award and Oration**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Aaron Jex**, WEHI
10:30am - 11:00am	
Tea breaks, Registration and Sponsor space	**Morning Tea Break Tuesday** Location: **Tea breaks, Registration and Sponsor space**
11:00am - 11:05am	**NWT: Tribute to Nick White delivered by Professor Colin Sutherland, LSHTM**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Katherine Andrews**, Griffith University
11:00am - 11:20am	**S3: Tropical Health Symposium sponsored by QIMR Berghofer, Centre for Tropical Health & Emerging Diseases**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Darren Gray**, QIMR Berghofer Session Chair: **Chika Zumuk**, Queensland Institute of Medical Research
11:00am - 11:30am	**S2: Canines Symposium sponsored by Elanco**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Swaid Abdullah**, The University of Queensland Session Chair: **Liisa Ahlstrom**, Elanco
11:05am - 11:45am	**S1: Nick White Memorial Symposium**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Katherine Andrews**, Griffith University Session Chair: **Colin Sutherland**, LSHTM
11:20am - 11:35am	**CP3: Tropical Health 15 min talk sponsored by QIMR Berghofer, Centre for Tropical Health & Emerging Diseases**
Lecture Theatre 3	Location: **Lecture Theatre 3**

	Session Chair: **Darren Gray**, QIMR Berghofer Session Chair: **Chika Zumuk**, Queensland Institute of Medical Research
11:30am - 11:45am	**CP2: Canines 15 minute talk sponsored by Elanco**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Swaid Abdullah**, The University of Queensland Session Chair: **Liisa Ahlstrom**, Elanco
11:35am - 11:55am	**CP3.1: Tropical Health 10 min talks sponsored by QIMR Berghofer, Centre for Tropical Health & Emerging Diseases**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Darren Gray**, QIMR Berghofer Session Chair: **Chika Zumuk**, Queensland Institute of Medical Research
11:45am - 12:05pm	**CP1: Nick White Memorial 10 min talks**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Katherine Andrews**, Griffith University Session Chair: **Colin Sutherland**, LSHTM
11:45am - 12:15pm	**CP2.1: Canines 10 minute talks sponsored by Elanco**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Swaid Abdullah**, The University of Queensland Session Chair: **Liisa Ahlstrom**, Elanco
11:55am - 12:15pm	**CP3.2: Tropical Health 5 min talks sponsored by QIMR Berghofer, Centre for Tropical Health & Emerging Diseases**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Darren Gray**, QIMR Berghofer Session Chair: **Chika Zumuk**, Queensland Institute of Medical Research
12:05pm - 12:15pm	**CP1.1: Nick White Memorial 5 min talks**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Katherine Andrews**, Griffith University Session Chair: **Colin Sutherland**, LSHTM
12:15pm - 12:30pm	**S1Q: Questions and Discussion Nick White Memorial Presentations**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Katherine Andrews**, Griffith University Session Chair: **Colin Sutherland**, LSHTM
12:15pm - 12:30pm	**S2Q: Questions & Discussion for Canines Symposium sponsored by Elanco**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Swaid Abdullah**, The University of Queensland Session Chair: **Liisa Ahlstrom**, Elanco
12:15pm - 12:30pm	**S3Q: Questions & Discussion: Tropical Health sponsored by QIMR Berghofer, Centre for Tropical Health & Emerging Diseases**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Darren Gray**, QIMR Berghofer Session Chair: **Chika Zumuk**, Queensland Institute of Medical Research
12:30pm - 1:30pm	**Lunch Tuesday**
Lunch Area	Location: **Lunch Area**
1:30pm - 1:35pm	**TBS: Tribute to Bob Sinden delivered by Professor Jake Baum, UNSW**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Alicja (Ala) Tabor**, The University Of Queensland Session Chair: **Alexander Gofton**, CSIRO This session will begin with a 5 minute Tribute to Bob Sinden, given by Jake Baum, UNSW
1:30pm - 1:45pm	**CP6: Wildlife 1: Fish, Snakes & Turtles 15 min talk**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Nathan Bott**, RMIT University Session Chair: **Amanda Ash**, Murdoch University
1:30pm - 2:00pm	**CP5: Immunology 1 - 15 min talks**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University Session Chair: **Hannah Siddle**, The University of Queensland
1:35pm - 1:50pm	**CP4: Cells, Molecules and Genes – Tribute to Bob Sinden - 15 min talk**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Alicja (Ala) Tabor**, The University Of Queensland Session Chair: **Alexander Gofton**, CSIRO This session will begin with a 5 minute Tribute to Bob Sinden, given by Jake Baum, UNSW
1:45pm - 2:25pm	**CP6.1: Wildlife 1: Fish, Snakes & Turtles 10 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Nathan Bott**, RMIT University Session Chair: **Amanda Ash**, Murdoch University
1:50pm - 2:40pm	**CP4.1: Cells, Molecules and Genes – Tribute to Bob Sinden - 10 min talks**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Alicja (Ala) Tabor**, The University Of Queensland Session Chair: **Alexander Gofton**, CSIRO

2:00pm - 2:30pm	**CP5.1: Immunology 1 - 10 min talks**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University Session Chair: **Hannah Siddle**, The University of Queensland
2:25pm - 2:45pm	**CP6.2: Wildlife 1: Fish, Snakes & Turtles 5 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Nathan Bott**, RMIT University Session Chair: **Amanda Ash**, Murdoch University
2:30pm - 2:45pm	**CP5.2: Immunology 1 - 5 min talks**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University Session Chair: **Hannah Siddle**, The University of Queensland
2:40pm - 2:50pm	**CP4.2: Cells, Molecules and Genes – Tribute to Bob Sinden - 5 min talks**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Alicja (Ala) Tabor**, The University Of Queensland Session Chair: **Alexander Gofton**, CSIRO
2:45pm - 3:00pm	**CP5Q: Questions & Discussion Immunology 1**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University Session Chair: **Hannah Siddle**, The University of Queensland
2:45pm - 3:00pm	**CP6Q: Questions & Discussion Wildlife 1: Fish, Snakes & Turtles**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Nathan Bott**, RMIT University Session Chair: **Amanda Ash**, Murdoch University
2:50pm - 3:00pm	**CP4Q: Questions & Discussion Cells, Cells, Molecules and Genes – Tribute to Bob Sinden**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Alicja (Ala) Tabor**, The University Of Queensland Session Chair: **Alexander Gofton**, CSIRO
3:00pm - 3:30pm	**Afternoon Tea Break Tuesday**
Tea breaks, Registration and Sponsor space	Location: **Tea breaks, Registration and Sponsor space**
3:30pm - 4:15pm	**P1: Elsevier Plenary Lecture Series International Journal for Parasitology: Parasites and Wildlife (IJP:PAW) Invited Lecturer**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Andrew Thompson**, Murdoch University
4:15pm - 5:00pm	**P2: Elsevier Plenary Lecture Series International Journal for Parasitology: Drugs and Drug Resistance (IJP:DDR) Invited Lecturer**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Sarah Preston**, Federation University Australia
Date: Wednesday, 01/July/2026	
9:00am - 9:10am	**Tribute: Tribute to Dame Bridget Ogilvie AC delivered by Professor Karen Day, The University of Melbourne**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Aaron Jex**, WEHI Professor Karen Day, The University of Melbourne, will deliver the Tribute to Dame Bridget Ogilvie AC.
9:10am - 9:45am	**BOM: Bridget Ogilvie Medal Award and Oration**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Aaron Jex**, WEHI
9:45am - 10:30am	**CP7: Education & Outreach - 15 min talks**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Michelle Power**, Macquarie University
10:30am - 11:00am	**Morning Tea Break Wednesday**
Tea breaks, Registration and Sponsor space	Location: **Tea breaks, Registration and Sponsor space**
11:00am - 11:15am	**CP10: Wildlife 2: Mammals, Birds, Lizards & Wetas 15 min talk**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Haylee Crawford-Weaver**, DCCEEW Session Chair: **Nicholas Fountain-Jones**, University of Tasmania
11:00am - 11:20am	**S4: Vaccines Symposium sponsored by Institute for Biomedicine and Glycomics, Griffith University**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University Session Chair: **Anouschka Akerman**, The University of Queensland

11:00am - 11:50am	**CP8: Drugs & Drug Resistance 1 - 10 min talks**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Christopher Hart**, Griffith University Session Chair: **Hannah Smith**, Griffith University
11:15am - 11:45am	**CP10.1: Wildlife 2: Mammals, Birds, Lizards & Wetas 10 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Haylee Crawford-Weaver**, DCCEEW Session Chair: **Nicholas Fountain-Jones**, University of Tasmania
11:20am - 11:35am	**CP9: Vaccines 15 min talk sponsored by Institute for Biomedicine and Glycomics, Griffith University**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University Session Chair: **Anouschka Akerman**, The University of Queensland
11:35am - 12:05pm	**CP9.1: Vaccines 10 min talks sponsored by Institute for Biomedicine and Glycomics, Griffith University**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University Session Chair: **Anouschka Akerman**, The University of Queensland
11:45am - 12:15pm	**CP10.2: Wildlife 2: Mammals, Birds, Lizards & Wetas 5 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Haylee Crawford-Weaver**, DCCEEW Session Chair: **Nicholas Fountain-Jones**, University of Tasmania
11:50am - 12:15pm	**CP8.1: Drugs and Drug Resistance 1 - 5 min talks**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Christopher Hart**, Griffith University Session Chair: **Hannah Smith**, Griffith University
12:05pm - 12:15pm	**CP9.2: Vaccines 5 min talks sponsored by Institute for Biomedicine and Glycomics, Griffith University**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University Session Chair: **Anouschka Akerman**, The University of Queensland
12:15pm - 12:30pm	**CP8Q: Questions & Discussion Drugs and Drug Resistance 1**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Christopher Hart**, Griffith University Session Chair: **Hannah Smith**, Griffith University
12:15pm - 12:30pm	**CP9Q: Questions & Discussion Vaccines sponsored by Institute for Biomedicine and Glycomics, Griffith University**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University Session Chair: **Anouschka Akerman**, The University of Queensland
12:15pm - 12:30pm	**CP10Q: Questions & Discussion Wildlife 2: Mammals, Birds, Lizards & Wetas**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Haylee Crawford-Weaver**, DCCEEW Session Chair: **Nicholas Fountain-Jones**, University of Tasmania
12:30pm - 1:30pm	**Lunch Wednesday**
Lunch Area	Location: **Lunch Area**
1:30pm - 1:50pm	**S5: Symposium Ticks, Mites, kissing bugs**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Katja Fischer**, QIMR Berghofer Session Chair: **Xavier Barton**, Murdoch University
1:30pm - 2:30pm	**CP11: Cells, Molecules & Genes 2 - 10 min talks**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Ellis Joch**, Griffith University Session Chair: **Wisam Dawood**, Griffith University
1:30pm - 2:30pm	**CP12: Sheep & Goats - 10 min talks**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Vern Bowles**, The University of Melbourne Session Chair: **Nichola Calvani**, The University of Sydney
1:50pm - 2:30pm	**CP13: Ticks, Mites, kissing bugs 10 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Katja Fischer**, QIMR Berghofer Session Chair: **Xavier Barton**, Murdoch University
2:30pm - 2:45pm	**CP11.1: Cells, Molecules & Genes 2 - 5 min talks**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Ellis Joch**, Griffith University Session Chair: **Wisam Dawood**, Griffith University
2:30pm - 2:45pm	**CP12.1: Sheep & Goats - 5 min talks**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Vern Bowles**, The University of Melbourne Session Chair: **Nichola Calvani**, The University of Sydney

2:30pm - 2:45pm	**CP13.1: Ticks, Mites, kissing bugs 5 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Katja Fischer**, QIMR Berghofer Session Chair: **Xavier Barton**, Murdoch University
2:45pm - 3:00pm	**CP11Q: Questions & Discussion Cells, Molecules & Genes 2**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Ellis Joch**, Griffith University Session Chair: **Wisam Dawood**, Griffith University
2:45pm - 3:00pm	**CP12Q: Questions & Discussion Sheep and Goats**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Vern Bowles**, The University of Melbourne Session Chair: **Nichola Calvani**, The University of Sydney
2:45pm - 3:00pm	**S5Q: Questions & Discussion Ticks, Mites, kissing bugs**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Katja Fischer**, QIMR Berghofer Session Chair: **Xavier Barton**, Murdoch University
3:00pm - 3:30pm	**Afternoon Tea Break Wednesday**
Tea breaks, Registration and Sponsor space	Location: **Tea breaks, Registration and Sponsor space**
3:30pm - 4:00pm	**Sprent: Sprent Award and Oration**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Aaron Jex**, WEHI
4:00pm - 6:15pm	**AGM: 2026 ASP Annual General Meeting**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Aaron Jex**, WEHI Session Chair: **Jake Baum**, UNSW Sydney
6:30pm - 9:00pm	**Student social event: ECR Student social event**
Date: Thursday, 02/July/2026	
9:00am - 9:45am	**P3: Elsevier Plenary Lecture Series International Journal for Parasitology (IJP) Invited Lecturer**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Brian Cooke**, James Cook University
9:45am - 10:30am	**CP14: Top Rated Contributed Abstracts 15 min talks**
Plenary Lecture Theatre	Location: **Plenary Lecture Theatre** Session Chair: **Danielle Stanisic**, Institute for Biomedicine and Glycomics, Griffith University
10:30am - 11:00am	**Morning Tea Break Thursday**
Tea breaks, Registration and Sponsor space	Location: **Tea breaks, Registration and Sponsor space**
11:00am - 11:15am	**CP17: Epidemiology & Diagnostics 15 min talk**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Deepani Fernando**, QIMR Berghofer Session Chair: **Luke Hall**, St Vincent's Hospital Sydney
11:00am - 11:20am	**S6: Drugs & Drug Resistance Symposium sponsored by Institute for Biomedicine and Glycomics, Griffith University**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Jacinta Macdonald**, Griffith University Session Chair: **Rohith Kutty**, Griffith University
11:00am - 11:20am	**S7: Horses & Cows 1 Symposium**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Abdul Jabbar**, The University of Melbourne Session Chair: **Narelle Dybing**, Murdoch University
11:15am - 12:05pm	**CP17.1: Epidemiology & Diagnostics 10 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Deepani Fernando**, QIMR Berghofer Session Chair: **Luke Hall**, St Vincent's Hospital Sydney
11:20am - 11:50am	**CP15: Drugs & Drug Resistance 2 - 10 min talks sponsored by Institute for Biomedicine and Glycomics, Griffith University**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Jacinta Macdonald**, Griffith University Session Chair: **Rohith Kutty**, Griffith University
11:20am - 11:50am	**CP16: Horses & Cows - 10 min talks**
Lecture Theatre 2	Location: **Lecture Theatre 2**

	Session Chair: **Abdul Jabbar**, The University of Melbourne Session Chair: **Narelle Dybing**, Murdoch University
11:50am - 12:05pm	**CP16.1: Horses & Cows - 5 min talks**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Abdul Jabbar**, The University of Melbourne Session Chair: **Narelle Dybing**, Murdoch University
11:50am - 12:15pm	**CP15.1: Drugs & Drug Resistance 2 - 5 min talks sponsored by Institute for Biomedicine and Glycomics, Griffith University**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Jacinta Macdonald**, Griffith University Session Chair: **Rohith Kutty**, Griffith University
12:05pm - 12:15pm	**CP17.2: Epidemiology & Diagnostics 5 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Deepani Fernando**, QIMR Berghofer Session Chair: **Luke Hall**, St Vincent's Hospital Sydney
12:05pm - 12:30pm	**S7Q: Questions & Discussion Horses & Cows 1**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Abdul Jabbar**, The University of Melbourne Session Chair: **Narelle Dybing**, Murdoch University
12:15pm - 12:30pm	**S6Q: Questions and Discussion Drugs & Drug Resistance Symposium sponsored by Institute for Biomedicine and Glycomics, Griffith University**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Jacinta Macdonald**, Griffith University Session Chair: **Rohith Kutty**, Griffith University
12:15pm - 12:30pm	**CP17Q: Questions and Discussion Epidemiology & Diagnostics**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Deepani Fernando**, QIMR Berghofer Session Chair: **Luke Hall**, St Vincent's Hospital Sydney
12:30pm - 1:30pm	**Lunch Thursday**
Lunch Area	Location: **Lunch Area**
1:30pm - 2:00pm	**CP20: Zoonoses & One Health 15 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Darren Gray**, QIMR Berghofer Session Chair: **Jessica Scott**, James Cook University
1:30pm - 2:15pm	**CP19: Immunology 2 - 15 min talks**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Alicja (Ala) Tabor**, The University Of Queensland Session Chair: **Hannah Siddle**, The University of Queensland
1:30pm - 2:40pm	**CP18: Cells, Molecules & Genes 3 - 10 min talks**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Shilpa Kapoor**, The University of Melbourne Session Chair: **Balu Balan**, Walter and Eliza Hall Institute
2:00pm - 2:30pm	**CP20.1: Zoonoses & One Health 1 - 10 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Darren Gray**, QIMR Berghofer Session Chair: **Jessica Scott**, James Cook University
2:15pm - 2:45pm	**CP19.1: Immunology 2 - 10 min talks**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Alicja (Ala) Tabor**, The University Of Queensland Session Chair: **Hannah Siddle**, The University of Queensland
2:30pm - 2:45pm	**CP20.2: Zoonoses & One Health 5 min talks**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Darren Gray**, QIMR Berghofer Session Chair: **Jessica Scott**, James Cook University
2:40pm - 2:45pm	**CP18.1: Cells, Molecules & Genes 3 - 5 min talks**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Shilpa Kapoor**, The University of Melbourne Session Chair: **Balu Balan**, Walter and Eliza Hall Institute
2:45pm - 3:00pm	**CP18Q: Questions and Discussion Cells, Molecules & Genes 3**
Lecture Theatre 1	Location: **Lecture Theatre 1** Session Chair: **Shilpa Kapoor**, The University of Melbourne Session Chair: **Balu Balan**, Walter and Eliza Hall Institute
2:45pm - 3:00pm	**CP19Q: Questions and Discussion Immunology 2**
Lecture Theatre 2	Location: **Lecture Theatre 2** Session Chair: **Alicja (Ala) Tabor**, The University Of Queensland Session Chair: **Hannah Siddle**, The University of Queensland
2:45pm - 3:00pm	**CP20Q: Questions and Discussion Zoonoses & One Health 1**
Lecture Theatre 3	Location: **Lecture Theatre 3** Session Chair: **Darren Gray**, QIMR Berghofer Session Chair: **Jessica Scott**, James Cook University

3:00pm - 3:30pm Tea breaks, Registration and Sponsor space	**Afternoon Tea Break Thursday** Location: **Tea breaks, Registration and Sponsor space**
3:30pm - 3:45pm Lecture Theatre 3	**CP23: Zoonoses & One Health 2 - 15 min talk** Location: **Lecture Theatre 3** Session Chair: **Catherine Gordon**, QIMR Berghofer Session Chair: **Fasil Shiferaw**, QIMR Berghofer
3:30pm - 4:00pm Lecture Theatre 1	**CP21: Cells, Molecules & Genes 4 - 15 min talks** Location: **Lecture Theatre 1** Session Chair: **Andrew Walker**, The University of Queensland Session Chair: **Natasha Sharma**, The University of Melbourne
3:30pm - 4:20pm Lecture Theatre 2	**CP22: Horses & Cows 2 - 10 min talks** Location: **Lecture Theatre 2** Session Chair: **Alicja (Ala) Tabor**, The University Of Queensland Session Chair: **Sara Taylor**, QIMR Berghofer
3:45pm - 4:25pm Lecture Theatre 3	**CP23.1: Zoonoses & One Health 2 - 10 min talks** Location: **Lecture Theatre 3** Session Chair: **Catherine Gordon**, QIMR Berghofer Session Chair: **Fasil Shiferaw**, QIMR Berghofer
4:00pm - 4:20pm Lecture Theatre 1	**CP21.1: Cells, Molecules & Genes 4 - 10 min talks** Location: **Lecture Theatre 1** Session Chair: **Andrew Walker**, The University of Queensland Session Chair: **Natasha Sharma**, The University of Melbourne
4:20pm - 4:30pm Lecture Theatre 1	**CP21.2: Cells, Molecules & Genes 4 - 5 min talks** Location: **Lecture Theatre 1** Session Chair: **Andrew Walker**, The University of Queensland Session Chair: **Natasha Sharma**, The University of Melbourne
4:20pm - 4:30pm Lecture Theatre 2	**CP22Q: Questions and Discussion Horses & Cows 2** Location: **Lecture Theatre 2** Session Chair: **Alicja (Ala) Tabor**, The University Of Queensland Session Chair: **Sara Taylor**, QIMR Berghofer
4:25pm - 4:35pm Lecture Theatre 3	**CP23Q: Questions and Discussion Zoonoses & One Health 2** Location: **Lecture Theatre 3** Session Chair: **Catherine Gordon**, QIMR Berghofer Session Chair: **Fasil Shiferaw**, QIMR Berghofer
4:30pm - 4:40pm Lecture Theatre 1	**CP21Q: Questions and Discussion Cells, Molecules & Genes 4** Location: **Lecture Theatre 1** Session Chair: **Andrew Walker**, The University of Queensland Session Chair: **Natasha Sharma**, The University of Melbourne
4:30pm - 5:45pm	**Free: Free time**
5:45pm - 6:00pm Mantra front entrance	**Meet: Meet in foyer for bus to Conference Dinner at Sea World** Location: **Mantra front entrance**
6:00pm - 6:30pm	**Bus: Bus to Conference Dinner at Sea World**
6:30pm - 10:00pm Sea World	**Dinner: Conference Dinner at Sea World** Location: **Sea World**

2026 Annual Conference of the Australian Society for Parasitology Inc.

29 June – 2 July, 2026 Mantra on View, Surfers Paradise, Gold Coast Australia

Presentations

Tuesday 30 June 2026

9:00am - 9:45am Welcome to Country Cultural event and Introduction

Location: Plenary Lecture Theatre

Session Chair: Danielle Stanisic, Institute for Biomedicine and Glycomics, Griffith University

Session Chair: Swaid Abdullah, The University of Queensland

Plenary Lecture Theatre

9:45am - 10:30am BMM: The Bancroft-Mackerras Medal for Excellence Award and Oration

Location: Plenary Lecture Theatre

Session Chair: Aaron Jex, WEHI

Plenary Lecture Theatre

S1: Nick White Memorial Symposium

Tribute to Nick White delivered by Colin Sutherland, LSHTM

Time: Tuesday, 30/June/2026: 11:00am - 11:45am · *Location:* Lecture Theatre 1
Session Chair: Katherine Andrews, Griffith University
Session Chair: Colin Sutherland, LSHTM

ID: 108 / **S1: 1**
Invited speaker abstract

Dimorphic apicoplast and mitochondrial genomes support full species status for the two causative agents of ovale malaria in humans

Colin Sutherland[1], Jan Šlapeta[2,3], Taane Clark[1], Susana Campino[1], Hans-Peter Fuehrer[4]
[1]LSHTM, United Kingdom; [2]Sydney School of Veterinary Science, Faculty of Science, The University of Sydney, NSW; [3]Sydney Institute for Infectious Diseases, The University of Sydney, Sydney, NSW, Australia; [4]Parasitology, Department of Biological Sciences and Pathobiology, University of Veterinary Medicine Vienna, Austria

Recently published whole-genome analyses of the two closely related parasites, *Plasmodium ovale curtisi* and *P. ovale wallikeri*, which cause human ovale malaria, provide compelling evidence that the nuclear genomes of these two organisms do not recombine and are therefore perfectly dimorphic at all loci examined. The same pattern is observed when comparing the two 4.3 kb mitochondrial genomes. Here, we present an analysis of new sequencing data from the *tufa* locus, encoded in the plastid-derived apicoplast organelle, that provides evidence that this third parasite genome is also dimorphic and co-segregates with specific dimorphs of the nuclear and mitochondrial genomes. These findings, together with other recent studies, support full species status for the two causative agents of ovale malaria in humans, necessitating a revision of the nomenclature used up until now. We propose redefining the original species name *Plasmodium ovale* Stephens by designating a neotype from Kenya for what was previously referred to as 'P. ovale curtisi'. A new species, *Plasmodium wallikeri* sp. n., is also described using a type specimen from West Africa. Morphological descriptions will be provided and sequence information defined for three genetic loci that distinguish these two species at nuclear, mitochondrial and apicoplast genome levels, respectively.

ID: 274 / **S1: 2**
Invited speaker abstract

Investigating resistance to the malaria drug proguanil

Katherine Andrews[1], Patrick Tumwebaze[1], Gillian M. Fisher[1], Yunan Qian[1], Wisam Dawood[1], Jacinta Macdonald[1], David A. Fidock[2], Oriana Kreutzfeld[3], Philip Rosenthal[3], Andrew G. Riches[4], John H. Ryan[4], Tina S. Skinner-Adams[1]
[1]Institute for Biomedicine and Glycomics, Griffith University, Nathan, Queensland, Australia; [2]Department of Microbiology and Immunology, Columbia University Irving Medical Center, New York, USA; [3]Department of Medicine, University of California San Francisco, California, USA; [4]Commonwealth Scientific and Industrial Research Organization, Biomedical Manufacturing, Clayton, Victoria, Australia

The combination of atovaquone and proguanil (e.g., Malarone®) has been used for decades for malaria prevention and treatment. Atovaquone inhibits cytochrome bc_1 (complex III), a component of the *Plasmodium* mitochondrial electron transport chain (mETC). Proguanil is a biguanide prodrug that is metabolized *in vivo* by liver cytochrome P450 (CYP2C19) enzymes into cycloguanil, a dihydrofolate reductase (DHFR) inhibitor that blocks the synthesis of pyrimidines which are required for nucleic acid synthesis. Proguanil can potentiate the activity of atovaquone *in vitro*, and we demonstrated that this drug also has slow action *in vitro* activity against *P. falciparum* (e.g., *Pf*3D7 96h IC_{50} 0.1 µM) that is independent of DHFR inhibition and isoprenoid metabolism and does not appear to be directly linked to pyrimidine synthesis. However, our understanding of the clinical implications of proguanil's intrinsic activity are complicated by an incomplete understanding of the slow action mechanism of this drug and the lack of information on clinical resistance to proguanil. To address this, we have utilised a range of approaches to investigate resistance mechanisms associated with proguanil, including generation of proguanil-resistant *P. falciparum* lines and examining differences in sensitivity to proguanil by *P. falciparum* lab lines, field isolates and the zoonotic *P. cynomolgi* species. These data will be discussed in the context of clinical use of proguanil in the atovaquone and proguanil combination.

S2: Canines Symposium sponsored by Elanco

Time: Tuesday, 30/June/2026: 11:00am - 11:30am · *Location:* Lecture Theatre 2
Session Chair: Swaid Abdullah, The University of Queensland
Session Chair: Liisa Ahlstrom, Elanco

ID: 263 / **S2: 1**
Invited speaker abstract

Speed-of-kill comparison of isoxazolines in the combination endectocide products Credelio™ PLUS (lotilaner), NexGard Spectra® (afoxolaner) and Simparica® Trio (sarolaner) against the Australian paralysis tick (*Ixodes holocyclus*) throughout one month

Liisa Ahlstrom[1], Rachel Lyons[1], Claudia Ellenberger[1], Kim Baker[1], Melissa Pittorino[1], Scott Wiseman[2], Bettina Schunack[3]

[1]Elanco Animal Health, Australia; [2]Elanco Animal Health, United Kingdom; [3]Elanco Animal Health GmbH, Germany

Paralysis ticks (*Ixodes holocyclus*) cause a severe, potentially fatal, toxicosis in dogs. A fast and sustained speed-of-kill throughout the dosing interval is a valuable acaricidal characteristic. Lotilaner has the longest half-life (35 days) of the oral isoxazolines[1], and a single dose kills paralysis ticks for over 11 weeks.[2]

A randomised, blinded, controlled study was conducted to compare the speed-of-kill of the monthly-dosed combination isoxazoline products Credelio™ PLUS (lotilaner, milbemycin oxime), NexGard Spectra® (afoxolaner, milbemycin oxime) and Simparica® Trio (sarolaner, moxidectin, pyrantel) against *Ixodes holocyclus.* Dogs (n=7/group) were treated (or left as untreated controls) on Day 0 and infested with 10-12 unfed adult female ticks on Days -2, 21, 28 and 35. Tick counts were performed 12, 18 and 24 hours post-treatment and post-reinfestation.

The initial speed-of-kill efficacy (12 h post-treatment) was rapid and similar for lotilaner and sarolaner (>80%), and significantly greater than afoxolaner (50%; $P<0.004$). On Day 21, efficacies of lotilaner, sarolaner and afoxolaner were 95.6%, 66.1% and 26.0%, respectively, 12 hours after reinfestation, with lotilaner and afoxolaner exceeding 97% and sarolaner nearly reaching 95% by 24 hours. On Day 28, efficacies of lotilaner, sarolaner and afoxolaner were 86.4%, 21.6% and 3.6%, respectively, 12 hours after reinfestation, with all products exceeding 96% and lotilaner reaching 100% by 24 hours. On Day 35, efficacies of lotilaner, sarolaner and afoxolaner were 76.6%, 23.4% and 6.9%, respectively, 12 hours after reinfestation. Lotilaner exceeded 95% efficacy already by 18 hours, sarolaner by 24 hours, while afoxolaner remained below 90%.

Lotilaner killed reinfesting ticks faster ($P<0.01$ at 12 hours) than sarolaner and afoxolaner and sustained its rapid speed-of-kill and efficacy beyond the label claim of one month. This offers reassurance to veterinarians and dog owners, addressing concerns about the potential increased risk of tick paralysis towards the end of the dosing interval of acaricides.

References:

[1] Toutain CE., et al. The intravenous and oral pharmacokinetics of lotilaner in dogs. *Parasit Vectors*. 2017;10(1):522.

[2] Baker, K., et al. Laboratory evaluations of the 3-month efficacy of oral lotilaner (Credelio™) against experimental infestations of dogs with the Australian paralysis tick, *Ixodes holocyclus*. *Parasit Vectors*. 2018;11(1):487.

S3: Tropical Health Symposium sponsored by QIMR Berghofer, Centre for Tropical Health & Emerging Diseases

Time: Tuesday, 30/June/2026: 11:00am - 11:20am · *Location:* Lecture Theatre 3
Session Chair: Darren Gray, QIMR Berghofer
Session Chair: Chika Zumuk, Queensland Institute of Medical Research

ID: 276 / **S3: 1**
Invited speaker abstract

One health approach to elimination of Schistosoma mekongi in Cambodia and Lao PDR: results from a pilot study

Catherine Gordon[1,2], Darren J Gray[2,3], Marcello Sato[4], Megumi Sato[5], Yasuhito Sako[6], Virak Khieu[7], Somphou Sayasone[8], Poom Adisakwattana[9], Kinley Wangdi[10]

[1]QIMR Berghofer, Infection & Inflammation department, Herston, QLD, Australia; [2]Center for Tropical Health and Emerging Diseases; [3]QIMR Berghofer, Population Health department, Herston, QLD, Australia; [4]Niigata University of Pharmacy and Medical and Life Sciences, Niigata, Japan; [5]Niigata University, Niigata, Japan; [6]Asahikawa Medical University, Asahikawa, Japan; [7]National Centre for Parasitology, Entomology and Malaria Control, Phnom Penh, Cambodia; [8]Lao Tropical and Public Health Institute, Vientiane, Lao PDR; [9]Mahidol University, Department of Helminthology, Bangkok, Thailand; [10]University of Canberra, Canberra, ACT, Australia

Background: *Schistosoma mekongi* remains a public health concern in Cambodia and Lao PDR despite substantial reductions in prevalence following repeated mass drug administration (MDA). As transmission becomes increasingly localised, sensitive and integrated surveillance approaches are needed to identify residual transmission, animal reservoirs, environmental risk, and behavioural factors that may sustain infection. We conducted a pilot One Health study to inform future schistosomiasis elimination activities by combining household surveys, latrine audits, animal sampling, molecular diagnostics, environmental DNA surveillance, and formative research to guide development of health education.

Methods: A mixed-methods cross-sectional pilot study was conducted in six historically endemic villages in Cambodia and two villages in Lao PDR. Household heads completed structured questionnaires on water use, sanitation, knowledge of *S. mekongi*, water contact and open defecation. Latrine audits assessed infrastructure, functionality, water availability and soap presence. In selected villages, faecal samples were collected from domestic animals and preserved for microscopy and qPCR. Environmental DNA samples were collected to support identification of potential transmission hotspots. Formative research, including draw-and-write activities, focus group discussions and key informant interviews, was undertaken with school children, parents, teachers and health officials. These data were used to identify knowledge gaps, misconceptions, preferred communication styles and culturally relevant storylines to support development of the Magic Glasses (MG) health education package for schistosomiasis.

Results: River water use remained common, particularly for cooking, washing and latrine water, and recent river contact was frequently reported across study villages in both countries. Latrine access varied between Cambodian villages, with some communities reporting substantial proportions of households without latrines; overall latrine coverage was higher in Lao PDR. Knowledge of *S. mekongi* was generally high, particularly regarding serious disease, but important gaps remained around transmission, animal reservoirs and the potential for reinfection after treatment. Open defecation and frequent river contact persisted in several villages. Participants strongly supported school-based education and interactive learning activities, informing development of the MG.

Conclusions: This pilot demonstrates the feasibility and value of integrated One Health surveillance for *S. mekongi*. Combining molecular, environmental, animal and community-based approaches strengthens detection while generating locally grounded formative data for health education.

CP3: Tropical Health 15 min talk sponsored by QIMR Berghofer, Centre for Tropical Health & Emerging Diseases

Time: Tuesday, 30/June/2026: 11:20am - 11:35am · *Location:* Lecture Theatre 3
Session Chair: Darren Gray, QIMR Berghofer
Session Chair: Chika Zumuk, Queensland Institute of Medical Research

ID: 121 / **CP3: 1**
Contributed abstract
Conference Topics: Epidemiology
Keywords: River blindness, Prevalence, Elimination, Spatio-temporal, Agro-ecological zones

Spatio-temporal modelling of onchocerciasis prevalence in Ghana: Identifying transmission hotspots for targeted elimination

Millicent Opoku[1,2,3,4], David Dean[3], Himal Shrestha[5], Kwadwo K. Frempong[4], Dziedzom K. de Souza[4], Sellase Pi-Bansa[4], Joseph H. N. Osei[6], Joseph Opare[7], Odame Asiedu[7], Ernest Mensah[7], Warwick N. Grant[2], Daniel A. Boakye[4,8], Shannon Hedtke[1,2]

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Onchocerciasis (river blindness), caused by *Onchocerca volvulus* and transmitted by *Simulium* blackflies, remains a public health challenge in sub-Saharan Africa. Although prevalence has declined following aerial larviciding under the Onchocerciasis Control Programme (OCP) and African Programme for Onchocerciasis Control (APOC) through community-directed treatment, achieving the World Health Organization elimination target of sustained microfilarial (mf) prevalence below 1% requires identifying areas of persistent transmission. Mf prevalence data from 1,353 surveys across 671 villages were obtained from the Expanded Special Project for Elimination of Neglected Tropical Diseases and the Ghana Neglected Tropical Diseases Programme. Environmental predictors were structured around four transmission constructs: climatic suitability, flowing-water probability, vector blood-feeding probability, and bioregional land-use context. A Bayesian spatiotemporal zero-inflated beta-binomial model was fitted using Stan via *brms* in R, incorporating ecozone-specific temporal smooths, a two-dimensional spatial smooth, and nested random effects. MF prevalence was spatially and temporally heterogeneous. Northern ecozones declined to near-elimination levels by 2015, whereas southern forest and forest-agriculture transition zones showed slower and less consistent declines. The Tano-Ankobra transmission zone emerged as a persistent hotspot. This first comprehensive spatiotemporal analysis of onchocerciasis across Ghana highlights the need for intensified, targeted interventions in southern Ghana to achieve elimination.

CP2: Canines 15 minute talk sponsored by Elanco

Time: Tuesday, 30/June/2026: 11:30am - 11:45am · *Location:* Lecture Theatre 2
Session Chair: Swaid Abdullah, The University of Queensland
Session Chair: Liisa Ahlstrom, Elanco

ID: 258 / **CP2: 1**
Contributed abstract
Conference Topics: Molecular Biology, Parasites of dogs, Protozoa, Veterinary Parasitology
Keywords: Canine leishmaniasis; kDNA qPCR; cross-amplification, misidentification

A widely used qPCR for *Leishmania infantum* is non-specific and hinders detection of globally emerging *Leishmania* species

Thuy Nguyen[1,2], Lucas Huggins[1], Andreia Fernandes Brilhante[3], Andrea Paun[4], Edwin Kniha[5], Smaragda Sotiraki[6], Panagiota Ligda[6], Kanok Preativatanyou[7], Padet Siriyasatien[7], Christopher Fernandez-Prada[8], Tawin Inpankaew[9], Carla Maia[10], Gad Baneth[2], Robin B. Gasser[1], Vito Colella[1]

[1]The University of Melbourne, Australia; [2]The Hebrew University of Jerusalem, Israel; [3]Federal University of Acre, Brazil; [4]National Institute of Allergy and Infectious Diseases, USA; [5]Medical University of Vienna, Austria; [6]Hellenic Agricultural Organization Demeter, Greece; [7]Chulalongkorn University, Thailand; [8]Université de Montréal, Canada; [9]Kasetsart University, Thailand; [10]Universidade NOVA de Lisboa, Portugal

Canine leishmaniasis (CanL) has important implications for both human and animal health. *Leishmania infantum* is the primary causative agent of CanL, with dogs acting as the main reservoir for human visceral leishmaniasis. A qPCR assay originally developed to aid CanL diagnosis has been widely used for the identification of *L. infantum* over the past two decades. However, evidence of cross-amplification of other *Leishmania* species raises concerns regarding species misidentification.

Through a systematic review of publications retrieved from major databases from 2006 to 2026, 177 studies were identified that used this CanL qPCR, of which only 51 (28.8%) applied a confirmatory molecular method.

We further evaluated the analytical and diagnostic performance of this assay using DNA from 15 cultured *Leishmania* species representing four subgenera, as well as vector and clinical samples from dogs and humans across endemic regions in the Old and New Worlds and performed species confirmation by nanopore sequencing targeting the heat shock protein 70 gene. We demonstrated that the assay amplified seven non-*L. infantum* species in cultured samples, and *L. major* and *L. braziliensis* from field samples. These findings demonstrate the potential of the CanL qPCR to obscure global *Leishmania* diversity, underscoring the need for improved diagnostic strategies.

CP3.1: Tropical Health 10 min talks sponsored by QIMR Berghofer, Centre for Tropical Health & Emerging Diseases

Time: Tuesday, 30/June/2026: 11:35am - 11:55am · *Location:* Lecture Theatre 3
Session Chair: Darren Gray, QIMR Berghofer
Session Chair: Chika Zumuk, Queensland Institute of Medical Research

ID: 253 / **CP3.1: 1**
Contributed abstract
Conference Topics: Epidemiology, Helminthology, Microscopy, Strongyloides
Keywords: Soil-transmitted helminths, epidemiology, urban slum, tea garden, Northeastern Bangladesh

Epidemiology of soil-transmitted helminth infections in urban slum and tea garden communities in Northeastern Bangladesh

Mandira Mukutmoni[1], Tilak Chandra Nath[2], Adam Bartlett[1], Susana Vaz Nery[1]
[1]Global Health Program, Kirby Institute, University of New South Wales, Sydney, Australia; [2]Department of Parasitology, Sylhet Agricultural University, Bangladesh

Soil-transmitted helminth (STH) infections remain prevalent in northeastern Bangladesh despite the national school-based deworming programme implemented since 2008. This study reports STH epidemiology in underprivileged communities of Sylhet and Moulvibazar districts.

A total of 772 stool samples were collected from 16 urban slum and tea garden communities between May and October 2025 and analysed using sodium nitrate flotation, Kato-Katz, and Baermann techniques. Descriptive statistics estimated prevalence, adjusted for clustering, with 95% confidence intervals.

Based on sodium-nitrate-flotation, overall STH prevalence was 35.1% (271/772; 95% CI 26.2-45.2), with *Ascaris lumbricoides* predominating at 31.1% (240/772; 21.8-42.2), followed by hookworm (38/772; 4.9%, 2.6-9.1) and *Trichuris trichiura* (34/772; 4.4%, 2.0-9.5). All infections were of light to moderate intensity. STH prevalence ranged from 12.5% to 70.9% across communities, with similar levels in tea garden (57/162; 35.2%) and slum settings (214/610; 35.1%). Using Kato-Katz, overall prevalence was 28.4% (219/772; 20.1-38.4), with *A. lumbricoides* predominant (193/772; 25.0%, 16.5-36.1) followed by *T. trichiura* (26/772; 3.4%, 1.3-8.2) and hookworm (19/772; 2.5%, 1.4-4.4). SNF was more sensitive than Kato-Katz, with concordance analysis planned. *Strongyloides* spp. prevalence by Baermann was lower than expected (0.4%; 3/772; 0.1-1.6).

These findings highlight persistent transmission and community-level variation, supporting community-based control strategies targeted to high-risk areas.

ID: 102 / **CP3.1: 2**
Contributed abstract
Conference Topics: Diagnostics, Molecular Biology
Keywords: Point-of-care, diagnostics, molecular, neglected tropical diseases, schistosomiasis

Development and field-testing of point-of-care diagnostics for schistosomiasis elimination in the Philippines

Emmanuel John Tabilin[1,2], Eleonor Avenido-Cervantes[3], Mary Lorraine Mationg[4], Darren Gray[4], Marianette Inobaya[3], Mario Jiz[4], Hong You[5], Catherine Gordon[1,2], Pengfei Cai[1]
[1]Applied Tropical and Molecular Parasitology, QIMR Berghofer, Australia; [2]Faculty of Medicine, The University of Queensland, Australia; [3]Research Institute of Tropical Medicine, Muntinlupa City, Philippines; [4]Global Health and Tropical Medicine, QIMR Berghofer, Australia; [5]Molecular Helminthology, QIMR Berghofer, Australia

Schistosomiasis caused by *Schistosoma japonicum* remains highly prevalent in the Philippines despite decades of preventive chemotherapy, partly due to the absence of accurate, field-deployable diagnostics. We recently developed two semi–point-of-care diagnostics (POCs): (1) an equipment-free cotton-syringe stool DNA extraction device paired with portable quantitative PCR, and (2) a latex microsphere-based lateral flow immunoassay detecting anti-SjSAP4 antibodies in serum. Laboratory validation using infected human samples showed diagnostic performance comparable to established methods—87.7%/92.1% sensitivity/specificity for the cotton-syringe system relative to Kato-Katz (KK) and commercial stool qPCR, and 80.6%/98.0% for the anti-SjSAP4 LFIA compared with KK. To evaluate field applicability, both POCs were deployed in six endemic Philippine villages, where community health workers were trained to perform the tests on residents (n=250 per village). Acceptability was assessed through questionnaires and focus group discussions. Preliminary findings highlight key challenges, including reluctance among health workers to handle stool samples and workflow bottlenecks during stool homogenization in the cotton-syringe method. Addressing these operational pain points will be essential for improving uptake and ensuring that these POCs can support more accurate, community-level schistosomiasis surveillance and monitoring.

CP1: Nick White Memorial 10 min talks

Time: Tuesday, 30/June/2026: 11:45am - 12:05pm · *Location:* Lecture Theatre 1
Session Chair: Katherine Andrews, Griffith University
Session Chair: Colin Sutherland, LSHTM

ID: 219 / CP1: 1
Contributed abstract
Conference Topics: Biochemistry, Drugs, Malaria
Keywords: Malaria, PfATP4, Drug resistance, Collateral sensitivity

Preserving Cipargamin Efficacy in *Plasmodium falciparum*: Understanding Resistance Pathways and Exploiting Collateral Sensitivity Strategies

Ruijia Liang[1], Deyun Qiu[1], Barbara Stokes[2], David Fidock[2], Adele Lehane[1]
[1]Research School of Biology, Australian National University, Canberra, Australian Capital Territory, Australia.; [2]Department of Microbiology & Immunology, Columbia University Irving Medical Center, New York, NY10032, USA.

Combating malaria caused by *Plasmodium falciparum* requires strategies to mitigate drug resistance. The clinical candidates cipargamin and SJ733 target the Na^+ pump PfATP4. A G358S mutation in PfATP4, identified in 68% of recrudescent cases in a cipargamin clinical trial, confers high-level resistance to both compounds while simultaneously increasing parasite sensitivity to PfATP4 inhibitors belonging to two distinct chemical classes ('I' and 'II'). To investigate whether collateral sensitivity could be leveraged in preserving cipargamin efficacy, we exposed 'hypermutator' parasites to class I and II compounds in combination with a high (20× IC_{50}) concentration of cipargamin. The cipargamin/class I combination led to an L354V mutation in PfATP4, while for cipargamin/class II, no viable parasites emerged across 6 selections. However, exposure of $PfATP4^{G358S}$ parasites to a class II compound drove the acquisition of an additional PfATP4 mutation (N355Y). The N355Y+G358S mutants exhibited > 2000-fold and 650-fold resistance to cipargamin and SJ733, respectively. The PfATP4-G358A mutation was also associated with treatment failure in the clinical trial. We show that this mutation confers 500-fold resistance to cipargamin, confirming its clinical significance. Together, these findings highlight that while multiple PfATP4 mutations can compromise cipargamin efficacy, combining certain PfATP4 inhibitors increases the barrier for resistance.

ID: 240 / CP1: 2
Contributed abstract
Conference Topics: Apicomplexa Biology, Cell Biology, Drugs, Host-parasite interactions, Malaria, Molecular Biology
Keywords: drug resistance, malaria, feeding, artemisinin

Understanding artemisinin resistance in the malaria parasite *Plasmodium falciparum* through high resolution imaging

Stuart Ralph, Long Huynh, Sophie Collier, Haowen Deng, Emma McHugh
Department of Biochemistry and Pharmacology, The University of Melbourne, Australia

Resistance to the frontline antimalarial drug artemisinin is primarily mediated by mutations in the *Plasmodium falciparum* protein Kelch13 (K13), whose function has remained unclear. Parasites carrying mutant K13 ingest red blood cell haemoglobin more slowly. Because artemisinin is activated by haem released during parasite feeding, reduced haemoglobin uptake likely lowers intracellular levels of toxic artemisinin-derived species. How wild-type or mutant K13 contributes to this feeding process was unknown. Using multiple imaging approaches, we show that K13 localises to the collar that maintains the cytostome, a stable parasite invagination used for uptake of host cytosol. Three-dimensional electron microscopy reveals that mislocalised K13 abolishes formation of the electron-dense collar that stabilises the cytostomal neck and disrupts cytostome formation itself. Consistent with this, haemoglobin degradation products, including haem and haemozoin, are reduced when K13 is inactivated. Using expansion microscopy together with super-resolution and lattice light-sheet microscopy, we further show that new K13 collars form and segregate to daughter cells before division, but that this biogenesis is delayed in mutant parasites. These findings indicate that artemisinin resistance arises through defective cytostome formation, reduced endocytosis, and diminished drug activation in resistant parasites.

CP2.1: Canines 10 minute talks sponsored by Elanco

Time: Tuesday, 30/June/2026: 11:45am - 12:15pm · *Location:* Lecture Theatre 2
Session Chair: Swaid Abdullah, The University of Queensland
Session Chair: Liisa Ahlstrom, Elanco

ID: 148 / CP2.1: 1
Contributed abstract
Conference Topics: Ectoparasites, Molecular Biology, One Health, Parasites of dogs, Veterinary Parasitology
Keywords: Flea, bacteriome, 16S RNA, Nanopore sequencing

Integrated morphological and molecular analysis of canine fleas in Cambodia and comparison of flea and host blood bacteriome

Saki Yamamoto[1,2], Shilpa Kapoor[1], Lucas Huggins[1], Vito Colella[1], Clare A. Anstead[1]
[1]Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Parkville, VIC 3010, Australia ; [2]Department of Biochemistry and Pharmacology, Bio21 Molecular Science and Biotechnology Institute, The University of Melbourne, Parkville, VIC 3010, Australia

Fleas (Siphonaptera) are globally important blood-feeding ectoparasites that infest companion animals, livestock, and humans. They are vectors of bacterial pathogens such as Rickettsia and Bartonella. Yet, many flea species and their associated bacterial communities in Cambodia remain poorly characterised. This project aims to identify flea species parasitising dogs in Cambodia and characterise their bacteriome. Fleas collected from dogs across multiple Cambodian regions were morphologically identified using established dichotomous keys. Genomic DNA was extracted from individual fleas, and full-length 16S rRNA gene nanopore metabarcoding will be performed, following previously developed and validated workflows to characterise flea-borne bacteria (FBB). This flea bacteriome data will be analysed alongside previously generated dog blood bacterial profiles, using paired samples from the same individual hosts. Preliminary morphological work revealed that most fleas parasitising Cambodian dogs belong to the genus Ctenocephalides, with C. orientis predominating. Sequencing will confirm species identity and the diversity of FBB pathogens, potentially detecting well known zoonotic agents and novel taxa that conventional PCR-based approaches may fail to detect. Integrating flea species identification with bacteriome data and host blood infection status provides initial evidence on the potential vector role of canine fleas in Cambodia and identifies gaps for future surveillance in under-resourced settings.

ID: 149 / CP2.1: 2
Contributed abstract
Conference Topics: Diagnostics, Epidemiology, Parasites of companion animals, Parasites of dogs, Protozoa, Veterinary Parasitology, Zoonoses
Keywords: toxoplasmosis, canine, owned dogs, risk factors, ELISA

Forty years on: What do handbags, dogs and *Toxoplasma gondii* have in common?

Jan Slapeta[1,2], Hanna Zecevik[1], Michael P. Ward[1]
[1]Sydney School of Veterinary Science, University of Sydney, Australia; [2]Sydney Infectious Diseases Institute, University of Sydney, Australia

Exposure to *Toxoplasma gondii* in dogs has been recognised for decades, including an Australian study conducted at the University Veterinary Teaching Hospital in Sydney (UVTHS) in 1982, which showed that exposure was common among dogs presented for veterinary care. Since then, major changes in canine lifestyle, urbanisation and veterinary practice have occurred, raising questions about whether exposure patterns have shifted and what factors now best explain risk. Using diagnostic serology data from approximately 500 dogs, we examined a contemporary cohort of client-owned dogs presented to a veterinary teaching hospital. Serological evidence of prior exposure remains common, consistent with historical findings. Age was the dominant predictor of seropositivity, with dogs aged four years or older showing substantially higher odds of exposure, reflecting cumulative lifetime contact with the parasite. Breed-associated exposure ecology further clarified risk: Toy breeds consistently showed the lowest likelihood of seropositivity, while other purebred dogs had markedly higher odds, suggesting lifestyle and environmental exposure, rather than fine breed distinctions, drive risk. From a veterinary perspective, canine *T. gondii* seropositivity reflects prior exposure rather than disease, and positive serology (particularly in older, non-Toy dogs) represents an expected background finding rather than evidence of toxoplasmosis.

ID: 150 / CP2.1: 3
Contributed abstract
Conference Topics: Diagnostics, Epidemiology, One Health, Parasites of companion animals, Parasites of dogs, Strongyloides, Zoonoses
Keywords: strongyloidiasis, quantitative PCR, temperate region, canine, veterinary

Retrospective screening reveals the rare occurrence of zoonotic *Strongyloides stercoralis* in dogs from temperate Australia, 2014-2024

Jaisy Chong[1], Jan Slapeta[1,2], Maira Nascimento Meggiolaro[1], Jennifer Green[1], Rogan Lee[3,4], Michael P. Ward[1], Nicolle Kirkwood[1]
[1]Sydney School of Veterinary Science, University of Sydney, Australia; [2]Sydney Infectious Diseases Institute, University of Sydney, Australia; [3]New South Wales Health Pathology, Centre for Infectious Diseases and Microbiology Laboratory Services, Level 3 Institute of Clinical Pathology and Medical Research (ICPMR), Westmead Hospital, Westmead, Australia; [4]School of Biomedical Sciences, Faculty of Medicine and Health, The University of Sydney, Australia

Strongyloides stercoralis is an intestinal nematode infecting humans and dogs, but its occurrence in dogs from temperate, traditionally non-endemic regions is poorly characterised, partly due to limited veterinary diagnostics. Recent reports from metropolitan areas raise concern that infections may be under-recognised. This study screened archived canine faecal DNA (n = 448) collected between 2014 and 2024 from two university veterinary teaching hospitals in Sydney, New South Wales, Australia, using a highly sensitive 18S rRNA real-time qPCR (limit of detection: two DNA copies). One sample (0.02%) was positive, containing approximately 6.8 × 10^3 18S rDNA copies, equivalent to 3.2 *Strongyloides ratti* third-stage larvae per 250

mg of faeces. Deep amplicon sequencing of partial *cox1* and 18S rDNA (HVR-I and HVR-IV) confirmed *S. stercoralis* potentially circulating between dogs and humans. The positive sample originated from a Border Collie puppy with gastrointestinal signs. Although rare, detection confirms the parasite's presence in companion dogs in a temperate urban setting. These findings support the utility of 18S rDNA-based qPCR for retrospective surveillance and its inclusion in molecular diagnostic panels for canine gastrointestinal disease and highlight the need for expanded surveillance in non-endemic regions.

CP3.2: Tropical Health 5 min talks sponsored by QIMR Berghofer, Centre for Tropical Health & Emerging Diseases

Time: Tuesday, 30/June/2026: 11:55am - 12:15pm · *Location:* Lecture Theatre 3
Session Chair: Darren Gray, QIMR Berghofer
Session Chair: Chika Zumuk, Queensland Institute of Medical Research

ID: 105 / **CP3.2: 1**
Contributed abstract
Conference Topics: Helminthology, Immunology, Microscopy, One Health, Vaccines, Veterinary Parasitology, Zoonoses
Keywords: Schistosomiasis, mRNA vaccines, Transmission blocking vaccines, Neglected tropical diseases, Schistosoma japonicum

Combatting Schistosomiasis japonica using mRNA Transmission Blocking Vaccines

Chika Zumuk[1,2], Hong You[1], Pengfei Cai[1], Severine Navarro[1], Malcolm Jones[1,3]
[1]Queensland Institute of Medical Research, Berghofer, Australia; [2]Faculty of Health, Medical and Behavioural Sciences, The University of Queensland; [3]School of Veterinary Sciences, The University of Queensland

Schistosoma japonicum, the Asian schistosome, is a zoonotic parasite that infects humans and more than 40 species of domestic animals. It is a principal causative agent of schistosomiasis, a neglected tropical disease affecting 240 million people globally. In endemic regions, bovines are major reservoirs responsible for up to 75-90% of environmental egg contamination that perpetuate transmission. Current control efforts rely heavily on chemotherapy with Praziquantel, though effective at reducing worm burdens, does not prevent reinfection with risk of developing drug resistance if used solely. These limitations highlight the urgent need for integrated strategies to achieve sustainable control. Mathematical modelling has indicated that coupling chemotherapy with an effective transmission-blocking vaccine (TBV) could achieve elimination of *S. japonicum* within a decade. In this context, my PhD project seeks to develop an mRNA TBV by evaluating parasite-derived proteins with demonstrated immunogenicity and protective potential. Preliminary murine trials demonstrated significant reductions in worm and egg burdens, as well as alleviation of liver pathology. The integration of such promising antigens in a multivalent mRNA vaccine represents a rational strategy to maximize protection and durability of response and combined with control measures offers a promising pathway toward sustainable schistosomiasis control and improved global public health outcomes.

ID: 197 / **CP3.2: 2**
Contributed abstract
Conference Topics: Epidemiology, Protozoa
Keywords: intestinal protozoa, soil-transmitted helminths, commensal protozoa, Papua New Guinea

An update on the assessment of intestinal parasite diversity in a tuberculosis-endemic community in rural Papua New Guinea

Jessica Scott[1], Daniel Pelowa[2], Wayne Melrose[1], Catherine Rush[1], Jeffrey Warner[1]
[1]James Cook University, Townsville, Australia; [2]Balimo District Hospital, Balimo, Western Province, Papua New Guinea

The prevalence and diversity of intestinal parasites in Papua New Guinea (PNG) and their potential impact as co-morbidities on endemic infectious diseases, such as tuberculosis (TB), remain poorly understood. This cross-sectional study provides an updated assessment of intestinal parasites (helminths and protozoa) in a TB-endemic rural PNG community. Participants were recruited from Balimo District Hospital, with stool and blood samples collected in June 2019 and January 2020. Faecal specimens (n=164) were examined by microscopy and qPCR, and plasma samples (n=121) were tested for *Strongyloides*-specific IgG antibodies using a commercial ELISA

Overall, 95.1% of participants had at least one type of intestinal parasite. Intestinal protozoa were detected in 85.4% of participants, most commonly detected were *Blastocystis* spp. (77.4%), *Entamoeba hartmanni* (29.3%), *E. coli* (23.3%), *Dientamoeba fragilis* (16.5%) and *E. polecki* (12.8%). Helminths were detected in 65.9%, predominantly *Necator americanus* (44.5%), *Ascaris lumbricoides* (37.8%) and *Strongyloides* spp. (17.7%). Polyparasitism was common, with 44.5% harbouring multiple protozoan and 31.1% infected with two or more helminths. These findings demonstrate a high burden and diversity of intestinal parasites in rural PNG. Whether co-infection with these parasites impacts the clinical outcome TB remains to be determined.

ID: 174 / **CP3.2: 3**
Contributed abstract
Conference Topics: Malaria, Other
Keywords: Insecticide Treated Nets, Vector Control, Malaria, Mosquito Control

Within-batch Inconsistencies and Elevated Temperature Decreases the Insecticidal Efficacy of Yahe LN Insecticide Treated Nets Delivered to Papua New Guinea: Implications for Quality Control, Transport and Storage

Holly Reichel[1], Melanie Koinari[1], Norelle Daly[1], Stephan Karl[1,2]
[1]Australian Institute of Tropical Health and Medicine, James Cook University, Australia; [2]Vector-Borne Diseases Unit, PNG Institute of Medical Research, Papua New Guinea

Insecticide treated nets (ITNs) are the most widely used mosquito control tool. To ensure efficacy and quality, ITN products undergo a prequalification process, with any changes to prequalified products to be reported to the World Health Organization. As a result, recipients can expect to receive products exhibiting consistent physical and insecticidal properties. A durability study showed that Yahe ITNs distributed in Papua New Guinea (PNG) in 2021 did not retain insecticidal efficacy for longer than several months. ITNs are frequently exposed to elevated temperatures during transport and storage and throughout their lifespan in the tropics. To better understand the potential impact of elevated temperature on insecticidal efficacy of Yahe, and to investigate why ITNs failed insecticidal efficacy tests after 6 months in PNG, Yahe ITNs were stored at elevated temperatures and evaluated using cone bioassays. Results revealed diverging physical and insecticidal properties of ITNs sampled from a single batch with ITNs exhibiting differences in colour, mesh size, weave, and insecticidal efficacy. Furthermore, ITNs exposed to 35- 50°C exhibited significantly decreased insecticidal efficacy. This study highlights inconsistencies in basic product properties of Yahe LN ITNs that are apparently not controlled or reported, and the importance of assessing current products for temperature-stability.

ID: 139 / **CP3.2: 4**
Contributed abstract
Conference Topics: Diagnostics, Epidemiology, Helminthology, Microscopy, Strongyloides
Keywords: strongyloidiasis, artificial intelligence, diagnostic, microscopy, neglected tropical disease

EnAIbling parasitic worm control – development of the first artificial intelligence diagnostic test for strongyloidiasis

Suzy Ossipow[1], Richard Bradbury[2], Catherine Gordon[1], Peter Ward[3], Matthew Watts[4], Darren Gray[1]
[1]QIMR Berghofer, Australia; [2]James Cook University, Australia; [3]ENAIBLERS, Sweden; [4]New South Wales Health Pathology, Westmead

Strongyloidiasis, “the most neglected tropical disease”, caused by the helminth *Strongyloides stercoralis*, is a major global concern. It can persist life-long following infection, unless treated, due to the auto-infective lifecycle. Infection can be fatal, particularly among patients with immunosuppression. Despite health risks, knowledge surrounding strongyloidiasis burden and diagnostics remains limited.

The World Health Organization (WHO) advocates for strongyloidiasis' inclusion in parasite control programs; however, with no population-based diagnostic tests and no large-scale surveys, large-scale treatments are not being provided. Global strongyloidiasis targets are off-track, and millions of people suffer from preventable debility.

Microscopy is a mainstay of population-based helminth surveys. Artificial intelligence (AI) improvements have led to AI-guided microscopy. A novel automated AI-based platform that images and digitises samples on standard microscopy slides to detect and quantify parasitic infections recently showed increased sensitivity and rapidity over human slide readers.

We are developing the first AI model for detecting *S. stercoralis* using larvae from lab and clinical samples, supporting AI training. We are trialling different preparations for *Strongyloides* spp. detection, and will undertake field validation in North East Arnhem Land. Our expected outcome is the first field-validated, AI-based *S. stercoralis* population-level diagnostic test, to allow population treatment programs to commence.

CP1.1: Nick White Memorial 5 min talks

Time: Tuesday, 30/June/2026: 12:05pm - 12:15pm · *Location:* Lecture Theatre 1
Session Chair: Katherine Andrews, Griffith University
Session Chair: Colin Sutherland, LSHTM

ID: 249 / **CP1.1: 1**
Contributed abstract
Conference Topics: Apicomplexa Biology, Cell Biology, Malaria, Microscopy, Molecular Biology, Protozoa
Keywords: malaria, microscopy, cellular biology, resistance

Using high-resolution imaging to understand how malaria parasites become resistant to frontline antimalarials

Long Huynh[1], Benjamin Liffner[2], Niall Geoghegan[3], Sabrina Absalon[2], Stuart Ralph[1]
[1]Department of Biochemistry and Pharmacology, Bio21 Molecular Science and Biotechnology Institute, The University of Melbourne, Parkville, Victoria, Australia; [2]Department of Pharmacology and Toxicology, Indiana University School of Medicine, Indianapolis, IN, USA; [3]The Walter & Eliza Hall Institute of Medical Research, Department of Medical Biology, The University of Melbourne, Parkville, VIC, Australia

Artemisinin resistance in *Plasmodium* parasites, driven by mutations in the parasite's Kelch 13 (K13) protein, threatens global malaria control. K13 is important for regulating the cytostome, a double-membraned invagination used to ingest host-cell haemoglobin. This process is important because haemoglobin digestion releases haem-iron as a toxic byproduct, which is required to activate artemisinin. Mutations in K13 cause slowed-feeding and lower haem levels, allowing parasites to survive drug exposure. However, the precise mechanisms by which K13 mutations impairs parasite feeding remains unclear.
We performed a variety of high-resolution and spatial-temporal microscopy techniques to investigate cellular changes when K13 is mutated. By expansion microscopy, we confirmed K13 to localise to distinctive ring-shaped structures (~160 nm) surrounding cytostomal necks. Using a machine-learning segmentation and automated quantification pipeline, we found K13 mutant parasites produced new cytostomes at significantly slower rates than the wild-type. K13 mutants had a 4h egress delay, producing fewer total K13 rings compared to wild-type, despite maintaining normal progeny counts. Furthermore, these mutants occasionally exhibited aberrant cytostome morphologies where K13 was absent from the cytostomal neck, correlating with decreased haemoglobin uptake efficiency.

These findings fundamentally advance understanding of artemisinin resistance by providing a mechanistic explanation for K13-mediated feeding defects.

ID: 153 / **CP1.1: 2**
Contributed abstract
Conference Topics: Drugs, Malaria, Protozoa
Keywords: Plasmodium, drug antagonism, delayed death inhibitors, mass spectrometry

Impacts of apicoplast-targeting antibiotics on dihydroartemisinin activation in *Plasmodium falciparum*

Zoe Tregloan-Dunn[1], Sophie Collier[1], Emily M. Crisafulli[1], Carlo Giannangelo[2], Jennifer Le[2], Darren Creek[2], Stuart A. Ralph[1]
[1]Department of Biochemistry and Pharmacology, The University of Melbourne, Australia; [2]Monash Institute of Pharmaceutical Sciences, Monash University, Australia

Malaria is a disease of significant and ongoing global burden, caused by parasites of the *Plasmodium* genus, with *Plasmodium falciparum* responsible for 90% of global malaria mortality. Effective drug treatment is instrumental to control of this disease, and the World Health Organisation (WHO) recommends artemisinin (ART) combinations for treatment of uncomplicated *P. falciparum* malaria. In this process of treatment, intentionally or otherwise, ART derivatives may be coadministered with apicoplast-targeting antibiotics such as doxycycline or clindamycin. However, previous work from our laboratory indicated an antagonistic relationship between these drugs. Antagonism between ART derivatives and apicoplast-targeting drugs may reduce efficacy and lengthen duration of treatment, as well as potentially increasing susceptibility to resistance mutations. In order to shed light on the mechanism of this antagonism, we are optimising a novel mass spectrometry assay that directly measures the degradation of ART under different conditions. Despite the discrete cellular targets of the drugs of interest, we posit that the downstream effects of these antibiotics on haemoglobin uptake are critical to ART activation and may be responsible for the antagonistic effect. Clarification of these drug class interactions may hold clinical significance for coadministration of antimalarial therapeutics.

CP6: Wildlife 1: Fish, Snakes & Turtles 15 min talk

Time: Tuesday, 30/June/2026: 1:30pm - 1:45pm · *Location:* Lecture Theatre 3
Session Chair: Nathan Bott, RMIT University
Session Chair: Amanda Ash, Murdoch University

ID: 183 / **CP6: 1**
Contributed abstract
Conference Topics: Coral Reef Parasites, Fish parasitology, Helminthology, Wildlife parasitology
Keywords: Taxonomy, Trematode, Life-cycle, Acanthocolpid

Life Cycle Speculation and Taxonomic Disruption Inspired by Discovery of an Unusual New Trematode on the Great Barrier Reef.

Ronen Sturrock, Storm Martin
Murdoch University

Acanthocolpids are a family of digenean trematodes mired with taxonomic instability and complexity. The life cycle is generally typified by trophic transmission via fish second-intermediate hosts and fish definitive hosts, with some exceptions. We described a new species of Acanthocolpidae from the Great Barrier Reef, which is the first known parasite for its definitive host, the sailfin snapper, *Symphorichthys spilurus* (Lutjanidae). Morphologically, this new species is consistent with the genus *Pseudolepidapedon*. However, phylogenetic analyses with our novel sequence data, the first for an Indo-Pacific species of the genus, also implicates some species of *Stephanostomum,* an infamously problematic and likely polyphyletic genus with over 90 species. These findings complicate the relationship and blur the boundaries between *Pseudolepidapedon* and *Stephanostomum*. Additionally, although the sailfin snapper is a large predatory fish, it feeds mostly on benthic invertebrates, and our new species bears some similarity to one of the exceptional species of acantholpids known to use molluscs, rather than fishes, as second-intermediate hosts. We discuss the potential taxonomic and life cycle revelations suggested by the discovery of this new species.

CP5: Immunology 1 - 15 min talks

Time: Tuesday, 30/June/2026: 1:30pm - 2:00pm · *Location:* Lecture Theatre 2
Session Chair: Danielle Stanisic, Institute for Biomedicine and Glycomics, Griffith University
Session Chair: Hannah Siddle, The University of Queensland

ID: 125 / **CP5: 1**
Contributed abstract
Conference Topics: Drugs, Immunology
Keywords: Rheumatoid arthritis, macrophage, hookworm, Ac-FAR-2, inflammation

Hookworm-inspired therapy for rheumatoid arthritis

Suchandan Sikder, Kim Miles, Connor McHugh, Darren Pickering, Haleagrahara Nagaraja, Rachael Ryan, Manoharan Kumar, Matt Field, Paul Giacomin, Alex Loukas, Roland Ruscher
James Cook University, Australia

Rheumatoid arthritis (RA) is a chronic autoimmune inflammatory disease affecting ~1% of the global population, with current therapies largely limited to symptom control and are associated with adverse effects. There is an urgent need for novel treatments targeting underlying disease mechanisms. Hookworms secrete immunomodulatory proteins that have evolved to regulate host immunity.

Here, we investigate a recombinant fatty acid- and retinol-binding protein, Ac-FAR-2, derived from the secretome of *Ancylostoma caninum*, as a hookworm-inspired therapeutic candidate for RA. Ac-FAR-2 significantly attenuated disease severity in a murine model of RA, with histological and confocal analyses revealing reduced macrophage infiltration in joint tissues. In vitro, Ac-FAR-2 suppressed inflammatory cytokine production in human peripheral blood mononuclear cells and THP-1-derived macrophages. This effect was associated with reduced co-stimulatory marker expression and impaired T-cell proliferation in co-culture systems.

Mechanistically, Ac-FAR-2 interacted with macrophage surface molecules and disrupted the arachidonic acid–prostaglandin E2 pathway. Transcriptomic profiling further demonstrated downregulation of NF-κB signaling, inflammasome-related genes, and other pro-inflammatory pathways in LPS-stimulated human macrophages.

These findings identify Ac-FAR-2 as a promising immunomodulatory candidate for RA and other macrophage-driven autoimmune diseases.

ID: 204 / **CP5: 2**
Contributed abstract
Conference Topics: Bioinformatics, Host-parasite interactions, Immunology, Malaria, Molecular Biology
Keywords: Malaria, multi-omics, bioinformatics, transcriptomics, host-parasite

Systems Immunology and Multi-omics approaches to understanding host immune heterogeneity in *Plasmodium* infection

Brenna Daily[1,4], Ashton Kelly[1], Martha Cooper[2,3], Quan Nguyen[1,3,4], Carla Proietti[1], Denise Doolan[1]
[1]Centre for Superbug Solutions, Institute for Moleculer Biosciences,The University of Queensland, Brisbane, Queensland, Australia; [2]Australian Institute of Tropical Health and Medicine, James Cook University, Cairns, Queensland, Australia; [3]Centre for Population and Disease Genomics, Institute for Moleculer Biosciences, The University of Queensland, Brisbane, Queensland, Australia; [4]Genomics and Machine Learning Lab, QIMR Berghofer Medical Research Institute, Brisbane, Queensland, Australia.

Malaria remains a major global health burden, with an estimated 282 million cases and 610,000 deaths across 80 countries in 2024. However, the mechanisms underlying protective immunity and inter-individual immune heterogeneity are still poorly understood, contributing to variation in susceptibility, disease severity, and vaccine efficacy.

This study applies a systems immunology and multi-omics approach to investigate the molecular basis of immune heterogeneity during Controlled Human Malaria Infection (CHMI). We aim to define an integrated framework of genes, proteins, and immune cell states that underpin *Plasmodium* parasite control and immune heterogeneity.

Using bulk RNA sequencing and single-cell data from peripheral blood mononuclear cells (PBMCs), we identified highly expressed and strongly correlated transcript pairs across mRNA, miRNA, and long non-coding RNAs (lncRNAs). These were incorporated into co-expression and regulatory networks to map transcriptional interactions. Correlation analyses revealed putative regulatory relationships, with lncRNA–mRNA pairs showing predominantly positive associations, suggesting coordinated expression across patient samples and over time.

Building on these findings, we are applying a probabilistic unsupervised machine learning framework to link transcriptional programs with proteomic signatures of immune activation. This integrative approach provides new insights into immune mechanisms driving parasite control while advancing multi-omics capabilities to the field of parasitology.

CP4: Cells, Molecules and Genes – Tribute to Bob Sinden - 15 min talk

Time: Tuesday, 30/June/2026: 1:35pm - 1:50pm · *Location:* Lecture Theatre 1
Session Chair: Alicja (Ala) Tabor, The University Of Queensland
Session Chair: Alexander Gofton, CSIRO

This session will begin with a 5 minute Tribute to Bob Sinden, given by Jake Baum, UNSW

ID: 239 / **CP4: 1**
Contributed abstract
Conference Topics: Fasciolosis/Liver fluke, Helminthology, Host-parasite interactions, Livestock Parasites
Keywords: Fasciola hepatica, Host-parasite interactions, In vivo, Pathogenesis, Invasion

The secret life of parasites: intravital microscopy opens a new frontier in helminth biology

Hayley Martinez DeCristi[1], Michael Kuligowski[2], Katharine Muscat[1], Javier Gonzalez-Miguel[3], Nichola Eliza Davies Calvani[1]
[1]Sydney School of Veterinary Science, Faculty of Science, The University of Sydney, NSW 2006, Australia; [2]Sydney Microscopy and Microanalysis, The University of Sydney, NSW 2006, Australia; [3]Laboratory of Helminth Parasites of Zoonotic Importance, Institute of Natural Resources and Agrobiology of Salamanca, Salamanca, Spain

Background and Aims

The earliest stages of helminth infection, during which parasites migrate through host tissues to their final niche, determine whether infection is successfully established and pathology ensues. Yet this critical window has remained largely inaccessible in vivo. Here, we present the first intravital microscopy (IVM) workflow to visualise any helminth parasite in real time within a living mammalian host. Using *Fasciola hepatica* as a model, we captured newly excysted juveniles (NEJ) during the earliest stages of infection in experimentally infected C57BL/6 mice.

Methods

Mice were infected with 175 *F. hepatica* metacercariae and serially imaged from 6 hours to 8 days post-infection (PI), with matched tissue and parasite sampling at 6, 12, 48, 120, 144, 168, 180 and 192 hours PI.

Results

Multiphoton IVM enabled real-time visualisation of parasite invasion, migration and host–parasite interactions within intestinal and hepatic tissues. These data provide the first direct view of early helminth pathogenesis as it unfolds in vivo, overcoming a major technical barrier that has constrained the field for decades.

Conclusion

This work establishes a new experimental framework for studying tissue-migrating helminths and provides a critical in vivo benchmark for refining emerging 3D co-culture models.

CP6.1: Wildlife 1: Fish, Snakes & Turtles 10 min talks

Time: Tuesday, 30/June/2026: 1:45pm - 2:25pm · *Location:* Lecture Theatre 3
Session Chair: Nathan Bott, RMIT University
Session Chair: Amanda Ash, Murdoch University

ID: 194 / **CP6.1: 1**
Contributed abstract
Conference Topics: Aquaculture, Diagnostics
Keywords: aquaculture; LAMP; diagnostics; bluefin tuna; blood flukes

Development and application of species-specific loop-mediated isothermal amplification (LAMP) assays for the rapid detection of blood flukes of bluefin tunas

Melissa J. Carabott[1], Cecilia Power[1], Sho Shirakashi[2], Paul A. Ramsland[1], Barbara F. Nowak[3], Nathan J. Bott[1]
[1]RMIT University, Australia; [2]Kindai University, Japan; [3]University of Tasmania, Australia

Blood flukes of the genus *Cardicola* (Digenea: Aporocotylidae) are a significant health concern in farmed and ranched bluefin tuna (*Thunnus* spp., BFT), contributing to substantial economic losses. Although recombinase polymerase assays (RPA) have been developed for two species of *Cardicola*, the high degree of sequence conservation across all three species infecting BFT limits reliable species-level discrimination. This study aimed to design, optimise and validate loop-mediated isothermal amplification (LAMP) for the detection of the three *Cardicola* species infecting BFT and apply these assays to Pacific bluefin tuna (*Thunnus orientalis*, PBT) cultured in Japan. Species-specific LAMP assays were successfully developed for *Cardicola forsteri*, *C. opisthorchis* and *C. orientalis* targeting ITS-2 ribosomal DNA. In addition, these assays were applied to DNA from three sample types targeting sites of significant pathology, the heart, gill necropsy and gill biopsy of 15 PBT. Results were compared to outputs of quantitative polymerase chain reactions (qPCR), the gold standard method for blood fluke detection in Australia, with LAMP demonstrating greater sensitivity. This research constitutes the first development and application of LAMP diagnostics for all three *Cardicola* species infecting BFT, providing a rapid, cost-effective and user-friendly alternative to conventional molecular diagnostics to support improved monitoring and management.

ID: 186 / CP6.1: 2
Contributed abstract
Conference Topics: Fish parasitology, Helminthology, Wildlife parasitology
Keywords: Trematodes, Sri Lanka, New genus, Opecoelidae

Unexpected relatives: an integrated taxonomic approach unites species separated by half a century of confusion

Lenin Manage[1], Erandi Pathirana[2], Amanda Ash[1], Storm Martin[1]
[1]Murdoch University, Australia; [2]University of Sri Jayewaradanepura

Plagioporus and *Podocotyle* are historically the largest, most problematic and among the oldest genera in the Opecoelidae, the richest family of the Trematoda. Both genera were defined by combinations of generalised characters prone to homoplasy. Nevertheless, the two concepts have always been considered mutually exclusive. Through an integrated approach, we present a close phylogenetic relationship, clearly congeneric, between a nominal species of each genus. We recollected the two nominal species from known hosts near to their type-localities, *Podocotyle parupenei* from goatfishes (Mullidae) on the Great Barrier Reef, Australia (type locality: Fiji) and *Plagioporus jagannathi* from threadfinbream (Nemipteridae) in Sri Lanka (type locality: India). *Podocotyle* is currently recognised in the subfamily Podocotylinae, but *Podocotyle parupenei* has since been recombined as *Podocotyloides parupenei* in the Hamacreadiinae. Likewise, *Plagioporus* currently belongs to the Sphaerostomatinae, but *Plagioporus jagannathi* was recombined as *Macvicaria jagannathi* in the Opistholebetinae. Our analysis revealed that two species are distinct, but closely related, andbelong to none of *Plagioporus*, *Podocotyle*, *Macvicaria*, or *Podocotyloides*, but instead resolve within a fifth subfamily, the Decemtestinae. We have proposed and delineated a new genus to accommodate these taxa, exemplifying how homoplasy has long obscured true relationships and how intergrated taxonomy can cut through.

ID: 152 / CP6.1: 3
Contributed abstract
Conference Topics: Apicomplexa Biology, Microscopy, Molecular Biology, Protozoa, Wildlife parasitology
Keywords: Apicomplexa; wildlife parasitology, Chelonian haemoparasites

Molecular and microscopic detection of *Haemocystidium* spp. in freshwater turtles in Australia

Isaac L. L. Pinto[1,2], T. Franciscus Scheelings[1], Maristela P. Peixoto[2], Robin B. Gasser[1], Anson V. Koehler[1]
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Haemocystidium spp. are apicomplexan parasites infecting reptiles, particularly freshwater turtles, yet their diversity and epidemiology remain poorly understood. This study aimed to detect and characterise *Haemocystidium* spp. in freshwater turtles from diverse populations in Australia. In total, 114 blood samples were collected from 8 turtle species. Blood smears were prepared from each turtle and examined by light microscopy, and genomic DNAs were isolated from matching blood samples using the DNeasy Blood & Tissue Kit (Qiagen). Molecular detection was performed using a nested PCR, targeting the small subunit (*SSU*) rRNA gene, employing primers HaemNF1/HaemNR2 in the first reaction and HaemF/HaemR2 in the second, generating an amplicon of ~480 bp. *Haemocystidium* DNA was detected in 36/114 (31.6%) samples by PCR, while intraerythrocytic stages observed in 25 samples by microscopy (21.9%) were consistent with *Haemocystidium* spp. Morphologically, parasites appeared as elongated or irregular forms within erythrocytes, occasionally causing host cell distortion. The higher detection rate achieved using PCR indicates an increased sensitivity compared with microscopy, although DNA sequencing is required to confirm amplicon specificity. These findings show that *Haemocystidium* infections are relatively common in freshwater turtles and emphasise the importance of combining molecular and morphological approaches for parasite detection.

ID: 248 / CP6.1: 4
Contributed abstract
Conference Topics: Biodiversity, Ecology, Host-parasite interactions, Invasive Species, Wildlife parasitology
Keywords: pentastome, Raillietiella orientalis

Invaded parasite communities of the banded water snake (*Nerodia fasciata*) in Florida, USA

Madison Harman[1], Natalie Claunch[2], Ben Gonzalez[3], Arik Hartmann[3], Zuania Colon-Pineiro[3], Nicolina Valore[3], Andres Calavia[3], Elyse Martin[3], Sasha Sandoval[3], Marcos Rodriguez-Lapido[3], Ashley Hamersma[3], W. James Whelpley[4], Maria Ojeda-Rojas[4], Michelle Bassis[4], Christina Romagosa[1], Mike Kinsella[5], Ana Longo[3], Melissa Miller[4]
[1]Department of Wildlife Ecology and Conservation, University of Florida, Gainesville, FL, USA; [2]United States Department of Agriculture, Gainesville, FL, USA; [3]Department of Biology, University of Florida, Gainesville, FL, USA; [4]Department of Wildlife Ecology and Conservation, University of Florida, Fort Lauderdale Research and Education Center, Davie, FL, USA; [5]HelmWest Laboratory, Missoula, MT

The state of Florida, USA is a global invasion hotspot where more than 150 species of non-native reptiles and amphibians have been introduced. One of the most detrimental, the Burmese python (*Python bivittatus*), is less well-known for having co-introduced an invasive parasite, Raillietiella orientalis. This pentastome has since spilled over into both native and invasive herpetofauna and is a suspected contributor to the decline of several native snake populations, including the banded water snake (*Nerodia fasciata*). To investigate the influence of invasive *R. orientalis* infection on the physiology and native parasite communities of *N. fasciata*, we conducted metabolic rate testing prior to euthanasia and necropsies. We examined snakes externally then screened the mouth, respiratory tract, gastrointestinal tract and body cavity for parasites. Invasive pentastomes were found in 74% (23/31) of snakes with an infection intensity of 1-45, while no native pentastomes were found. Hosts with invasive pentastomes had a higher prevalence of nematodes, trematodes, cestodes, acanthocephalans, and respiratory mites relative to snakes without pentastomes. Results are discussed in regard to the potential impacts of invasive species on native parasite communities and resulting influence on hosts.

CP4.1: Cells, Molecules and Genes – Tribute to Bob Sinden - 10 min talks

Time: Tuesday, 30/June/2026: 1:50pm - 2:40pm · *Location:* Lecture Theatre 1
Session Chair: Alicja (Ala) Tabor, The University Of Queensland
Session Chair: Alexander Gofton, CSIRO

ID: 226 / CP4.1: 1
Contributed abstract
Conference Topics: Malaria, Molecular Biology
Keywords: malaria, transmission, population dynamics, barcoding

Defining transmission bottlenecks across the malaria parasite mosquito-to-vertebrate lifecycle using cellular barcoding

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Plasmodium parasites, the causative agents of malaria, must cycle between mosquito vectors and vertebrate hosts, encountering population bottlenecks at each transition. Parasite numbers can drop from billions in the bloodstream to only a few in the mosquito midgut after feeding, and again during transmission back to a host, where only a small fraction of sporozoites establish a liver infection. These bottlenecks shape parasite population structure and key selective pressures, influencing drug resistance and vaccine escape. However, the magnitude of these bottlenecks has not been precisely quantified.

Here, we use cellular barcoding to track parasite populations at high resolution across the lifecycle. We generated a library of *P. berghei* parasites containing over 1,000 unique DNA barcodes integrated into a neutral genomic locus. This diverse population was transmitted between mice and mosquitoes via natural bites or intravenous sporozoite injection, with samples collected across the lifecycle stages for barcode sequencing.

Our initial analyses quantify changes in barcode diversity throughout the lifecycle, identifying where parasite diversity is lost or maintained. These results define key transmission bottlenecks and highlight stages most susceptible to intervention. This framework can be extended to assess how drugs and vaccines impact parasite population dynamics, informing more precise malaria control strategies.

ID: 118 / CP4.1: 2
Contributed abstract
Conference Topics: Apicomplexa Biology, Malaria, Protozoa
Keywords: Plasmodium falciparum, mosquito transmission, vitamin B6, gene editing

Transmission studies of gene-edited *Plasmodium falciparum* indicate crucial role of parasite pyridoxal 5'-phosphate (PLP) biosynthesis in mosquito stage development

Lindsay Stewart[1,2], Mojca Kristan[1,2], Mariana Reis Wunderlich[1,2], Annie Tremp[1,2], Michaela Bikarova[1,2], Naoki Matoba[1,2], Rosie Bridgwater[1], Mufuliat Toyin Famodimu[1], Eduardo Alves[1], Colin Sutherland[1,2], Michael Delves[1]
[1]Faculty of Infectious and Tropical Diseases, LSHTM, United Kingdom; [2]Human Malaria Transmission Facility, LSHTM, London, UK

The LSHTM Human Malaria Transmission Facility provides access to human malaria parasite transmission both for research groups within the London School of Hygiene & Tropical Medicine and external collaborators worldwide. Our specialist team works with these collaborators to design and execute studies relating to the transmission of *Plasmodium* parasites using gametocytes grown *in vitro* in the laboratory or collected directly from clinical samples received by the UK HSA Malaria Reference Laboratory and fed to insectary-reared *Anopheles* mosquitoes. As a case study illustrating the support offered by the HMTF, we present new data indicating an important role for pyridoxal 5'-phosphate biosynthesis (vitamin B6; PLP) in mosquito stage development of *P. falciparum*. Enzymes in this pathway were found to be highly enriched in an analysis of the stage V gametocyte "translatome", the subset of the total gametocyte proteome actively incorporating a pulse of labelled methionine in late-stage gametocytes. Disruption of PDX2, one of two subunits of pyridoxal 5'-phosphate synthase, reduced gametocyte infectivity to *Anopheles coluzzi* mosquitoes. This reduction was manifest in three measures: oocyst numbers per mosquito midgut, oocyst diameter and, most profoundly, number of salivary gland sporozoites per mosquito. All three negative outcomes were rescued by vitamin B6 supplementation.

ID: 123 / CP4.1: 3
Contributed abstract
Conference Topics: Apicomplexa Biology, Cell Biology, Malaria, Molecular Biology
Keywords: Malaria, Transmission-Blocking Vaccine, Pfs230, Pfs48/45, HAP2

A HAP2-like protein interacts with vaccine candidate Pfs230 and is essential for efficient malaria transmission

Ezra Bekkering[1], Sanne Grievink[1], Mariangela Longobardi[1], Rianne Stoter[1], Geert-Jan van Gemert[1], Randy Yoo[2,3], Jean-Philippe Julien[2,3], Taco Kooij[1], Matthijs Jore[1]
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Transmission-blocking vaccines aim to induce antibody responses that prevent human-to-mosquito malaria transmission by targeting the parasite inside the mosquito midgut. The furthest advanced transmission-blocking vaccine candidates are based on the Pfs230:Pfs48/45 complex. During the preparation of a cryo-EM structure of the endogenous Pfs230:Pfs48/45 complex, we found a previously unidentified protein that interacts with Pfs230. This protein is without described function and is conserved throughout all *Plasmodium spp.*. Based on structural homology modelling, we have tentatively dubbed this protein PfHAP2-novel (PfHAP2n). We generated a *PfHAP2n*^{KO} parasite line that is able to undergo gametocytogenesis and

gametogenesis. However, just like *Pfs230*^{KO} parasites, male microgametes are no longer able to attach to erythrocytes and no longer form “exflagellation centres”. *PfHAP2n*^{KO} parasites have a strong, male-dependent reduction in mosquito infectivity. We have recombinantly produced PfHAP2n, and we are testing whether these constructs can elicit malaria transmission-blocking antibodies in immunization studies. Our results have uncovered a novel protein that is essential for malaria transmission, which might be a suitable target for future transmission-blocking vaccine development.

ID: 244 / **CP4.1: 4**
Contributed abstract
Conference Topics: Apicomplexa Biology, Biochemistry, Cell Biology, Malaria, Microscopy, Molecular Biology
Keywords: Invasion machineries, Rhoptries, Mosquito, Liver Cells.

Investigating CERLI1 and CERLI2 function during the invasion process of *Plasmodium falciparum* sporozoites into salivary glands of *Anopheles* mosquito and human liver cells.

Leonhard Arinanto[1], Benjamin Liffner[2], Danny Wilson[2], Anton Cozijnsen[1], Geoffrey McFadden[1], Dean Goodman[1]
[1]University of Melbourne, School of BioScience, Australia; [2]Adelaide University, School of Biological Sciences, Australia

Plasmodium falciparum sporozoites must successfully invade *Anopheles* mosquito salivary glands and human liver hepatocytes to establish infection. While CERLI1 and CERLI2 are known critical mediators of blood-stage invasion, their potential roles during these sporozoite-specific events remain unexplored.

This study investigates CERLI1 and CERLI2 function during sporozoite transmission. Using CRISPR/Cas9, we generated transgenic parasite lines with inducible knockouts for CERLI1 and CERLI2. Phenotypic analyses revealed that inducing these knockouts significantly reduces the sporozoite load within the salivary glands of *Anopheles* mosquitoes. Furthermore, utilizing ultrastructure expansion microscopy (U-ExM), we demonstrated that this impaired salivary gland colonization correlates with structural defects in the rhoptry organelles.

Collectively, our findings establish CERLI1 and CERLI2 as essential, multi-stage mediators of the *Plasmodium* invasion machinery. Elucidating their function in the mosquito vector, alongside pending investigations into hepatocyte entry, provides vital insights into the fundamental biology of parasite transmission.

ID: 233 / **CP4.1: 5**
Contributed abstract
Conference Topics: Apicomplexa Biology, Cell Biology, Host-parasite interactions, Malaria, Microscopy
Keywords: Expansion Microscopy, Confocal Microscopy, Malaria, Mosquito, Method Development

Developing Expansion Microscopy for Mosquitoes to Investigate the Biology of Mosquitoes and Mosquito-Transmitted Parasites

Joe van den Bergh[1,2], Leonhard Arinanto[3], Geoff McFadden[3], Dean Goodman[3], Craig Williams[4], Ryan O'Handley[5], Danny Wilson[1,2], Benjamin Liffner[1,2]
[1]Adelaide University, School of Biological Sciences, Adelaide, South Australia, Australia.; [2]Adelaide University, Institute of Photonics and Advanced Sensing, Adelaide, South Australia, Australia.; [3]The University of Melbourne, School of Biosciences, Melbourne, Victoria, Australia.; [4]Adelaide University, College of Health, Adelaide, South Australia, Australia.; [5]Adelaide University, School of Animal and Veterinary Sciences, Adelaide, South Australia, Australia.

Light microscopy is the most widely used tool in the study of cell biology but many of the subcellular structures of parasites are too small to see even with the best light microscopes. Recently, a technique called expansion microscopy that physically enlarges parasites has revolutionised parasite cell biology. To date, expansion microscopy has only been applied on either parasites grown *in vitro* or from isolated host tissues. We wanted to perform expansion microscopy on whole mosquitoes, to simultaneously visualise the ultrastructure of both malaria parasites and their mosquito hosts, but the presence of the Chitin-rich mosquito cuticle prevents expansion. Here, we develop expansion microscopy for whole mosquitoes by first digesting the cuticle with enzymes. We validate that the mosquitoes expand as expected, show preservation of mosquito anatomy, and visualise it at exquisite detail. The application of this methodology allows for the co-visualisation of parasite and mosquito ultrastructure including microvilli, salivary glands, gut-microbiome and ovaries. Additionally, we have validated this technique for other arthropods: *Ixodes holocyclus* and *Drosophila melanogaster*, highlighting its versatility across arthropods. While developed to investigate cell biology, application of this technique could greatly improve the resolution of spatial omics techniques in the study of vector-parasite interactions.

CP5.1: Immunology 1 - 10 min talks

Time: Tuesday, 30/June/2026: 2:00pm - 2:30pm · *Location:* Lecture Theatre 2
Session Chair: Danielle Stanisic, Institute for Biomedicine and Glycomics, Griffith University
Session Chair: Hannah Siddle, The University of Queensland

ID: 241 / **CP5.1: 1**
Contributed abstract
Conference Topics: Apicomplexa Biology, Cell Biology, Host-parasite interactions, Immunology, Malaria, Protozoa
Keywords: B Cell Immunology, Malaria, Single-cell genomics, Host-parasite Interactions, Vaccinology, Monoclonals

Identifying Novel Non-CSP Antigen targets on the Surface of Malaria *Plasmodium Falciparum* Sporozoites

Jem Murdoch[1], Ian Cockburn[2], Deborah Burnett[1], Jake Baum[1]
[1]UNSW, Australia; [2]ANU, Australia

Human infection with malaria begins with the injection of sporozoites by a feeding mosquito. The sporozoite surface has a dense layer of one, immunodominant protein, called circumsporozoite protein (CSP). This protein contains 38 NANP amino acid tandem repeats which are highly immunogenic. The current licensed malaria vaccines being rolled out in Africa (RTS,S and R21) both target CSP, however they require multiple boosters to maintain high antibody titre, with vaccine efficacy waning overtime. Given CSP's immunodominance and prevalence, could it be acting as an immunological decoy, evolved to evade an immune response from other critical cell surface proteins on the parasite? Here, we have used a CSP-tolerant mouse model, immunising mice with human infective *Plasmodium falciparum* sporozoites to induce an immune response towards non-CSP antigens on the surface of the sporozoite. We then performed 10x single-cell sequencing of the expanded B cell clones from immunized mice and generated a panel of monoclonal antibodies. Using these monoclonals alongside, polyclonal sera we have identified non-CSP sporozoite surface antigens as potential targets for next generation malaria vaccines.

ID: 238 / **CP5.1: 2**
Contributed abstract
Conference Topics: Immunology, Malaria
Keywords: Plasmodium vivax, IgG, IgM, Natural acquired immunity, Clinical protection

Naturally acquired IgG and IgM responses to *Plasmodium vivax* and association of protection from clinical malaria

Janani Karunarathne[1,2], Ivo Mueller[1,2], Rhea Longley[1,2,3]
[1]Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria, Australia; [2]Department of Medical Biology, The University of Melbourne, Parkville, Victoria, Australia; [3]Faculty of Tropical Medicine, Mahidol University, Bangkok, Thailand

Plasmodium vivax is the most widespread *Plasmodium* species causing human malaria, remains a major global health risk. Effective interventions against *P. vivax* remain challenging due to its distinct biology. Although vaccines exist for *Plasmodium falciparum*, *P. vivax* lacks a licensed vaccine and has few candidates in clinical trials. Naturally acquired clinical immunity to malaria, driven by antibody responses that develop through repeated exposure in endemic areas, provides promising insights for vaccine development. This study evaluated whether IgG and IgM responses to *P. vivax* antigens are associated with protection against clinical malaria across transmission settings. Antibody responses to 61 antigens were simultaneously measured using the Luminex INTELLIFLEX platform in longitudinal cohorts from low transmission settings (Brazil, n = 258; Thailand n = 70) and a high transmission setting (Papua New Guinea, n = 184), where participants were followed over time for clinical malaria episodes used in the data analysis. The study shows that high IgG levels against RBP2b, EBPII, MSP3a, MSP5, RBP2a, CyRPA, RIPR, Pv-fam-a (PVX_090265), MSP7, RAMA, RBP2c-non-binding region and stAR related lipid transfer protein are strongly associated with protection from clinical malaria across all three cohorts. Overall, this study has identified both well-studied vaccine candidates and underexplored candidates.

ID: 227 / **CP5.1: 3**
Contributed abstract
Conference Topics: Immunology, Malaria, Vaccines
Keywords: Antibodies, Vaccine, HIV, Malaria

Impact of HIV infection on malaria antibody responses induced by the RTS,S vaccine or naturally acquired in adults

Grace Wright[1,2], Liriye Kurtovic[1,2,3], D. Herbert Opi[1,2,3], Cythia Lee[4], Chris Ockenhouse[4], Emily Locke[4], Lucas Otieno[5], Nate Copeland[5], NCT04661579 Clinical Trial Team[4,5], James G. Beeson[1,2,3]
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Plasmodium falciparum malaria remains a major global health burden. Co-infection with HIV, common among adults in malaria endemic regions, increases susceptibility to malaria and disease severity. However, the impact of HIV on vaccine induced and naturally acquired malaria immunity remains poorly understood. We evaluated a cohort of Kenyan adults naturally exposed to malaria who were vaccinated with the RTS,S malaria vaccine as part of a phase-IIb clinical trial. Individuals were either HIV-negative (n=204) or HIV-positive (n=45) at baseline. Using a multi-antigen multi-functional assay platform, we quantified antibody responses (IgG, Fc-receptor binding and complement fixation) to 35 malaria antigens, including the RTS,S vaccine antigen CSP, in plasma samples collected 28 days after vaccination. There was no difference in CSP IgG responses, including functional activities between HIV-negative and HIV-positive individuals. However, IgG and functional antibody responses to a majority of the non-vaccine malaria antigens were significantly higher in HIV-negative compared to HIV-positive individuals. HIV infection was associated with widespread impairment in the acquisition of naturally acquired malaria immunity, but did not

substantially impact RTS,S vaccine induced immunity. Our findings suggest that malaria vaccines could provide benefit to HIV-positive people who have impaired acquired immunity and are at a higher risk of malaria.

CP6.2: Wildlife 1: Fish, Snakes & Turtles 5 min talks

Time: Tuesday, 30/June/2026: 2:25pm - 2:45pm · *Location:* Lecture Theatre 3
Session Chair: Nathan Bott, RMIT University
Session Chair: Amanda Ash, Murdoch University

ID: 130 / **CP6.2: 1**
Contributed abstract
Conference Topics: Fish parasitology
Keywords: Myxozoa, Marine fish parasites, Bali, Biodiversity, Long-read sequencing

Genomic and taxonomic identification of myxosporean parasites infecting fishes in Bali, Indonesia

Diana Bulla Castaneda[1], Cristian Trisca[1], Cecilia Power[1], Terry Miller[2], Nathan Bott[1]
[1]School of Science, RMIT University, Melbourne, VIC, Australia; [2]Queensland Museum Kurilpa, Brisbane, QLD, Australia

Bali, an Indonesian island located at the centre of the Coral triangle, is considered a marine biodiversity hotspot; however, the diversity and taxonomy of many parasitic groups in fishes remain poorly understood. Myxosporeans a taxonomically diverse group, are obligate, spore-forming parasitic cnidarians. Their true diversity is likely much higher than currently described, due to cryptic species and limited number of studies in region. To date, no myxosporean species have been formally described from Bali. This study aims to identify myxozoan infections in marine fishes from Bali. Gall bladder and muscle samples from 18 host families, including commercially significant families Carangidae, Siganidae, Lutjanidae and Scombridae, were obtained from the Queensland Museum research collection. Morphological traits of myxospores were described using light microscopy following the guidelines of Lom & Arthur (1989). DNA was extracted from spore preparations, and ribosomal DNA regions (18S-28S) were amplified using universal and myxosporean-specific primers. PCR amplicons were prepared for long-read sequencing (Oxford Nanopore PromethION), and the resulting sequences were analysed using Geneious Prime. Based on morphological and molecular approaches, these samples are provisionally assigned to the genera *Kudoa* spp. and *Ceratomyxa* spp., pending formal description. This dataset will provide the first comprehensive baseline for myxosporean in Bali.

ID: 265 / **CP6.2: 2**
Contributed abstract
Conference Topics: Aquaculture, Fish parasitology
Keywords: Myxosporean parasites

Towards a better understanding of myxosporean parasites infecting Australian fishes

Cristian Trisca[1], Diana Bulla Castaneda[1], Cecilia Power[1], Terry Miller[2], Nathan J. Bott[1]
[1]RMIT University Melbourne, STEM College, School of Science; [2]Queensland Museum, Kurilpa, Head of Biodiversity and Geosciences

Myxosporean parasites are obligate spore-forming, microscopic, parasitic cnidarians. The genera *Unicapsula* and *Kudoa* are increasingly recognised as contributors to post-harvest myoliquefaction ("jellymeat") in commercially important marine fishes. Despite the economic and social impact, these parasites remain understudied, particularly in Australian waters. Molecular data remains limited, with most studies relying on partial 18S and 28S ribosomal DNA (rDNA) sequences. This study focuses on the genetic characterisation of myxosporean parasites infecting wild-caught Yellowtail Kingfish (*Seriola lalandi)* and Mahi Mahi (*Coryphaena hippurus*), and the identification of infections in previously unreported species, including Australasian Snapper (*Pagrus auratus*) and Australian Bonito (*Sarda australis*). Long-range amplification of the complete rDNA operon (18S-ITS1-5.8S-ITS2-28S) was performed using LongAMP Taq DNA polymerase followed by Oxford Nanopore long-read sequencing. This resulted in the generation of high-coverage, near-complete rDNA operons without the need for multiple overlapping PCRs, addressing a key limitation in current myxosporean molecular studies. These findings expand the known host range of myxosporean parasites in Australian fisheries and provide genomic resources for future diagnostics and ecological studies. This work also highlights the need for improved molecular surveillance of myxosporean parasites in Australian waters using long-read sequencing approaches.

ID: 179 / **CP6.2: 3**
Contributed abstract
Conference Topics: Biodiversity, Fish parasitology, Helminthology, Wildlife parasitology
Keywords: Trematoda, Digenea, Opecoelidae, new species, homology

A new spined trematode in a spineless family

Storm Martin, Allyn Lim, Helen Armstrong
Murdoch University, Australia

The Opecoelidae is the richest family of the Trematoda, with sexual adults in a broad range of marine and freshwater fishes. Opecoelids are usually recognisable for a combination of generalised characters and the lack of specialised features; the most conspicuously absent feature is tegument spines. We present a new opecoelid, representing a new genus, with tegument spines, justified as belonging to the family on the basis of morphological and phylogenetic study. The new species was found in the northern pearl perch *Glaucosoma buergeri* (Glaucosomatidae) from Ningaloo Reef, Western Australia. It is the first trematode known from any pearl perch, which is a small but commercially important family comprising four species, three of which are endemic to Australian waters. The most intriguing question raised by this discovery is determining whether the spines in this opecoelid can be considered homologous with those found in many other trematode families – we will present a case that the spines are indeed homologous.

ID: 111 / **CP6.2: 4**
Contributed abstract

Conference Topics: Aquaculture, Ecology, Fish parasitology, Invasive Species, Wildlife parasitology
Keywords: introduced species, parasite ecology, host-parasite relationships, freshwater fish

Parasites of larval and juvenile freshwater fish in Australia: how small is too small to be infected?

Di Barton, Leia Rogers, Shokoofeh Shamsi
Charles Sturt University, Australia

Knowledge of the parasitic infections of freshwater fish in Australia is sporadic but is concentrated on reports of infection in larger specimens. The dynamics of infection with parasites in larval and juvenile freshwater fish is unknown. Thus, this study utilised fish specimens previously collected for a river health monitoring program to determine levels of parasitic infection: the native *Macullochella peelii*, *Retropinna semoni*, *Hypseleotris* spp., *Phylipnodon grandiceps*, *Macquaria ambigua*, and the introduced *Cyprinus carpio*. Dissections of fish found six different morphotypes: the monogenean *Dactylogyrus extensus*, the adult nematode *Procamallanus* (*Spirocamallanus*) sp., the mite *Hydrozetes* sp., and the unidentified larval cysts, larval nematodes and adult nematodes. Histological examination determined infections with the protozoans *Trichodina* sp. and *Chilodonella* sp. The smallest fish found to be infected with a parasite was a 6mm *C. carpio*.

CP5.2: Immunology 1 - 5 min talks

Time: Tuesday, 30/June/2026: 2:30pm - 2:45pm · *Location:* Lecture Theatre 2
Session Chair: Danielle Stanisic, Institute for Biomedicine and Glycomics, Griffith University
Session Chair: Hannah Siddle, The University of Queensland

ID: 146 / CP5.2: 1
Contributed abstract
Conference Topics: Immunology, Malaria, Vaccines
Keywords: pre-erythrocytic malaria, antigen discovery, cellular immunology

Single-cell dissection of protective T cell immunity in a genetically attenuated *Plasmodium* sporozoite vaccine model

Grace Rochfort Peters[1], Feyza Colakoglu Veli[1], Harry Pollard[1], Chiyun Lee[2], Andrew Balmer[2,3], Venkata Krishna Kanth Makani[4], Richard Pearson[2], Mike Johnson[1], Masahiro Ono[5], Alok Joglekar[4], Daniel Fernandez Ruiz[1], Jake Baum[1,5]
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Despite major progress over the past 25 years, malaria remains a significant global health burden, and protection from current vaccines is limited. Pre-erythrocytic vaccines targeting the liver stage of infection are therefore promising. Immunisation with genetically attenuated *Plasmodium* parasites, such as *P. falciparum* GA2, can induce strong, even sterile, protection in humans. Liver-resident memory $CD8^+$ T cells play a key role in this protection, yet their antigenic targets remain poorly defined.

To address this, we isolated reactive T cells following immunisation with a protective dose of *P. berghei* GA2. Antigen-responsive clones were identified using the “Timer of Cell Kinetics and Activity” (Tocky) mouse model, which detects recently activated T cells via *Nr4a3*-driven fluorescent protein expression and enables analysis of signalling dynamics. These cells underwent single-cell RNA and TCR sequencing to define transcriptional profiles and clonal expansion as compared to mock-immunised controls.

A subset of expanded TCRs was expressed in murine $CD8^+$ Jurkat cells and used to screen ~200,000 predicted epitopes via a chimeric SABR platform. This system presents pMHC complexes linked to signalling domains, enabling antigen discovery. This pipeline will identify targets for validation and inform next-generation malaria vaccine design.

ID: 177 / CP5.2: 2
Contributed abstract
Conference Topics: Genomics, Immunology, Malaria
Keywords: antigenic diversity, polymorphisms, immune responses

Naturally Acquired Antibody Responses to Polymorphic MSP1 Domains in Papua New Guinean Children

Katie M. Stanhope[1,2], Myo T. Naung[1,2], Kirsty M. McCann[1,2], Lee M. Yeoh[1,3], Maria Kaius-Ome[4], Daisy Mantila[4], Moses Laman[4], Jo-Anne Chan[1,5,6], Leanne J. Robinson[1,4], Ivo Mueller[7,8], James G. Beeson[1,3,9], Alyssa E. Barry[1,2]
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Malaria vaccine development is heavily challenged by the extensive genetic diversity of *Plasmodium falciparum* antigens. The full-length merozoite surface protein 1 (MSP1) is a promising blood-stage vaccine candidate, but its global diversity remains a critical hurdle for broad efficacy. To inform rational vaccine design, we evaluated the population genetic diversity and patterns of selection across MSP1 domains, followed by serological analysis of one highly polymorphic region.

Using 10,974 *P. falciparum* genomes from 26 countries globally, our population genomic analyses identified Block 4 as a putative target of immune selection, with high levels of polymorphism and strong signatures of balancing selection. We synthesised a peptide representing the most common variant of the MSP1 Block 4 domain and measured IgG antibody reactivity against it using Enzyme-Linked Immunosorbent Assays (ELISAs), utilising sera from Papua New Guinean children exposed to natural infection.

Despite the extensive polymorphism and balancing selection, we found antibody reactivity to the MSP1 Block 4 peptide in children developing immunity. Further work will include testing additional circulating variants individually and in combination using competition ELISAs to identify antigenically distinct variants, defining their relevance for multivalent vaccine design.

ID: 266 / **CP5.2: 3**
Contributed abstract
Conference Topics: Malaria
Keywords: Plasmodium berghei

Investigating the functions of platelets in protection against malaria infection

Nicole Lim, Brendan McMorran
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While platelets are known for their clotting function, they have immune functions as well. A recent discovery that platelets preferentially bind to senescent erythrocytes and aid in their clearance suggests that platelets are involved in erythrophagocytosis. This preferential binding is also observed in blood diseases including malaria. This project aims to explore the immune protective role of platelets in the clearance of infected erythrocytes during blood stage malarial infection.

We first determine whether platelets aid in the clearance of infected erythrocytes by infecting platelet-depleted mice with *Plasmodium berghei*. We observed a decrease in phagocytosed infected erythrocytes in the spleens of platelet-depleted mice compared to controls.

Next, we investigate whether it is platelets directly affect splenic macrophage function or is binding to erythrocyte an essential first step. We co-incubate murine splenic macrophages with erythrocytes in the presence of platelets. We observed increased erythrophagocytosis in the presence of platelets *ex vivo*. We will compare the expression level of functional markers of splenic macrophages in platelet-depleted mice to controls.

Finally, we will look at a cohort of spleen-intact and splenectomised malaria patients to determine whether our findings can also be observed in humans.

CP4.2: Cells, Molecules and Genes – Tribute to Bob Sinden - 5 min talks

Time: Tuesday, 30/June/2026: 2:40pm - 2:50pm · *Location:* Lecture Theatre 1
Session Chair: Alicja (Ala) Tabor, The University Of Queensland
Session Chair: Alexander Gofton, CSIRO

ID: 229 / **CP4.2: 1**
Contributed abstract
Conference Topics: Apicomplexa Biology, Cell Biology, Drugs, Malaria, Microscopy
Keywords: Microscopy, Gameotcytes, drug resistance, protein structure

Investigating the role of Kelch13 in *Plasmodium falciparum* gametocytes

Haowen Deng, Stuart Ralph, Matthew Dixon, Sophie Collier
University of Melbourne, Australia

Resistance *of Plasmodium falciparum* to artemisinin, the current frontline anti-malarial, greatly threatens global malaria control. Mutations in the parasites' Kelch13 (K13) protein is the major driver behind this resistance. In asexual stages, K13 forms ring at neck of parasites' cytostome, the membrane invagination responsible for haemoglobin uptake from RBC. Activation of artemisinin requires reaction with haem released from digested haemoglobin. K13 mutation is thought to cause resistance through decreasing haemoglobin uptake, causing reduced activation of artemisinin. How K13 modulates this process is undetermined. Despite continued spread of resistance in endemic regions, the role of K13 and K13 mutations on parasite transmission remain unresolved. Using an endogenously tagged version of K13, we imaged K13 throughout gametocyte development, using standard fixes as well as expansion microscopy. Similar to its asexual counterpart, K13 was found to form ring structures throughout gametocyte development, though the amount and the location of these rings varies across stages. In late-stage gametocytes, K13 also forms a hollow tubular structure, likely serving a distinct function compared to the rings. To test if K13 modulates gametocyte sensitivity to artemisinin, we have employed the Knock sideway (KS) system and KS of K13 potentially increases gametocyte survival under artemisinin treatment.

ID: 246 / **CP4.2: 2**
Contributed abstract
Conference Topics: Cell Biology, Genomics, Malaria, Molecular Biology
Keywords: malaria, gametocytes, sex ratio, Plasmodium berghei, inbreeding and outbreeding.

Exploring the regulation of sex ratios in *Plasmodium berghei*.

Xuexin Xia, Sophie Collier, Geoffrey McFadden, Dean Goodman
The University of Melbourne, Australia

Malaria, caused by single-cell parasites of the genus *Plasmodium*, currently has a widespread impact on global public health security. As a sexually reproducing species, understanding the sexual biology of *Plasmodium* is fundamental to developing approaches to control malaria transmission. It is widely accepted that *Plasmodium* exhibits a female-biased sex ratio (male to female is <1) during gametocyte development and actively adjusts this ratio to promote outcrossing.

However, due to the lack of straightforward research models, how *Plasmodium* controls and changes its sex ratio is still unclear. By genetically manipulating sexual differentiation in *Plasmodium berghei,* we could artificially bias sex ratio. Using this system, we found that, contrary to existing evidence, when the growth of the parasite stabilizes, the sex ratio of the gametocytes (male to female) is close to 1:1. Furthermore, isogenic parasite lines do not modify sex ratio in response to an overabundance of one sex.

P1: Elsevier Plenary Lecture Series International Journal for Parasitology: Parasites and Wildlife (IJP:PAW) Invited Lecturer

Time: Tuesday, 30/June/2026: 3:30pm - 4:15pm · *Location:* Plenary Lecture Theatre
Session Chair: Andrew Thompson, Murdoch University

ID: 273 / **P1: 1**
Invited speaker abstract

Marine mammal parasites in a changing ocean: Ecology, pathology, and conservation implications

Kristina Lehnert
Institute of Terrestrial and Aquatic Wildlife Research, University of Veterinary Medicine Hannover, Germany

Marine mammals are long-lived apex predators that integrate ecological signals across their ocean habitat. Their diverse and often still cryptic parasite fauna reflects their evolutionary history, adaptations and trophic links in marine ecosystems. Gastro-intestinal helminths, respiratory nematodes and arthropod parasites are frequently found in harbour and grey seals as well as harbour porpoises of the North and Baltic Sea. As opportunistic hunters, they consume diverse fish species and are exposed to trophically transmitted gastro-intestinal helminths with complex life cycles involving invertebrate and fish hosts. Variation in parasite infection patterns reflect differences in diet, host immune traits, and environmental conditions that influence parasite life cycles. Given the zoonotic potential of some helminths, species identification and monitoring their epidemiology in marine food webs is critical within a One Health framework. The respiratory tract of marine mammals is crucial to enable efficient oxygen exchange in diving animals. Lung nematodes belonging to the Metastrongyloidea are among the most pathogenic parasites in odontocetes and seals and can cause severe pathology and mortality. Little is known about parasite fauna of orcas. The first record of lungworms occurred in neonate killer whales and indicated a direct transmission and a new pseudaliid species in orcas. Arthropod parasites in marine mammals have developed unique traits to adapt to the marine environment and their vagile marine mammal hosts over long evolutionary time scales. Seal lice and nasal mites are directly transmitted between their hosts and reflect social interactions and population dynamics of their hosts. Marine mammal parasites can serve as valuable bio indicators for wildlife health and host ecology. Understanding host parasite interactions in marine wildlife is essential for assessing epidemiology of infectious pathogens, biodiversity conservation, and ecosystem resilience under ongoing environmental change.

P2: Elsevier Plenary Lecture Series International Journal for Parasitology: Drugs and Drug Resistance (IJP:DDR) Invited Lecturer

Time: Tuesday, 30/June/2026: 4:15pm - 5:00pm · *Location:* Plenary Lecture Theatre
Session Chair: Sarah Preston, Federation University Australia

ID: 275 / **P2: 1**
Invited speaker abstract

Progress understanding the mechanism of action of praziquantel yields opportunity for development of novel anthelmintics

Jonathan Marchant
Department of Cell Biology, Neurobiology & Anatomy, Medical College of Wisconsin, Milwaukee, WI 53226

The drug praziquantel (PZQ) has been used for over 40 years to treat many parasitic worm infections, and serves as the key clinical therapy for schistosomiasis. Our laboratory has proposed that the target of PZQ is a parasite ion channel belonging to the transient receptor potential melastatin (TRPM) subfamily, named $TRPM_{PZQ}$. Structural insight across orthologs of $TRPM_{PZQ}$ in different parasites provides opportunity to expand our toolbox of ligands at these targets, which in turn provides new approaches to modulating parasitic flatworm biology and understanding the endogenous role played these fascinating channel throughout the parasitic lifecycle.

In this presentation, I will discuss our emerging understanding of the workings of $TRPM_{PZQ}$, highlight new opportunities for targeting $TRPM_{PZQ}$ and other members of the TRPM family for the purpose of developing new anthelmintics, and speculate on how identification of $TRPM_{PZQ}$ informs the likelihood and possible trajectories toward clinical resistance to PZQ.

Wednesday, 01/July/2026

9:00am - 9:10am Tribute: Tribute to Dame Bridget Ogilvie AC delivered by Professor Karen Day, The University of Melbourne

Location: Plenary Lecture Theatre

Session Chair: Aaron Jex, WEHI

Professor Karen Day, The University of Melbourne, will deliver the Tribute to Dame Bridget Ogilvie AC.

Plenary Lecture Theatre

9:10am - 9:45am BOM: Bridget Ogilvie Medal Award and Oration

Location: Plenary Lecture Theatre

Session Chair: Aaron Jex, WEHI

CP7: Education & Outreach - 15 min talks

Time: Wednesday, 01/July/2026: 9:45am - 10:30am · *Location:* Plenary Lecture Theatre
Session Chair: Michelle Power, Macquarie University

ID: 100 / **CP7: 1**
Contributed abstract
Conference Topics: Education/Outreach
Keywords: Australian parasitology outreach

20 years of inspiring parasitology outreach

Lisa Jones, **Nick Smith**
Australian Society for Parasitology Inc., Australia

Parasites are a part of everyone's life; they infect our pets, the meat and crops we eat, and us. They also infect our iconic marsupial wildlife and the fish in our unique oceans and reefs, sometimes with devastating consequences. Australia, like everywhere else, also has a large number of common, human parasites, particularly in our remote communities. Some of these can be chronic, incapacitating and even life-threatening. Bernard Lee Singleton's magnificent painting, Gula Guri mayin (which means "Heal the body"), explores themes of parasites and health. The Australian Society for Parasitology is known for its inspirational and groundbreaking outreach programs over the past two decades using games, art and technology to encourage new and innovative ways of engaging with audiences in the science of parasites. ASP researchers have developed and delivered hands-on activities to engage audiences in science and communicate about parasites and their impact on people in Australia and around the world. In this presentation audience survey data collected over 20 years will be presented to investigate changes in themes, styles and audiences of the ASP outreach program. Be inspired, delighted and maybe you'll be reminising along with the presenter, and finish with some hands-on fun!

ID: 247 / **CP7: 2**
Contributed abstract
Conference Topics: Education/Outreach, One Health, Zoonoses
Keywords: Education, One Health, Pandemic, Surveillance

From farm to outbreak: One Health parasitology education on one campus

Charlotte Oskam
Murdoch University, Australia

Preparing the One Health workforce to detect and respond to zoonotic and parasitic disease threats demands practised, integrated decision-making across human, animal, and environmental health sectors. Australia's only veterinary school with a working farm on campus, Murdoch University co-locates a teaching veterinary hospital, research farm, bushland and wetland reserves, and public health facilities within walking distance of metropolitan hospitals, providing a natural living laboratory for parasitology and zoonotic disease surveillance training. BMS501 *Zoonoses, Pandemic Surveillance and Preparedness* is a core unit within Murdoch's Master of Infectious Disease Surveillance and Control. Field and laboratory practicals use the campus environment directly, with students conducting real sample collection, parasite identification, and vector surveillance across human, animal, and environmental interfaces on site. Students build preparedness plans, One Health stakeholder maps, and risk communication products in team-based workshops. Indigenous-led teaching anchors the unit in culturally safe governance and First Nations perspectives on surveillance. The unit culminates in a large-scale simulated outbreak using the EpiGames mobile application, opened to staff and students from human health, veterinary, and environmental science backgrounds. When the human, animal, and environmental health interfaces are literally on campus, authentic One Health parasitology training becomes possible.

ID: 267 / **CP7: 3**
Contributed abstract
Conference Topics: Education/Outreach
Keywords: Home-school groups, education, science communication

Delivering Microbes & Parasites Workshops for Home-School Groups

Rina Wong
Curtin Medical Research Institute and Curtin Health Nexus, Curtin Faculty of Health Sciences Pro-Vice Chancellors Office, Curtin University, Bentley campus, Western Australia 6102, Australia Dr Rina, PO Box 393, Osborne Park, Western Australia 6917, Australia

Home-school groups often face challenges in delivering science education, including limited access to specialised equipment and reduced confidence among parents when teaching complex concepts. These gaps create a valuable opportunity for researchers and academics to contribute their expertise through outreach, bringing authentic, hands-on science experiences directly to learners.

A series of Microbes & Parasites Workshops (n=7), developed and delivered by a parasitologist and supported by the Engaging Children in Science (ECIS) STEM volunteer team, demonstrates this impact across Western Australia. The workshops have reached home-school groups in Ocean Reef (2021), Baskerville (2022), Midland (2024, 2026), Izzelle's (2025) and Gwelup (2026). Each session blends hands-on science with creative STEAM activities, drawing on expertise in diagnostic microbiology, infectious diseases research and science communication. Through interactive demonstrations, storytelling and visual exploration, learners investigate bacteria, fungi, viruses and parasites, discovering how microbes shape human health and the environment.

Aligned with the WA Science Curriculum, the workshops build curiosity, scientific thinking through inquiry, observation and age-appropriate experimentation. The inclusive approach supports diverse learners, including children aged 4–16 and those with ADHD and autism, to ask questions, think critically and see themselves as young scientists. Challenges, insights and impact from this work will be shared.

S4: Vaccines Symposium sponsored by Institute for Biomedicine and Glycomics, Griffith University

Time: Wednesday, 01/July/2026: 11:00am - 11:20am · *Location:* Lecture Theatre 2
Session Chair: Danielle Stanisic, Institute for Biomedicine and Glycomics, Griffith University
Session Chair: Anouschka Akerman, The University of Queensland

ID: 279 / **S4: 1**
Invited speaker abstract

A whole organism vaccine platform to target parasites of medical and veterinary importance

Danielle Stanisic
Institute for Biomedicine and Glycomics, Griffith University, Southport, Australia,

Malaria continues to be a major cause of illness and death worldwide. Although efforts to develop a malaria vaccine date back to the 1940s, achieving a highly effective formulation that provides durable protection has remained a significant challenge.

Our work has shown that a chemically attenuated, whole parasite blood-stage malaria vaccine can elicit robust, CD4+ T cell–dependent immunity that protects against diverse malaria parasite strains in pre-clinical rodent models. In malaria-naïve human volunteers, a similar chemically attenuated *Plasmodium falciparum* blood-stage vaccine prevented the onset of blood-stage infection in a subset of individuals following controlled blood-stage parasite challenge. To our knowledge, this represents the first instance in which a blood-stage malaria vaccine has completely averted infection in human volunteers.

Despite these promising findings, the use of chemically attenuated parasites in endemic settings is constrained by various practical and logistical considerations. To address these limitations, we reformulated the vaccine by incorporating blood-stage parasites into liposomes. In rodent models, a whole blood-stage parasite liposomal formulation retained high efficacy after freezing or lyophilisation, supporting its suitability for field deployment.

Building on these results, we extended this platform to the related apicomplexan parasite *Babesia*, which causes babesiosis in humans as well as in livestock and companion animals. A lyophilised, liposome-based whole-parasite *Babesia* blood-stage vaccine induced strong cross-species protection in rodent models, highlighting its potential for both human and veterinary use.

Both the malaria- and babesiosis liposomal vaccine candidates are now advancing into clinical trials.

CP10: Wildlife 2: Mammals, Birds, Lizards & Wetas 15 min talk

Time: Wednesday, 01/July/2026: 11:00am - 11:15am · *Location:* Lecture Theatre 3
Session Chair: Haylee Crawford-Weaver, DCCEEW
Session Chair: Nicholas Fountain-Jones, University of Tasmania

ID: 124 / **CP10: 1**
Contributed abstract
Conference Topics: Biodiversity, Ectoparasites, Veterinary Parasitology, Wildlife parasitology
Keywords: Ixodes hirsti, Artificial tick feeding, Vector biology, Ticks, Microbiome

First *in vitro* feeding of an Australian wildlife tick, *Ixodes hirsti,* provides insights into host cues, feeding biology, morphology and changes in microbiome structure.

Vincent C. Duru[1,2,3], Bahar E. Mustafa[1], Abdul Ghafar[1], Ian Beveridge[1], Charles G. Gauci[1], Khawla Elati[2,3], Ard M. Nijhof[2,3], Abdul Jabbar[1]

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Artificial tick feeding systems (ATFS) provide ethical alternatives to *in vivo* models for studying tick biology, yet their application remains largely restricted to a few species. *Ixodes hirsti*, an Australian marsupial tick, remains understudied due to challenges in maintaining its life cycle under laboratory conditions. Here, we report the first successful *in vitro* feeding of I. *hirsti* larvae, and microbiome profiling combined with morphological characterisation of moulted nymphs. Larvae derived from field-collected engorged females were fed using silicone membrane supplemented with raw kangaroo hair and/or hair extract. Microbiome composition of the larvae was assessed using 16S rRNA amplicon sequencing. Nymphs were characterised using morphological (scanning electron microscopy) and molecular (targeting mitochondrial markers – *cox1* and 16S) approaches. Attachment success differed significantly among treatments ($p = 0.001$), with hair extract yielding the highest attachment rate (71%). However, kangaroo hair improved feeding performance, reducing time to engorgement (9.17 ± 0.72 days) and increasing engorgement weight (0.91 ± 0.01 mg). Blood feeding reduced microbial richness and evenness, increased microbiome variability ($p < 0.001$), and enriched *Stenotrophomonas,* suggesting feeding-associated shifts in microbial composition. This study provides a proof-of-concept for a host-free platform for studying Australian wildlife ticks, enabling controlled investigations of vector biology and tick–microbe interactions.

CP8: Drugs & Drug Resistance 1 - 10 min talks

Time: Wednesday, 01/July/2026: 11:00am - 11:50am · *Location:* Lecture Theatre 1
Session Chair: Christopher Hart, Griffith University
Session Chair: Hannah Smith, Griffith University

ID: 119 / **CP8: 1**
Contributed abstract
Conference Topics: Drugs, Livestock Parasites, One Health, Parasites of companion animals, Parasites of dogs, Protozoa, Veterinary Parasitology
Keywords: Giardia drug discovery

Combating treatment refractory giardiasis with new antigiardial compounds and novel drug combination strategies

Tina Skinner-Adams[1,2], Keely Fayd'Herbe[1,2], Christopher Hart[1,2], Andrew Riches[3], John Ryan[3]

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On an annual basis >300 million people develop giardiasis, a disease that impacts child development and the long-term health of many adults. However, there is no vaccine for this disease and treatment options are failing due to multiple factors including drug resistant parasites. Moreover, current treatment strategies are monotherapies that do little to combat the development of drug resistance. To improve this position combination therapies that include new compounds with unique mechanisms of action are needed. However, little has been done to identify best practice combination therapies for giardiasis.

As a mechanism to pave the way towards the development of highly effective therapies for giardiasis we have been investigating the activity of novel compounds and compound combinations against *Giardia* parasites *in vitro*. These studies have identified multiple synergistic compound combinations that may be useful in the *in vivo* including combinations that have Interaction (I) values >3.0 ($p<0.05$). While *in vivo* studies are now needed to determine how these combination therapies work in clinical settings, taken together with pharmacokinetic data, these results are exciting and suggest that novel antigiardial combination therapies can be developed to help combat treatment refractory giardiasis.

ID: 164 / **CP8: 2**
Contributed abstract
Conference Topics: Drugs, Host-parasite interactions, Molecular Biology
Keywords: Toxoplasma, Quinolones, Host Response

Effects of endochin-like quinolones (ELQs) on *Toxoplasma gondii* infection and responses of human retinal pigment epithelial cells: implications for the treatment of ocular toxoplasmosis

Liam M. Ashander[1], Melissa H. Brown[2,3], Giles Best[1], J. Stone Doggett[4], Katherine M. Liebman[4], Keryn A. Williams[1], Justine R. Smith[1]

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Toxoplasma gondii causes ocular toxoplasmosis, a vision-threatening retinal infection. Current therapeutics are not curative. ELQs are promising drugs not yet evaluated for ocular toxoplasmosis.

Human retinal pigment epithelial cells (ARPE-19 line) were infected with *T. gondii* GT-1 or GPHT tachyzoites and treated with ELQ-316 or ELQ-685. Tachyzoite growth 50% inhibitory concentrations (IC_{50s}) were calculated. Tachyzoite invasion, replication, and egress were assayed. Cell viability was evaluated by XTT assay. Cell response transcripts were measured by RT-qPCR.

ELQ IC_{50s} for GT-1 and GPHT were nanomolar-range and approximately equivalent (ELQ-316: 22.96±4.33nM vs. 21.23±3.92nM; ELQ-685: 1.08±0.34nM vs. 0.88±0.20nM; unpaired t-test, $p>0.05$). Both treatments significantly reduced replication (tachyzoites/rosette: ELQs ≤1.77±0.07 vs. untreated ≥6.85±0.66, $p<0.0001$; one-way ANOVA). Tachyzoite invasion and egress were not impacted by either drug. ELQs ≤12μM did not reduce cell viability (mitochondrial respiration (arbitrary units): ELQ 12μM 2.37±0.13 vs. untreated 2.19±0.07; $p>0.05$; one-way ANOVA). In GT-1-infected cells, ELQ treatment reduced 7 transcripts (*CCL2*, *CXCL8*, *ICAM1*, *IL1B*, *IL6*, *NFKB1*, *PDCD1LG2;* p≤0.0042) and increased 3 transcripts (*CXCL10*, *TGFB2*, *VCAM1*; p≤0.0048), versus untreated controls (one-way ANOVA). In GPHT-infected cells, ELQ treatment reduced 5 transcripts (*CXCL8*, *ICAM1*, *IL6*, *NFKB1*, *REL;* p≤0.043).

ELQs were non-toxic in ARPE-19 cells, reduced *T. gondii* replication, and altered host inflammatory mediator expression.

ID: 142 / **CP8: 3**
Contributed abstract
Conference Topics: Apicomplexa Biology, Biochemistry, Drugs, Malaria, Molecular Biology, Protozoa
Keywords: malaria, drug discovery

Identifying the mechanism of action of the antiplasmodial pantothenate analogue AH-2-45 in *Plasmodium falciparum*

Scarlett Cox[1], Zaynab Radih[1], Chunling Blue Lan[2], Karine Auclair[2], Kevin J. Saliba[1]
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Plasmodium falciparum, the deadliest human malaria parasite, has developed resistance to all clinically used antimalarials, highlighting the need for new compounds with novel modes of action. Pantothenate analogues kill *P. falciparum* by targeting the biosynthesis or utilisation of coenzyme A (CoA), an essential enzyme cofactor. Pantothenamides (PanAms), pantothenate analogues in which the carboxyl group is replaced by an amide group, exhibit potent *in vitro* activity. Unfortunately, PanAms are degraded *in vivo* by human pantetheinase. Modification of the labile amide bond has given rise to pantetheinase-resistant PanAm mimics. AH-2-45, a ring-substituted PanAm mimic, exhibits nanomolar antiplasmodial activity and is metabolised by CoA biosynthesis enzymes into a CoA antimetabolite, proposed to inhibit downstream CoA-dependent pathways. However, its precise target remains unknown. Whole-genome sequencing of *in vitro*-generated AH-2-45-resistant *P. falciparum* revealed a missense mutation in the gene encoding the endoplasmic reticulum-resident glycerol-3-phosphate 1-O-acyltransferase (PfGPAT), an essential CoA-dependent enzyme involved in phospholipid biosynthesis. We are currently genetically validating PfGPAT as the AH-2-45 resistance determinant and characterising the functional impact of the resistance-associated mutation using PfGPAT activity assays. Elucidating this mechanism may establish PfGPAT as a novel antimalarial drug target and guide structural optimisations of the AH-2-45 antimetabolite and related PanAm mimics.

ID: 154 / **CP8: 4**
Contributed abstract
Conference Topics: Apicomplexa Biology, Drugs, Host-parasite interactions, Malaria
Keywords: Plasmodium, Anopheles, transmission, inhibitor

Plasmepsin IX and X dual inhibitor impairs sporozoite development in mosquitoes and their infectivity in human hepatocytes

Elena Lantero Escolar[1,2], John A. McCauley[3], David B. Olsen[3], Alan F. Cowman[1,2], Justin A. Boddey[1,2]
[1]Walter and Eliza Hall Institute of Medical Research, Australia; [2]University of Melbourne, Australia; [3]Merck & Co., Inc., USA

WM382 is an inhibitor for Plasmepsin IX and X in *Plasmodium* spp blood and liver stages. These proteases are essential and ubiquitous in *Plasmodium* spp., where they process diverse proteins involved in egress and invasion of host cells. This makes them ideal targets for drug intervention. Plasmepsin IX and X are also expressed in sporozoites. To decipher their role in this stage, WM382 was administered to mosquitoes after *Plasmodium falciparum* infection. The number of developing oocysts was the same irrespective of WM382 treatment, though a modest increase in size was detected following drug administration, suggesting differential parasite development. Furthermore, the number of salivary gland sporozoites was dramatically reduced when mosquitoes were treated with WM382. This phenotype points to a defect in egress of *P. falciparum* sporozoites from the oocyst. We identified accumulation of unprocessed precursors of the essential protein AMA1 in haemolymph and salivary gland sporozoites when treating with WM382, while processing of CSP remained unaffected. Some WM382-treated sporozoites could still invade salivary glands but they displayed significant loss-of-function phenotypes during cell traversal and infection of human HC04 hepatocytes. These results show a role of plasmepsin IX/X during the mosquito stages, raising the possibility of control interventions during transmission.

ID: 250 / **CP8: 5**
Contributed abstract
Conference Topics: Ectoparasites, Livestock Parasites, Molecular Biology
Keywords: resistance, acaracides, Rhipicephalus, genomics, cattle

Defining chemical resistance in the Australian cattle tick (Rhipicephalus australis)

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Cattle ticks are a burden on global agriculture, with chemical treatments the primary method of control. However, ticks are being selected for resistance to these chemicals. Current tests for chemical resistance are slow and require laboratory facilities, inhibiting testing. In this project, we aim to undertake deep sequencing to define polymorphisms controlling resistance and build a database for monitoring of resistance changes. In preliminary studies, a survey and sampling system has been established to capture cattle ticks from across the infested range. Based on initial collections, resistance has been assessed across ten properties, defining resistance to synthetic pyrethroids (SP), amitraz, fluazuron and macrocyclic lactones. A newly developed rapid test for chemical resistance (RaTexT^{TM}) was used to define resistance to a synthetic pyrethroid, deltamethrin. Resistance to deltamethrin has been found at 100% of properties regardless of whether this chemical is currently in use. In contrast, fluazuron resistance was found at 40% of properties and only at properties where it is currently in use. Future work will assess the allele frequencies of the known polymorphism driving SP resistance, *kdr,* to determine the accuracy of rapid testing methods and deep sequencing will be undertaken on resistant and susceptible ticks for each chemical class.

CP10.1: Wildlife 2: Mammals, Birds, Lizards & Wetas 10 min talks

Time: Wednesday, 01/July/2026: 11:15am - 11:45am · *Location:* Lecture Theatre 3
Session Chair: Haylee Crawford-Weaver, DCCEEW
Session Chair: Nicholas Fountain-Jones, University of Tasmania

ID: 210 / **CP10.1: 1**
Contributed abstract
Conference Topics: Bioinformatics, Host-parasite interactions, Wildlife parasitology
Keywords: hairworm, parasite-host interaction, microbiome, bioinformatics

Let's Swap: Microbial Sharing Between Hairworm Life Stages and Their Hosts

Rosemary Presburger[1], **Robert Poulin**[1], **Priscila Madi Salloum**[2]
[1]University of Otago, New Zealand; [2]University of Aukland, New Zealand

Parasites and their hosts engage in an 'evolutionary arms race.' As a parasite evolved tools to infect its host, the host will develop adaptations to evade infection. Microbiomes may also play key roles in the arms race between the two antagonists. We characterised the entire microbiome of parasite-host interactions, using the New Zealand native hairworms *Gordius paranensis* and *Euchordodes nigromaculatus* and their cave wētā hosts (Rhaphidophoridae) to investigate host and parasite microbiome overlap. Infected wētā, uninfected wētā, juvenile hairworms, and free-living mature hairworms all have microbiomes including viruses, bacteria, fungi, archaea and protists. We found that parasites harbour their own unique microbial taxa, in addition to microbes shared with infected wētā but not with uninfected wētā. This suggests hairworms do not rely on their host to develop a microbiome. As hairworms progress through their life stages, they exhibit a core microbiome; however, microbial community composition varies significantly across life stages, suggesting the environment has a significant effect on the parasite's microbiome. Therefore, hairworm microbiomes are dynamic across the life cycle and modulated by the host and environment. The hairworm's diverse microbiome raises the intriguing possibility that its symbiotic microbes contribute to the iconic host manipulation (suicidal water-jumping) associated with hairworms.

ID: 131 / **CP10.1: 2**
Contributed abstract
Conference Topics: Diagnostics, One Health, Protozoa, Wildlife parasitology, Zoonoses
Keywords: Toxoplasma gondii, Red Foxes, Sentinels, Epidemiology

Serological and molecular detection of *Toxoplasma gondii* in naturally infected red foxes (*Vulpes vulpes*) from Victoria, Australia

Tharaka Liyanage[1], **Leonardo Brustenga**[1,2], **Panayotis Loukopoulos**[1], **Megan Fisher**[1], **Jessica Haining**[1], **Charles G. Gauci**[1], **Livia Lucentini**[3], **Alessandro D. Uboldi**[4,5], **Christopher J. Tonkin**[4,5], **Giulia Morganti**[2], **Giulia Rigamonti**[2], **Fabrizio Passamonti**[2], **Fabrizia Veronesi**[2], **Jasmin Hufschmid**[1], **Abdul Jabbar**[1]
[1]Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Werribee, Victoria, Australia; [2]Department of Veterinary Medicine, University of Perugia, Perugia, Italy; [3]Department of Chemistry, Biology and Biotechnology, University of Perugia, Perugia, Italy; [4]Department of Medical Biology, The University of Melbourne, Parkville, Victoria, Australia; [5]The Walter and Eliza Hall Institute of Medical Research, Parkville, Victoria, Australia

Toxoplasma gondii is a zoonotic parasite with a global distribution that can infect a wide range of warm-blooded hosts. This study investigated, for the first time, the seroprevalence and genetic variability of *T. gondii* in red foxes (*Vulpes vulpes*) from Victoria, Australia. Animals from both regional Victoria and the metropolitan Melbourne area were sourced from trappers and shooters involved in pest control. Sera were screened for anti-*T. gondii* IgG antibodies using a modified agglutination test, and tissue samples were tested using a qPCR assay targeting the 529 bp repeated element. qPCR positive samples were genotyped using a five-marker (L358, 5'SAG2, 3'SAG2, c22-8, GRA6) polymerase chain reaction-restriction fragment length polymorphism protocol. Anti-*T. gondii* antibodies were detected in 38.9% (30/77) of foxes, and parasite DNA was identified in 23.4% (18/77) of animals. Genotyping revealed a predominance of *T. gondii* clonal Type II whereas two isolates showed new alleles attributable to Type II-like genotypes. These findings suggest that Australian red foxes are frequently exposed to *T. gondii* and may play an important role as epidemiological sentinels to assess the circulation of *T. gondii* in the Australian environment, with implications for both wildlife conservation and public health.

ID: 257 / **CP10.1: 3**
Contributed abstract
Conference Topics: Biodiversity, Microscopy, Wildlife parasitology
Keywords: Paramphistome, macropods, microCT, mitochondrial genomes, ribosomal DNA

Integrative taxonomy of *Gemellicotyle* sp. nov. (Digenea: Paramphistomidae) from the western grey kangaroo: combining histological, microCT imaging, and molecular data

Tanapan Sukee[1], Ian Beveridge[1], Sunita B. Sumanam[1], Jay Black[2], David Blair[3], Anson V. Koehler[1], Robin B. Gasser[1], Neil D. Young[1]

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Integrating traditional morphology with three-dimensional imaging and genetic data supports robust species hypotheses. The paramphistomoid fauna of Australian macropodids is poorly characterised, with two recognised genera, *Gemellicotyle* and *Macropotrema*, each containing a single species. Their conical form hampers morphological study, and no molecular data exists, making them ideal candidates for integrative taxonomy. This study describes *Gemellicotyle* sp. nov. from the caecum of the western grey kangaroo, *Macropus fuliginosus*. Whole-mount microscopy, serial histology, and X-ray microcomputed tomography (microCT) were combined to generate 3D reconstructions and printed models, enabling characterisation of internal and external morphology. These datasets were integrated with mitochondrial genome and nuclear ribosomal sequence data obtained using long-read sequencing. *Gemellicotyle* sp. nov. differs from *G. wallabicola* by lacking a central acetabular protuberance, having simple rather than sinuous caeca, and possessing more extensive vitellaria. MicroCT reconstructions and 3D models revealed 100 acetabular projections composed of radial muscle fibres, and a complex of lymphatic channels surrounding the acetabulum. This integrative approach refines species delineation and uncovers new knowledge on an Australian endemic paramphistome that depend on freshwater snail intermediate hosts to complete its lifecycle. This species may already be extinct due to changes in climate and habitats.

CP9: Vaccines 15 min talk sponsored by Institute for Biomedicine and Glycomics, Griffith University

Time: Wednesday, 01/July/2026: 11:20am - 11:35am · *Location:* Lecture Theatre 2
Session Chair: Danielle Stanisic, Institute for Biomedicine and Glycomics, Griffith University
Session Chair: Anouschka Akerman, The University of Queensland

ID: 211 / **CP9: 1**
Contributed abstract
Conference Topics: Immunology, Malaria, Vaccines
Keywords: Malaria, Vaccines, T-cells, Liver-stage

Genome-based, human-informed development of a T cell-based vaccine against malaria

Anouschka Akerman[1], Maggie King[1], Jonathan Tan[1], Mengistu Seid[2], Ashton M Kelly[1], Yomani Sarathkumara[1], Brenna Daily[1], Daniel J Browne[2], Jamie L Brady[2], David J Pattinson[2], Sarah Draper[3], Lauren Holz[4], Ian F Hermans[5], William R Heath[4], Gavin F Painter[3], Carla Proietti[1], Denise L Doolan[1]

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Malaria remains a major global health challenge. Current vaccines RTS,S and R21 targeting the surface circumsporozoite protein provide limited and strain-specific protection, highlighting the need for durable, cross-species protection. Robust T-cell-mediated responses against conserved liver-stage proteins offer strong potential for cross-species protection, but optimal targets are unknown. We have pursued a genome-based, human-informed approach to rational malaria vaccine design by profiling T-cell and antibody reactivity against the complete *P. falciparum* proteome in malaria-immune individuals. We identified distinct, largely non-overlapping repertoires of T-cell and antibody targets. The most immunodominant T-cell antigens exhibit minimal antibody activity and higher conservation across species, supporting the potential of T-cell targets for cross-species protection. These data support a new paradigm for development of a vaccine to prevent infection and disease.

We prioritized 25 T-cell targets for functional evaluation of immunogenicity and protective capacity in a murine malaria model, using liver-targeted mRNA or DNA-adenovirus platforms. Lead candidates all induced robust liver-resident memory T-cells, with antigen- and platform-specific immune profiles, and conferred protection against *P. yoelii* sporozoite challenge, reducing both liver- and blood-stage parasitemia. These results validate our genome-based vaccine design, focused on T-cell antigens and provide validated candidates for a next-generation, cross-species malaria vaccine targeting liver stage.

CP9.1: Vaccines 10 min talks sponsored by Institute for Biomedicine and Glycomics, Griffith University

Time: Wednesday, 01/July/2026: 11:35am - 12:05pm · *Location:* Lecture Theatre 2
Session Chair: Danielle Stanisic, Institute for Biomedicine and Glycomics, Griffith University
Session Chair: Anouschka Akerman, The University of Queensland

ID: 199 / **CP9.1: 1**
Contributed abstract
Conference Topics: Helminthology, Immunology, Vaccines
Keywords: Human hookworm, subunit vaccine

Rational discovery of a novel human hookworm subunit vaccine

Eti Sarkar, Suchandan Sikder, Connor McHugh, Paul Giacomin, Alex Loukas
James Cook University, Australia

Human hookworm infection, primarily caused by Necator americanus, remains a major global health burden, with vaccine development limited by insufficient identification of protective antigens. This study utilised a proteome microarray of recombinant secreted proteins from adult N. americanus, screened with sera from individuals vaccinated with irradiated third-stage larvae (iL3), to identify immunodominant targets. Selected antigens were characterised and their orthologs identified in the rodent hookworm Nippostrongylus brasiliensis.

Recombinant orthologs were evaluated in a mouse challenge model. One antigen, NBR17057 (ortholog of Na_11335), conferred significant protection, reducing intestinal worm burden and faecal egg output ($p < 0.05$) and inducing strong antigen-specific IgG responses. These findings support Na_11335 as a promising subunit vaccine candidate targeting the infective larval stage. Additional L3-stage antigens are being investigated to discover an effective novel human vaccine.

ID: 182 / **CP9.1: 2**
Contributed abstract
Conference Topics: Bioinformatics, Genomics, Immunology, Malaria, Protozoa, Vaccines
Keywords: Plasmodium falciparum, vaccine, multi-stage, CSP, RH5

Title: A population genetics view of a multi-stage malaria vaccine candidate

Edith Spiers, Mun Hua Tan, Karen Day
University of Melbourne, Australia

Multi-stage, multi-antigen vaccination against malaria is a favoured strategy to improve on the modest efficacy offered by existing vaccines. However, current multi-stage vaccine design neglects the extensive antigenic diversity which exists within populations of *Plasmodium falciparum* circulating in endemic areas, which has contributed to the low efficacy of multiple single-stage candidates. Using parasite genomes from the MalariaGEN Pf8 release, we combined population genetics with immunoinformatics to assess the potential for vaccine escape of the pre-erythrocytic and blood-stage targets CSP and RH5. We found that the proportion of parasites in sub-Saharan Africa encoding both vaccine-matched alleles for these targets was very low. When we predicted the effects of observed amino acid substitutions on antibody and HLA binding, we found no impact on the RH5 component of the vaccine. However, the majority of parasites sampled had the potential to escape both anti-CSP C-terminal antibodies and vaccine-induced CD4+ T-cell memory responses. We conclude that CSP-based vaccines as components of multi-stage vaccines may not provide the advantage of increased efficacy due to pre-existing polymorphisms in the C-terminal of CSP.

ID: 208 / **CP9.1: 3**
Contributed abstract
Conference Topics: Malaria, Vaccines
Keywords: Whole blood-stage, CAF01-adjuvanted, malaria, vaccine, P. yoelii

Impact of malaria infection on priming and boosting immunity induced by a CAF01-adjuvanted whole parasite *P. yoelii* blood-stage malaria vaccine

John Ategeka, Mark Burgess, Hamidreza Sadegh, Guilherme de Souza, Danielle I. Stanisic, Michael F. Good
Institute for Biomedicine and Glycomics, Griffith University, Australia

Introduction: Malaria remains a significant health problem. Globally, 282 million malaria cases and 610,000 malaria deaths occurred in 2024. The WHO recommends RTS,S/AS01 and R21-Matrix-M pre-erythrocytic vaccines to prevent malaria in children <5 yrs in moderate-to-high transmission settings. These vaccines are only partially effective. Evidence suggests that pre-erythrocytic vaccines are not boosted by natural malaria infection. We therefore assessed whether a whole-parasite blood-stage vaccine is more efficient in the presence of a malaria infection.

Methods: Different groups of mice received a controlled malaria infection with *P. yoelii* before or after vaccination. Mice were vaccinated with three doses of either killed 10^5, 10^6 or 10^7 *P. yoelii* pRBCs formulated with the liposomal adjuvant, CAF01. Four weeks after the final vaccine dose or controlled malaria infection, mice from each group were challenged with homologous parasites.

Results: A priming malaria infection did not provide additional protection against parasitaemia to mice that received 10^7 or 10^6 *P. yoelii* pRBCs in the vaccine; however, it provided additional protection to those vaccinated with a lower dose. Boosting vaccination with malaria infection enhanced control of parasitaemia in all vaccinated groups. Reducing parasite vaccine doses in the presence of infection would lower the cost of vaccinating populations.

CP10.2: Wildlife 2: Mammals, Birds, Lizards & Wetas 5 min talks

Time: Wednesday, 01/July/2026: 11:45am - 12:15pm · *Location:* Lecture Theatre 3
Session Chair: Haylee Crawford-Weaver, DCCEEW
Session Chair: Nicholas Fountain-Jones, University of Tasmania

ID: 144 / **CP10.2: 1**
Contributed abstract
Conference Topics: Ectoparasites, Host-parasite interactions, Wildlife parasitology
Keywords: Molecular phlyogeny, Amblyomma, Cernyomma

Molecualar phylogeny of Australian Amblyomma with a focus on subgenus Cernyomma

Mingeun Cho
The University of Queensland, Australia

The genus Amblyomma is a monophyletic lineage of hard ticks, many species of which parasitize reptiles. The last taxonomic revision of the Amblyomma of Australia was by the great Bob Roberts of Brisbane in 1970. That revision relied solely on morphology. I will present the first molecular phylogenetic tree of the Amblyomma of Australia, with emphasis on the subgenus Cernyomma. My phylogenetic analyses revealed some cryptic species and new host-associations. Additionally, I will present a new diagnostic key of them.

ID: 175 / **CP10.2: 2**
Contributed abstract
Conference Topics: Ecology, Invasive Species, Protozoa, Wildlife parasitology
Keywords: Sarcocystidae, Wildlife, Invasives

Sarcocystidae parasites in Australian invasive mammals: insights from DNA sequencing

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Australia is home to a diverse array of invasive mammals, including pigs, deer, cats, foxes, and rabbits. Although these five species have been reported as hosts to protozoan parasites of the family Sarcocystidae in their native ranges, there has been little investigation into the parasite load of these invasive species in Australia. This study aims to fill in some of this gap by studying species from 3 genera within the Sarcocystidae in selected invasive mammalian hosts. Tissues were collected through opportunistic sampling of deceased invasive mammal species around south-eastern Australia and underwent molecular analysis through PCR and DNA sequencing. This has resulted in species from the genera *Toxoplasma* and *Sarcocystis* being identified in invasive mammals. From these identifications a better understanding of the indirect impact invasive animal hosts may be having on an environment through the transmission of parasites through ecosystems, can be drawn. This includes possible spillover into native wildlife hosts which may affect efforts for conservation and relocations. As well as this, spillover into domestic hosts, such as in farming, can affect yields, profits, and risk human health, or represent a risk to household pets.

ID: 268 / **CP10.2: 3**
Contributed abstract
Conference Topics: Ectoparasites, Immunology
Keywords: adaptive humoral immunity, Ixodes holocyclus

Omics investigation of bandicoot immunity to the eastern paralysis tick, Ixodes holocyclus.

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[1]School of Chemistry and Molecular Biosciences (SCMB), The University of Queensland, Australia.; [2]Institute for Molecular Bioscience (IMB), The University of Queensland, Australia.

The eastern paralysis tick, *Ixodes holocyclus*, causes potentially fatal toxin-mediated paralysis in susceptible domestic animals, livestock, and humans, often requiring urgent medical or veterinary intervention. Yet bandicoots are major natural hosts and appear comparatively tolerant of tick infestation. The mechanism underlying this host difference remains unresolved. This project investigates whether adaptive humoral immunity may contribute to bandicoot protection against the eastern paralysis tick. We are using a comparative omics approach to identify and annotate immunoglobulin-related sequences in Queensland bandicoots, focusing on *Isoodon macrourus* and *Perameles nasuta*, with *Perameles gunnii* included as a closely related reference species. Publicly available bandicoot expressed sequence tags, and the available *P. nasuta* transcriptome are being analyzed alongside collaborator-supplied *P. gunnii* genomic data and marsupial immunoglobulin datasets using homology-based annotation. Blood-derived samples from tick-infested *I. macrourus* and *P. nasuta* have also been submitted for whole-genome sequencing by Pacific Biosciences high-fidelity long-read sequencing and Hi-C scaffolding to improve recovery of immune loci, including antibody regions. Peripheral blood RNA sequencing will further support identification of expressed immunoglobulin transcripts. Annotated candidate sequences will guide degenerate primer design for future amplification of bandicoot antibody regions and test whether humoral immune responses contribute to natural tolerance of *I. holocyclus*.

ID: 259 / **CP10.2: 4**
Contributed abstract
Conference Topics: Biodiversity, Ecology, Ectoparasites, Helminthology, Invasive Species, One Health, Wildlife parasitology
Keywords: Necropsy, opportunistic, Malleefowl, wildlife, vulnerable

Fowl play in the Fitz-Stirling: What a mallee fowl revealed after death

Narelle A Dybing[1], Natalie K Grassi[2]

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Opportunistic sampling provides a valuable avenue for understanding the health and disease status of wildlife that are otherwise difficult to study. We report findings from a necropsy conducted on a road-killed Malleefowl (*Leipoa ocellata*) collected in the Fitz-Stirling region of Western Australia during a broader dietary ecology project. Although the original study did not target disease, the carcass presented a rare opportunity to conduct to investigate both ectoparasite and endoparasite communities in this cryptic and nationally vulnerable species. Few parasitological studies have been conducted on this vulnerable species and those that exist are dated. With increasing human encroachment into wildlife habitats and the spread of invasive species, understanding parasite loads and associated risks is becoming increasingly important. This is particularly relevant for Malleefowl, whose ground dwelling and mound building behaviours may increase exposure to soil dwelling parasites, and whose declining populations mean biological samples are rarely available. As many native species face pressure from habitat loss and fragmentation, climate change, and emerging diseases, integrating opportunistic necropsies into ecological and conservation programs can substantially enhance surveillance capacity. This case study underscores the importance of maximising information gained from unexpected wildlife encounters, including road-kill specimens, to better inform management and conservation strategies.

ID: 261 / **CP10.2: 5**
Contributed abstract
Conference Topics: Apicomplexa Biology, Helminthology, Host-parasite interactions, One Health, Wildlife parasitology
Keywords: one health, sea lion, hookworm

From land to the sea: First detection of *Neospora caninum* in an Australian marine mammal, *Neophoca cinerea* (Australian Sea Lion)

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[1]School of Natural Sciences, Faculty of Science and Engineering, Macquarie University, Sydney, Australia; [2]Sydney School of Veterinary Science, Faculty of Science, The University of Sydney, Camperdown, NSW, Australia

Parasitism poses significant health challenges for the endangered Australian sea lion (*Neophoca cinerea*). Australian sea lion pups experience a high prevalence of the endemic hookworm (*Uncinaria sanguinis*) which is associated with reduced pup body condition and increased mortality. We examined faecal DNA from Australian sea lion pups (n = 63) to further explore host parasite interactions with an emphasis on apicomplexan parasitism. Screening of samples using an Apicomplexan genera PCR targeting the 18S rDNA gene resulted in 51 of the 63 DNA positive samples. DNA sequencing revealed a single parsasite sequence with high similarity between samples from Australian sea lion pups (99.9%) across 900 basepairs. A BlastN Seach identified the sequence from Australian sea lion pups to be 99.7% similar to *Neopsora caninum.* This first report of *N. caninum* in Australian sea lion pups demonstrates the environmental continuum that connects parasites associated with terrestrial animals to marine wildlife, and the significance of One Health for wild marine species. Understanding of the impact of *N. caninum*, and the significance of co-infection with hookworm, for pup health is important for Autstralian sea lion conservation and future management of this endangered marine mammal.

ID: 112 / **CP10.2: 6**
Contributed abstract
Conference Topics: Ectoparasites, Veterinary Parasitology, Wildlife parasitology
Keywords: Marine mammals, Halarachnidae, Respiratory mites, Captive animal

Detection of *Orthohalarachne attenuata* (Acari: Halarachnidae) in captive Pacific walrus (*Odobenus orsmarus divergens*)

Di Barton[1], Chris Perkins[2], Paolo Martelli[2], Natalie Jefferson[1], Shokoofeh Shamsi[1]
[1]Charles Sturt University, Australia; [2]Ocean Park Corporation, Hong Kong SAR

Nasal mites of the family Halarachnidae are common parasites of pinnipeds, although rarely reported in walruses. Two captive walruses at the Ocean Park Aquarium, Hong Kong, were found to be infected with nasal mites identified as *Orthohalarachne attenuata* (Banks, 1910). Long-term, low-dose ivermectin treatment for heartworm prevention (0.01 mg/kg) had no effect on parasite burden. In contrast, anti-parasitic doses of ivermectin (0.2mg/kg), administered repeatedly over a short-term period (4 doses, 2 weeks apart, followed by 3 doses administered monthly), appeared to successfully clear the parasitic infection, with no signs of re-infection observed for over 12 months post-treatment.

CP8.1: Drugs and Drug Resistance 1 - 5 min talks

Time: Wednesday, 01/July/2026: 11:50am - 12:15pm · *Location:* Lecture Theatre 1
Session Chair: Christopher Hart, Griffith University
Session Chair: Hannah Smith, Griffith University

ID: 137 / **CP8.1: 1**
Contributed abstract
Conference Topics: Drugs, Molecular Biology, Protozoa
Keywords: Giardia, drug discovery, mode of action

Defining the target of potent and selective drug-leads in *Giardia duodenalis*

Keely Fayd'Herbe[1,2], Christopher Hart[1], Andrew Riches[3], Ghizal Siddiqui[4], Jack Ryan[3], Tina Skinner-Adams[1,2]
[1]Institute for Biomedicine and Glycomics, Griffith University, Nathan, Brisbane, Queensland 4111, Australia; [2]School of Environment and Science, Griffith University, Nathan, Brisbane, Queensland 4111, Australia; [3]Commonwealth Scientific and Industrial Research Organization, Biomedical Manufacturing, Clayton, Victoria 3168, Australia; [4]Monash Proteomics & Metabolomics Platform, Monash University, VIC, Australia

Giardia duodenalis is a parasitic protist and the causative agent of giardiasis, a diarrhoeal disease that infects approximately one billion people annually and results in over 300 million cases of acute or chronic illness. Clinical presentations range from self-limiting disease to persistent and debilitating symptoms. Current treatment relies on a limited number of drugs from few chemical classes, many of which suffer from significant drawbacks, including prolonged treatment regimens, variable efficacy, severe side effects, and reduced effectiveness due to emerging drug resistance. To address these limitations, our team has developed a series of novel antiparasitic compounds, including lead candidates with substantially improved efficacy and selectivity compared with existing therapies.

While the mode of action of these compounds remains unclear, untargeted proteomic and drug-combination studies suggest that they may act on the parasite cytoskeleton or phosphorylation-based signalling machinery via previously unexplored mechanisms. These findings, along with ongoing efforts to define the mode of action of this compound series, will be discussed.

ID: 158 / **CP8.1: 2**
Contributed abstract
Conference Topics: Apicomplexa Biology, Biochemistry, Cell Biology, Drugs, Malaria
Keywords: Pantothenate, Coenzyme A, Drug Target, Metabolism

Investigating PPCS as a Novel Antimalarial Drug Target in *Plasmodium falciparum*

Eva El Hassan[1], Weronika Szablowska[1], Lara E. Skibbie[2], Xiangning Liu[1], Qiuyuan Li[1], Henry D. Schrecker[2], Rory C. Smith[2], Cynthia S. Dowd[2], Kevin J. Saliba[1]
[1]Australian National University, Australia; [2]George Washington University

Malaria caused by *Plasmodium falciparum* remains a major global health challenge, with increasing resistance to frontline treatments highlighting an urgent need for new antimalarial therapies. The coenzyme A (CoA) biosynthesis pathway of *P. falciparum* represents a promising antimalarial drug target. Phosphopantothenoylcysteine synthetase (PPCS), the second enzyme in the pathway, is of particular interest due to its role as a flux-control step. To identify inhibitors of *Pf*PPCS, we have screened ~50 compounds initially designed to inhibit the bacterial PPCS. RCS-33 and HDS-44 emerged as promising candidates, with antiplasmodial IC_{50} values of ~1 µM. Although the activity of the compounds can be somewhat modulated by overexpression of *Pf*PPCS, consistent with them being on target, their mechanism of action remains to be confirmed. We are currently generating RCS-33- and HDS-44-resistant parasites to try and identify the target *via* whole-genome sequencing of resistant parasites. We will also use an enzyme assay and purified *Pf*PPCS to test directly whether the compounds are able to inhibit *Pf*PPCS activity.

ID: 173 / **CP8.1: 3**
Contributed abstract
Conference Topics: Malaria
Keywords: repurposing, target, -omics, drug development

Mechanistic insights into repurposed compounds as potential antimalarials

Chris Taylor, Carlo Giannangelo, Darren Creek
Monash Uni, Australia

Malaria remains a leading cause of morbidity and mortality, with 608,000 deaths reported in 2022, predominantly among children under five in sub-Saharan Africa. The disease, predominantly caused by Plasmodium falciparum, faces escalating challenges due to the emergence of strains resistant to all frontline antimalarials, including artemisinins. This highlights an urgent need for new therapeutic strategies. While de novo drug discovery is slow and costly, repurposing existing compounds offers a rapid and cost-effective alternative.
We screened the Structural Genomics Consortium's Donated Chemical Probes Library, which consists of compounds originally developed for human diseases, to identify candidates with antimalarial potential. From 200 compounds, 11 demonstrated sub-micromolar potency against the P. falciparum Pf3D7 reference strain and retained activity across five resistant lines. Their equipotent profiles suggest novel mechanisms of action distinct from existing drug classes. In vitro pulse activity assays further characterized compound speed-of-action and asexual stage-specific antimalarial activity.
Future work will employ unbiased multi-omics approaches, including as untargeted metabolomics, solvent proteome profiling and limited-proteolysis mass spectrometry, to elucidate molecular targets and validate these candidates as starting points for drug development. These findings highlight the potential of compound repurposing to accelerate antimalarial discovery and identify new druggable pathways to combat resistance.

ID: 126 / **CP8.1: 4**
Contributed abstract
Conference Topics: Biochemistry, Cell Biology, Drugs, Host-parasite interactions, Malaria, Molecular Biology, Protozoa, Proteomics
Keywords: Plasmodium falciparum, host signalling pathway, anticancer drugs, antiplasmodal, drug-resistance

Repurposing anticancer drugs as antimalarials

Kwong Sum Lam[1], Jack Adderley[2], Christian Doerig[2], Alexander G. Maier[1]
[1]Australian National University; [2]RMIT University

The rapid emergence of drug-resistant *Plasmodium falciparum* necessitates novel antimalarial strategies. Because parasite survival relies on extensive modification of host erythrocytes, host-directed therapies represent an attractive approach. Here, we evaluated a panel of anticancer kinase inhibitors as potential antimalarials, initially hypothesising that they act through inhibition of host signalling pathways. We focused on the proposed requirement for activation of the human MAPK pathway in infected erythrocytes for parasite proliferation. The MAPK pathway tightly regulates cellular proliferation and survival, and its dysregulation in cancer has driven the development of numerous selective kinase inhibitors. Several compounds, displayed submicromolar potency against both asexual erythrocytic-stage parasites and sexual gametocytes, indicating potential for dual curative and transmission-blocking efficacy. However, detailed mechanistic studies of MEK1 inhibitors provided little evidence for host-directed activity. Overexpression of human MEK1 in infected erythrocytes did not alter drug potency, parasites exposed continuously to one MEK1 inhibitor rapidly evolved resistance, and another inhibitor exhibited marked strain-dependent activity across *P. falciparum* isolates. These observations support a parasite-directed mode of action. Collectively, our findings demonstrate that human MEK1 is not essential for parasite proliferation. Despite host toxicity, parasite-selective activity provides a platform for designing kinase inhibitor derivatives.

ID: 140 / **CP8.1: 5**
Contributed abstract
Conference Topics: Veterinary Parasitology
Keywords: Goat; anthelmintic resistance; veterinarians; parasite control practices; Sri Lanka

A questionnaire survey of veterinarians reveals gaps in parasite control practices contributing to anthelmintic resistance in goats in Sri Lanka

Suraj Walimunige[1], Dulari Thilakarathne[2], Ghazanfar Abbas[1], Anil Kalupahana[2], Jayanthe Rajapakshe[2], Robin Gasser[1], Abdul Jabbar[1]
[1]University of Melbourne, Australia; [2]University of Peradeniya, Sri Lanka

Gastrointestinal parasitism and inappropriate anthelmintic use are major constraints to goat production and key drivers of anthelmintic resistance (AR), particularly in low- and middle-income countries. This study evaluated parasite control practices recommended by veterinarians to goat farmers in Sri Lanka to identify critical gaps contributing to AR. An online questionnaire was distributed to 877 veterinarians following a pilot survey, and 122 completed responses (14%) were analysed. Data were collected on respondent demographics, knowledge of gastrointestinal helminths, diagnostic and control practices, anthelmintic use and AR awareness. Although respondents demonstrated sound knowledge (median confidence score: 70; mean: 65), diagnostic approaches relied on clinical signs (35%) and visual observation of worms in faeces (22%), with limited use of faecal egg counts (FEC). Limited diagnostic access (39%) and low farmer engagement (36%) were key barriers to recommending FEC. Albendazole, levamisole, and ivermectin were widely recommended; however, 41% of veterinarians estimated doses visually, increasing under-dosing risk. While 92% recognised AR as a major concern, 71% had never performed faecal egg count reduction tests. Multiple correspondence analysis identified distinct clusters of practices, separating experience-based from evidence-based approaches. These findings highlight critical gaps and the need for improved diagnostics, targeted training, farmer engagement, and national AR surveillance.

CP9.2: Vaccines 5 min talks sponsored by Institute for Biomedicine and Glycomics, Griffith University

Time: Wednesday, 01/July/2026: 12:05pm - 12:15pm · *Location:* Lecture Theatre 2
Session Chair: Danielle Stanisic, Institute for Biomedicine and Glycomics, Griffith University
Session Chair: Anouschka Akerman, The University of Queensland

ID: 145 / CP9.2: 1
Contributed abstract
Conference Topics: Malaria, Vaccines
Keywords: Malaria, multi-stage, vaccine, antigen, density

Enhancing antigen density on nanoparticle platforms to enable multi-stage malaria vaccines

Anne Nguyen[1], Jaanaky Vigneswaran[1], Daniel Luque[1,2], Michael Johnson[1], Jake Baum[1]
[1]School of Biomedical Sciences, University of New South Wales; [2]Electron Microscopy Unit , University of New South Wales

Malaria remains a major global health challenge, with substantial morbidity and mortality despite ongoing control efforts. Current vaccines targeting the pre-erythrocytic stage provide only partial and waning protection, highlighting the need for improved strategies. One promising approach is the development of multi-stage vaccines that target different stages of the parasite lifecycle to enhance overall efficacy and durability. Virus-like particles (VLPs) provide an attractive platform for vaccine design due to their ability to present antigens in a highly repetitive and ordered manner, thereby promoting robust immune responses. Increasing evidence suggests that the density of antigens on nanoparticle platforms plays a critical role in shaping immunogenicity. In this study, we explored engineering approaches to optimise antigen display on VLPs and systematically modulate antigen density. We demonstrate that engineered VLPs can maintain structural integrity while accommodating varying levels of antigen presentation. Importantly, increasing antigen density was associated with enhanced antibody responses, supporting the concept that antigen valency is a key determinant of immunogenicity. These findings provide a foundation for the rational design of next-generation nanoparticle vaccines including multi-stage/multi-antigen designs that are currently in progress in our lab.

ID: 165 / CP9.2: 2
Contributed abstract
Conference Topics: Immunology, Malaria, Vaccines
Keywords: Malaria, Vaccine, Blood-stage, Perforin

Investigation of perforin inhibition on malaria disease progression during *Plasmodium yoelii* blood-stage infection.

Hamidreza Sadegh[1], Mark Burgess[1], Skye Van Esch[1], Joe Trapani[2], Michael Good[1], Danielle Stanisic[1]
[1]Institute for Biomedicine and Glycomics, Griffith University, Southport, Australia; [2]Peter MacCallum Cancer Centre, Melbourne, Victoria, Australia

Malaria remains a major global health challenge due to the complexity of the *Plasmodium* life cycle, the partial efficacy of current vaccines, and disease severity driven by both parasite factors and host immune responses, including perforin-mediated cytotoxicity. We investigated the impact of treatment timing with a perforin inhibitory compound on disease progression in vaccinated and unvaccinated mice infected with *Plasmodium yoelii*. Vaccinated mice received killed, whole *P. yoelii* blood-stage parasites formulated with liposomes and the TLR4 agonist 3D-(6-acyl)-PHAD. Mice were challenged with 10^5 *P. yoelii* parasitised red blood cells and treated with a perforin inhibitory compound or vehicle control using two regimens: early (days -1 to 3 post-infection) or late (days 4 to 8 post-infection). Mice were monitored over 26 days. Early perforin inhibitory treatment showed a trend towards reduced parasitaemia and clinical severity; however, these changes were not statistically significant compared with the control groups in either vaccinated or unvaccinated mice. Notably, unvaccinated mice treated with the perforin inhibitory compound exhibited a 6–8 day extension in survival compared to controls, regardless of early or late treatment, although all mice ultimately succumbed to infection. These findings suggest perforin-mediated cytotoxicity contributes to malaria pathology and may be a therapeutic target.

S5: Symposium Ticks, Mites, kissing bugs

Time: Wednesday, 01/July/2026: 1:30pm - 1:50pm · *Location:* Lecture Theatre 3
Session Chair: Katja Fischer, QIMR Berghofer
Session Chair: Xavier Barton, Murdoch University

ID: 269 / **S5: 1**
Invited speaker abstract

Scabies Diagnostics: Past, present and future

Deepani D Fernando[1], Grant Hansman[2], Katja Fischer[1]
[1]Infection and Inflammation Program, QIMR Berghofer, Brisbane, Australia; [2]Institute for Glycomics, Griffith University, Gold Coast, Australia

Scabies is a common, debilitating neglected tropical disease that disproportionately affects overcrowded and resource-limited populations worldwide. Beyond the primary infestation, secondary bacterial infections can lead to severe complications, including sepsis, glomerulonephritis, and rheumatic fever and heart disease, contributing to significant morbidity and mortality. Early and accurate diagnosis is therefore critical for effective disease control.

Historically, scabies diagnosis has relied on the direct visualisation of mites, with microscopic examination of skin scrapings remaining the most widely used confirmatory method. More recently, non-invasive imaging techniques such as dermatoscopy, videomicroscopy, and reflectance confocal microscopy have improved diagnostic capability; however, their accessibility remains limited. To date, no molecular or serological diagnostic test has been successfully translated into routine clinical use. PCR- and ELISA-based approaches have shown promise but are constrained by their reliance on specialised equipment, cost, processing time, and technical expertise. While the International Alliance for the Control of Scabies has introduced standardised diagnostic criteria, a major gap persists in the availability of practical, point-of-care tools for near-patient diagnosis.

To address this unmet need, our work focuses on developing next-generation point-of-care diagnostics for scabies. Using proteomic approaches, we have identified highly abundant, scabies-specific faecal proteins as novel antigenic biomarkers. Our translational pipeline includes recombinant antigen production, monoclonal antibody and nanobody generation, and the development of ELISA- and lateral flow assays targeting both mite-derived proteins and host immune responses. These platforms are being validated through preclinical studies in a porcine scabies model and multicentre clinical trials in collaboration with industry and international partners.

CP11: Cells, Molecules & Genes 2 - 10 min talks

Time: Wednesday, 01/July/2026: 1:30pm - 2:30pm · *Location:* Lecture Theatre 1
Session Chair: Ellis Joch, Griffith University
Session Chair: Wisam Dawood, Griffith University

ID: 232 / **CP11: 1**
Contributed abstract
Conference Topics: Apicomplexa Biology, Cell Biology, Malaria, Microscopy, Protozoa
Keywords: Plasmodium, Cell Biology, Microscopy

Investigating apical-basal polarity establishment in malaria parasites.

Benjamin Liffner[1], Thiago Luiz Alves e Silva[2], Inês Bento[3], Joel Vega-Rodriguez[2], Maria Mota[3], Sabrina Absalon[4]
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Almost all cells have a shape, size, and organisation specialised for their functions. During replication, malaria parasites are amorphous and disorganised. By contrast, their host-cell-invading ‘zoite’ stages exhibit extreme apical-basal polarity, with invasion-specialised organelles at their apical end. It is currently unclear when this transition begins, how it is initiated, or what proteins control it.

Using ultrastructure-expansion microscopy, we imaged blood-stage, liver-stage, and mosquito-stage malaria parasites from replication until the completion of zoite formation to identify how this disorganised to hyper-polarised transition occurs. In all three lifecycle stages, the first sign of polarity establishment in the parasite was the anchoring of a structure called the centriolar plaque to the parasite plasma membrane. Subsequently, the parasite would begin to build its invasion-specialised organelles at this site, suggesting that this anchoring event represents the establishment of apical-basal polarity. In blood-stage parasites we observed a dramatic repositioning of the Golgi following centriolar plaque anchoring, providing a potential mechanism for how this event triggers apical organelle biogenesis. Work is currently ongoing to define the centriolar plaque proteins that coordinate the establishment of polarity within the parasite. Collectively, this study provides new insights into the biology of daughter cell formation in malaria parasites.

ID: 135 / **CP11: 2**
Contributed abstract
Conference Topics: Apicomplexa Biology, Biochemistry, Malaria, Molecular Biology
Keywords: Apicoplast, transporter, Plasmodium falciparum

Two novel apicoplast transporters with different, crucial roles in malaria parasite life cycle

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Malaria parasites depend on the apicoplast, an intriguing organelle of algal origin, for survival throughout the life cycle. Transport of metabolites across the apicoplast membranes is poorly understood, and only 11 transporter proteins have been confirmed to localize to the organelle to date. We report apicoplast localization of two previously uncharacterized transporters in *Plasmodium falciparum*. Knockdown of apicoplast transporter 1 (*at1*) resulted in death of asexual blood-stage parasites. Knockout of *at1* in PfMev parasites, which have a metabolic apicoplast bypass, resulted in disruption of apicoplast morphology and loss of the organellar genome, suggesting that AT1 is involved in apicoplast housekeeping. Knockout of apicoplast transporter 2 (*at2*) did not affect asexual blood-stage parasites, nor gametocyte and gamete formation. In the mosquito, however, oocyst size was significantly decreased and no sporozoites were observed in salivary glands up until day 21, phenocopying knockouts of fatty acid metabolism. Metabolomics, drug assays, transport assays in yeast, and protein modeling provided further information on candidate substrates for both transporters. Taken together, we identified two novel apicoplast transporters, with AT1 being essential for asexual blood stages by supporting apicoplast housekeeping, and AT2 being important for parasite growth in mosquitoes, possibly by facilitating fatty acid metabolism.

ID: 185 / **CP11: 3**
Contributed abstract
Conference Topics: Apicomplexa Biology, Biochemistry, Malaria
Keywords: Type IV P-type ATPase, PfATP2, flippase, Plasmodium falciprum

PfATP2 drives phosphatidylserine flipping and modulates antimalarial sensitivity in *Plasmodium falciparum*

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Type IV P-type ATPases (P4-ATPases) are critical regulators of membrane lipid asymmetry in eukaryotic cells. *Plasmodium falciparum* is predicted to encode six P4-ATPases, but their roles remain to be defined. Recently, amplification of the gene encoding one of these, PfATP2, was associated with resistance to the antiplasmodial compounds MMV007224 and MMV665794. Here, we show that PfATP2 is a plasma membrane localised P4-ATPase that functions as a phospholipid flippase and is important for parasite growth. Using genetically modified parasites, we found that the PfATP2 expression level of parasites correlates with the rate by which they internalise a fluorescent analogue of phosphatidylserine (NBD-PS). Overexpression of PfATP2 enhanced NBD-PS translocation, whereas conditional knockdown significantly impaired this process. Further, exposure of parasites to MMV007224 and MMV665794 gave rise to a reduction in NBD-PS internalisation. PfATP2 knockdown parasites were hypersensitive to growth inhibition by MMV007224 and MMV665794, while PfATP2

overexpressing parasites were resistant to the compounds. Taken together, these findings establish PfATP2 as a major contributor to ATP-dependent phosphatidylserine internalisation on the parasite plasma membrane and a potential target of MMV007224 and MMV665794. We are currently investigating whether a reduction in PfATP2-mediated phospholipid flipping affects the activities of other transporters on the parasite plasma membrane.

ID: 203 / CP11: 4
Contributed abstract
Conference Topics: Cell Biology, Malaria, Microscopy, Molecular Biology
Keywords: P. falciparum, VDAC, mitochondria, target-discovery

The voltage dependent anion channel is a mitochondrial protein critical to the growth of *P. falciparum*

Mitchell Trickey[1,2,3], Joyanta Modak[2,3], Natalie Counihan[2,3], Mrittika Chowdury[2,3], Darren Creek[1], Tania de Koning-Ward[2,3]

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Despite global gains combating malaria, the increasing incidence of antimalarial drug resistance to front line therapeutics demands new drugs with novel targets be developed. Potential targets for the design of therapeutic drugs include channel proteins that are critical for the movement of essential cargo within the parasite. Here, the essentiality of the voltage dependent anion channel (VDAC) was investigated in the deadliest species of malaria *P. falciparum*, via protein knockdown and localisation studies, followed by mitochondrial drug sensitivity studies and metabolomic analysis. Knockdown of *vdac* led to a survival defect in the RBC stages. Furthermore, the failure to generate conventional knockouts indicated VDAC is essential for parasite survival. Immunofluorescent microscopy successfully localised VDAC to the mitochondria, while the knockdown of VDAC was shown to sensitise parasites to mitochondrial target drugs atovaquone and proguanil, providing further indication for a role at the parasite mitochondria. Analysis of the parasite metabolic profile following VDAC knockdown is currently being used to investigate a possible role in the pyrimidine biosynthesis pathway at the outer mitochondrial membrane. Whilst the precise role of VDAC at the mitochondria requires further investigation, this channel protein is an essential and unique target for the future design of novel antimalarial therapeutics.

ID: 220 / CP11: 5
Contributed abstract
Conference Topics: Malaria
Keywords: Transmission; Pfs16; oligomeric complex; Parasitophorous vacuole membrane

Pfs16 forms an oligomeric complex with a membrane-spanning pore in the malaria parasite parasitophorous vacuole membrane

Emmanuel Gyamfi[1], Grace Peters[1], Claire Sayers[1], Kate Michie[1], Stephen Fairweather[2], Giel Van Doreen[2], Jake Baum[1]

[1]UNSW Sydney, Australia; [2]Australian National University

Pfs16 is a 16 kDa protein expressed early in the process of gametocyte development in *Plasmodium falciparum*. It localises to the parasitophorous vacuole membrane (PVM) of gametocytes. Previous studies have focused exclusively on its monomeric form. However, AlphaFold modelling predicts that Pfs16 assembles into an oligomeric complex containing a membrane-spanning pore. Given the essential role of Pfs16 in parasite transmission, we aimed to characterise this oligomeric structure and its function, which could provide new insights for transmission-blocking strategies. A combination of molecular and structural biology techniques was employed, including chemical crosslinking, Western blotting, native gel electrophoresis, and surface biotinylation, to explore its oligomeric subunits. Its functional activity was characterised using electrophysiological analysis in the *Xenopus* oocyte expression system. Both *in silico* modelling and experimental data indicate that Pfs16 forms a pentameric complex. Electrophysiological analysis in *Xenopus* oocytes has demonstrated that the membrane-spanning pore exhibits ion-conducting activity, supporting the presence of a functional pore. These results provide evidence that Pfs16 assembles into an oligomeric, likely pentameric, ion channel. Given its essential role in gametocyte development and transmission, targeting this complex may represent a promising strategy for the development of transmission-blocking interventions.

ID: 212 / CP11: 6
Contributed abstract
Conference Topics: Apicomplexa Biology, Cell Biology
Keywords: Toxoplasma gondii, Amino acid, Transporter, Metabolic redundancy

Functional Redundancy Between Amino Acid Uptake and Biosynthesis in *Toxoplasma gondii*

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The intracellular apicomplexan parasite *Toxoplasma gondii* relies on both nutrient scavenging and biosynthetic pathways to acquire amino acids required for growth and survival. Our previous work identified the plasma membrane transporter *Tg*ApiAT2 as the primary glutamine transporter and, through a CRISPR-based screen, revealed that multiple amino acid biosynthetic pathways become fitness-conferring upon *Tg*ApiAT2 disruption, suggesting functional redundancy between uptake and synthesis. Here, we experimentally validate this model by generating double mutants lacking *Tg*ApiAT2 alongside key enzymes in amino acid biosynthesis. These mutants exhibit severe growth defects, demonstrating that parasites depend on compensatory mechanisms to maintain amino acid homeostasis. To further characterise *Tg*ApiAT2 function, we performed radiolabeled uptake assays, confirming that *Tg*ApiAT2 mediates the uptake of numerous non-essential amino acids. Together, our findings provide direct functional evidence for redundancy between amino acid uptake and synthesis in *T. gondii*, highlighting the metabolic flexibility that underpins parasite adaptation to variable host nutrient environments.

CP12: Sheep & Goats - 10 min talks

Time: Wednesday, 01/July/2026: 1:30pm - 2:30pm · *Location:* Lecture Theatre 2
Session Chair: Vern Bowles, The University of Melbourne
Session Chair: Nichola Calvani, The University of Sydney

ID: 228 / **CP12: 1**
Contributed abstract
Conference Topics: Fasciolosis/Liver fluke, Helminthology, Livestock Parasites, Zoonoses
Keywords: Liver Fluke, 3D Cell Culture, Confocal Imaging, Scanning Electron Microscopy

Growth and development of *Fasciola hepatica* on an *in-vitro* 3D cell culture model: how does it compare to *in-vivo*?

Hayley Martinez DeCristi[1], Michael Kuligowski[2], Katharine Muscat[1], Javier Gonzalez Miguel[3], Nichola Eliza Davies Calvani[1]
[1]University of Sydney, Sydney School of Veterinary Science, Australia; [2]Sydney Microscopy and Microanalysis, Australia; [3]Laboratory of Helminth Parasites of Zoonotic Importance, Institute of Natural Resources and Agrobiology of Salamanca, Spain

Background and Aims

Fasciola hepatica is a globally distributed helminth of importance to both human and animal health. Critical information on how *F. hepatica* interacts with its mammalian host is lacking due to a reliance on animal models. This study aimed to validate a recently-developed HepG2-derived 3D spheroid co-culture model for *F. hepatica* newly excysted juveniles (NEJ) against *in-vivo* infection, using advanced microscopy methods.

Methods

In-vivo samples from C57BL/6 mice, infected with 175 metacercariae each, and NEJ grown *in-vitro* were collected at 12, 48, 120, 144, 168, 180, and 192 hours post infection. Comparison of the growth and development of external morphological (spines, sensory papillae, suckers) and internal anatomical (musculature, gut and uterine development) features between the two culture conditions were made using fluorescent confocal and scanning electron microscopy.

Results

The data obtained provides the first detailed morphological comparison of the growth and development of *F. hepatica* NEJ cultured *in-vitro* and *in-vivo* and serves as a benchmark to enhance future models.

Conclusion

The progression of animal-free models will enable exploration of the intricacies of early infection. Future work will incorporate spatial transcriptomic analysis to elucidate temporal shifts in *F. hepatica* development and the subsequent discovery of drug and vaccine targets.

ID: 160 / **CP12: 2**
Contributed abstract
Conference Topics: Drugs, Fasciolosis/Liver fluke
Keywords: Fasciola hepatica, Miracidial motility assay (MMA), Newly excysted juveniles (NEJ), WMicrotracker ONE, Anthelmintics

A practical miracidial motility assay for assessing *Fasciola hepatica* sensitivity to compounds *in vitro*

Mengwei Zheng[1], Aya Taki[1], Tanapan Sukee[1], Jane Hodgkinson[2], Terry Spithill[3], Robin Gasser[1], Neil Young[1]
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Fasciola hepatica causes fasciolosis in livestock and humans worldwide, yet reliable tools to assess drug efficacy against the early developmental stages of this parasite are lacking. Here, we developed an automated miracidial motility assay (MMA) using the WMicroTracker ONE infrared detection system to quantify the sensitivity of *F. hepatica* miracidia to anthelmintic compounds including clorsulon (CLORS), closantel (CLOS), triclabendazole (TCBZ) and triclabendazole-sulphoxide (TCBZ-SO). Systematic optimisation of assay conditions, including inoculum size, observation window and solvent concentration yielded a reliable platform for evaluating the sensitivity of *F. hepatica* miracidia from diverse geographic isolates to these compounds. Our results demonstrated that three compounds (CLOS, TCBZ and TCBZ-SO) produced concentration-dependent motility inhibition, whereas CLORS had no effect. CLOS displayed the highest potency among isolates from New South Wales (NSW), Tasmania (TAS) and Victoria (VIC), whereas TCBZ and TCBZ-SO exhibited isolate-specific sensitivity patterns. Miracidial responses of the NSW and TAS isolates to TCBZ, TCBZ-SO and CLOS were also compared in vitro with those of newly excysted juveniles (NEJs) produced from the same isolates. Overall, the findings show that MMA provides a reproducible, host-independent and high-throughput phenotypic platform for assessing miracidial sensitivity to compounds.

ID: 163 / **CP12: 3**
Contributed abstract
Conference Topics: Fasciolosis/Liver fluke
Keywords: Climate change, fasciolosis, liver fluke, risk modelling, Australia

High-Resolution Climate Modelling of Fasciolosis Risk in Australia: A One Health Early-Warning Framework

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Fasciolosis, caused by the liver fluke *Fasciola hepatica*, is a climate-sensitive parasitic disease that threatens livestock productivity and farm profitability, with broader implications for food security, rural livelihoods, and sustainable food systems. This study aimed to quantify historical trends and project future fasciolosis risk across Victoria under changing climate conditions. High-resolution (5 km²) climate data were retrieved for 40,504 grid points across Victoria to validate a Growing Degree Days (GDD) model for estimating fasciolosis risk over the past 50 years (1975–2024) and to project future risk centred at 2050 and 2090 under medium (RCP 4.5) and high (RCP 8.5) representative concentration pathways. Linear regression analysis demonstrated a strong relationship between observed fasciolosis prevalence and modelled risk values ($R^2 = 0.94$, $p < 0.003$). Historical analyses revealed substantial interannual variability, with consistently higher risk in eastern Victoria, followed by western and northern regions. Under future climate scenarios, risk increased spatially by up to two-fold in Barwon and three-fold in the Great South Coast, particularly under the 2090 RCP 8.5 scenario. These findings provide spatially explicit evidence to support climate-responsive surveillance, risk-based control strategies, and integrated animal health and environmental policy development.

ID: 171 / CP12: 4
Contributed abstract
Conference Topics: Diagnostics, Livestock Parasites
Keywords: Larval Culture, Nemabiome deep amplicon sequencing, Victoria, Gastrointestinal nematodes

COMPARISON OF LARVAL CULTURE TO NEMABIOME DEEP-AMPLICON SEQUENCING

Rebecca Farnell[1], Steve Cotton[2], David Piedrafita[1], Christiane Bahlo[1], Sarah Preston[1]
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Gastrointestinal nematodes impose economic burdens on the sheep industry, impacting on animal welfare and management costs. Species surveillance is essential, yet larval culture is inefficient for large-scale monitoring. Nemabiome deep-amplicon sequencing allows for sensitive detection of nematode species directly from eggs without having to hatch eggs for 10-14 days like larval culture.

This study compared larval culture with nemabiome sequencing to speciate worms from bulk faecal samples from 27 farms. Spearman's correlations showed significant positive correlations for *Haemonchus contortus* (r_s=0.56, p<0.01), and *Teladorsagia circumcincta* (r_s=0.58, p <0.01) and a moderate positive correlation for *Trichostrongylus spp* (r_s=0.34, p=0.07). Positive correlations between the two techniques provides some confidence that nemabiome sequencing is useful for large-scale surveillance of nematode species across Victoria, which is being completed on saleyard samples collected four times a year for a two-year period.

Initial findings on 100 samples show that Victoria's most abundant species are *Teladorsagia circumcincta* (92%), *Trichostrongylus vitrinus* (78%) and *Haemonchus contortus* (54%). Given the high prevalence and pathogenicity of *T. vitrinus* compared to other *Trichostrongylus* species more research should focus on understanding this worm to enhance management strategies. Less common species included *Trichostrongylus colubriformis*, *Chabertia ovina*, *Trichostrongylus axei*, *Oesophagostomum venulosum, Nematodirus spathiger* and *Trichurus ovis*.

ID: 260 / CP12: 5
Contributed abstract
Conference Topics: Cell Biology, Host-parasite interactions, Immunology, Livestock Parasites, One Health, Vaccines
Keywords: Ovine

Mimicking Natural Immunity: Trickle Infection Induces Protective Responses to *Trichostrongylus colubriformis*

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Increased drench resistance in sheep parasite populations necessitates alternative control measures. Unlike Barbervax, no vaccines exist for sheep scour worms. Detailed knowledge of protective immunity is essential for vaccine development designed to mimic natural host responses. In this study, 17-week-old parasite-naïve Merino lambs were exposed to weekly trickle infections of 6,000 infective *Trichostrongylus colubriformis* larvae for 16 weeks; controls remained parasite-free. After 16 weeks, mean faecal egg counts were 1518 and 0. Histologically, there was a time-dependent increase in intestinal goblet cells and decrease in jejunal mast cells in infected lambs. Flow cytometry at week 16 identified $Ki67^+GATA3^+CD4^+$ T cells, indicating Th2 polarisation and expansion, with increased plasma and mucus anti-larval IgA and IgG. After drenching and challenge with 10,000 larvae, trickle-infected lambs rejected ~83% of larvae and had significantly lower worm burdens than controls. Anti-L3 *T. colubriformis* secretory IgA and IgG in bile may indicate portal immunoglobulin recycling from the gut. Additionally, in vitro antigen re-stimulation of cryopreserved intestinal cells showed increased $Ki67^+GATA3^+CD4^+$ proliferating leukocytes with soluble L3 antigens. These results indicate 16 weeks of exposure creates a hostile intestinal environment for L3 establishment. Ongoing work assesses responses to L3/L4 antigens and associated ovine intestinal gene expression profiles.

ID: 162 / **CP12: 6**
Contributed abstract
Conference Topics: Drugs, Livestock Parasites, Veterinary Parasitology
Keywords: Haemonchus contortus, Phenotypic screening, Structure–activity optimisation, Proteome integral solubility alteration assay, Nematocidal compounds

Chemical perturbation reveals a cytoskeletal–trafficking vulnerability in *Haemonchus contortus*

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Phenotypic screening readily identifies compounds that impair worm motility or development, the intrinsic biological processes underlying chemical sensitivity in parasitic nematodes remain poorly defined. Here, we identified a hit compound with a pyridyl scaffold from a phenotypic screen against the parasitic nematode, *Haemonchus contortus*, and using structure–activity optimisation we generated a potent chemical probe, WEHI-864. To uncover protein networks associated with the mechanism of action, thermal proteome profiling and time-resolved quantitative proteomics interrogated WEHI-864-induced perturbations in *H. contortus*. Across larval and adult stages of this major parasite of livestock, proteome integral solubility alteration (PISA) assay revealed reproducible alterations in proteins associated with cytoskeletal organisation and intracellular trafficking, including actin- and motor-related components. Complementary quantitative proteomics identified induction of an aspartyl protease and suppression of secretory CAP family proteins. Integrated analysis of these datasets supports a model in which chemical perturbation of cytoskeletal and trafficking proteins is associated with secondary modulation of proteolytic pathways, coinciding with rapid impairment of motility. These findings indicate that linked structural and proteolytic responses contribute to chemical sensitivity in *H. contortus* and demonstrate how integrative proteomics can resolve organism-level responses to chemical perturbation beyond single-target paradigms.

CP13: Ticks, Mites, kissing bugs 10 min talks

Time: Wednesday, 01/July/2026: 1:50pm - 2:30pm · *Location:* Lecture Theatre 3
Session Chair: Katja Fischer, QIMR Berghofer
Session Chair: Xavier Barton, Murdoch University

ID: 242 / **CP13: 1**
Contributed abstract
Conference Topics: Ectoparasites, Host-parasite interactions
Keywords: Scabies, NTD, scabicide, microbiome, sequencing

IMPACT OF STANDARD SCABICIDE TREATMENT ON SKIN MICROBIAL DYSBIOSIS IN SCABIES

Nuzhat Surve[1,2], Martha Zakrzewski[1], Pallavi Surase[2], Siddhi Chikhalkar[3], Sara Taylor[1], Deepani Fernando[1], Gita Nataraj[2], Chaya A Kumar[2], Katja Fischer[1]
[1]Infection & Inflammation Program, QIMR Berghofer, Brisbane, Australia; [2]Dept. of Microbiology, Seth Gordhandas Sunderdas Medical college and King Edward Memorial Hospital, Mumbai, India; [3]Dept. of Dermatology, Seth Gordhandas Sunderdas Medical college and King Edward Memorial Hospital, Mumbai, India

Background and Aims: Scabies is a neglected tropical skin disease affecting over 400 million people annually, causing a significant public health burden particularly among resource limited regions worldwide. Mite infection alters the skin microbiome and often leads to secondary bacterial skin infections and further serious sequelae. This study investigates whether scabicide therapy fully eliminates pathogens and restores the skin microbiome at the sites of scabies lesion.

Methods: Full-length 16S rRNA sequencing on PacBio platform was performed for 1224 skin scrapings samples collected from scabies lesions and their corresponding uninfected control sites. Participants received a prescribed scabies treatment, i.e. permethrin, ivermectin or a combination of permethrin and ivermectin. Samples were collected at three time points: before treatment, 5–7 days after the initial visit, and 4–6 weeks after the initial visit.

Results: Sequencing identified 114,403 Amplicon sequence variants across 1224 samples and 52 kit controls. Preliminary analysis indicates that the treatments restore bacterial diversity and reduce bacterial pathogen abundance in the healed sites, though the efficiency of pathogen reduction varied between young children and adults.

Conclusions: Early results show the complex interactions between scabies treatment, skin microbiome and host factors such as age with implications for managing secondary infections.

ID: 190 / **CP13: 2**
Contributed abstract
Conference Topics: Biochemistry, Bioinformatics, Ectoparasites, Host-parasite interactions, Proteomics
Keywords: Triatominae, venom, saliva, anticoagulant

Venom exaptation and adaptation during the trophic switch to blood-feeding by kissing bugs

Andrew Walker
The University of Queensland, Australia

Kissing bugs are known to produce anticoagulant venom that facilitates blood-feeding. However, it is unknown how this saliva evolved and if the venom produced by the entomophagous ancestors of kissing bugs would have helped or hindered the trophic shift. In this study, we show that venoms produced by extant predatory assassin bugs have strong anticoagulant properties mediated chiefly by proteolytic degradation of fibrinogen, and additionally contain anticoagulant disulfide-rich

peptides. However, venom produced by predatory species also has pain-inducing and membrane-permeabilizing activities that would be maladaptive for blood-feeding, and which venom of the blood-feeding species lack. This study demonstrates that venom produced by the predatory ancestors of kissing bugs was exapted for the trophic switch to blood-feeding by virtue of its anticoagulant properties. Further adaptation to blood-feeding occurred by downregulation of venom toxins with proteolytic, cytolytic, and pain-inducing activities, and upregulation and neofunctionalization of toxins with anticoagulant activityindependent of proteolysis.

ID: 107 / CP13: 3
Contributed abstract
Conference Topics: Ectoparasites
Keywords: scabies, microbiome, symbionts

Life-stage resolved microbiota of *Sarcoptes scabiei* reveals a stable core bacterial community comprised of opportunistic pathogens

Sara Taylor, Martha Zakrzewski, Deepani D Fernando, Kajta Fischer
Infection and Inflammation program, QIMR Berghofer, Brisbane, Australia

Scabies is a neglected tropical disease affecting approximately 400 million people globally. The obligate lifecycle of *Sarcoptes scabiei* has limited our understanding of its biology, largely due to difficulties in maintaining the parasite outside its host. The development of an *ex-vivo* culture system has facilitated improved investigation of the mite's complex biology. With the known prevalence of symbionts amongst haematophagous arthropods and the strong association with secondary bacterial infections, this study has attempted to provide the first life-stage specific microbiota and to identify core components of the *S. scabiei* microbiota. Over 48,000 individual parasites were collected across five key life-stages (eggs, larvae, nymphs, males and females). 16S full-length rRNA amplicon sequencing was performed on the PacBio platform. A total of 3,500,187 reads and 487 amplicon sequence variants (ASVs) were identified. The genera *Corynebacterium*, *Serratia* and *Acinetobacter* were present across all life-stages with the opportunistic pathogens *Acinetobacter baumannii* and *Serratia marcescens* identified as key constituents of the scabies microbiome. This study provides the first evidence of a stable core microbiota across all life stages of *S. scabiei*, offering new insights that could improve understanding of mite biology and management of scabies and associated secondary bacterial infections.

ID: 218 / CP13: 4
Contributed abstract
Conference Topics: Ectoparasites, Host-parasite interactions, Molecular Biology
Keywords: Sarcoptes scabiei, scabies, itch, non-histaminergic signalling, keratinocytes

Elucidating keratinocyte-mediated non-histaminergic signalling in scabies-associated itch

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Scabies, caused by *Sarcoptes scabiei*, is a highly prevalent skin disease characterised by severe and persistent pruritus manifesting in over 90% of patients. The limited effectiveness of antihistamines suggests a dominant role for non-histaminergic itch pathways, yet the underlying mechanisms remain poorly defined.Using a porcine scabies model, we localised itch mediators, such as PAR-2, MRGPRX2, tryptase, histamine, IL-31, periostin, NK-1R, β tubulin III and substance P, during infection using immune-histochemistry. Significant upregulation of PAR-2, MRGPRX2, tryptase, histamine, IL-31, periostin, NK-1R and substance P was observed following infection, while β tubulin III expression was reduced. Building on these findings, we aim to investigate keratinocyte-associated receptors (PAR-1, PAR-2, and MRGPRX2) in mediating itch responses to mite stimuli. HaCaT keratinocytes will be treated with whole mite extracts and recombinant proteins (SMIPP-Cc, Sar s 1c), followed by total RNA extraction, cDNA synthesis, and qPCR analysis of target gene expression, normalised to internal control GAPDH expression.These findings support the involvement of both histaminergic and non-histaminergic pathways in scabies itch. The second part of the study is expected to define keratinocyte-specific receptor responses, providing mechanistic insight into non-histaminergic itch signalling and identifying potential therapeutic targets for treatment-resistant and chronic pruritus.

CP11.1: Cells, Molecules & Genes 2 - 5 min talks

Time: Wednesday, 01/July/2026: 2:30pm - 2:45pm · *Location:* Lecture Theatre 1
Session Chair: Ellis Joch, Griffith University
Session Chair: Wisam Dawood, Griffith University

ID: 214 / **CP11.1: 1**
Contributed abstract
Conference Topics: Apicomplexa Biology, Drugs, Malaria, Molecular Biology, Protozoa
Keywords: Malaria, Resistance, Plasmodium, Antimalarial

Investigating phospholipid transport by the essential Plasmodium falciparum protein PfCSC1

Nathan Yau, Deyun Qiu, Adele M Lehane
Australian National University, Australia

PfCSC1 is a protein found in *Plasmodium falciparum* that belongs to a family of osmosensitive cation channels, some members of which have been found to double as scramblases (ATP-independent phospholipid transporters). Mutations in PfCSC1 or a putative rhomboid protease (PfROM8) are associated with resistance against specific compounds, termed PfROM8/PfCSC1-linked compounds (R/CLCs). To understand the mode of action of these compounds, the function of PfCSC1 must be understood. Previous research shows that PfCSC1 is an essential ion channel that can be activated by R/CLCs, with Na+ being one of its substrates.

Here, I present evidence that PfCSC1 doubles as a scramblase, capable of phospholipid transport in the parasite plasma membrane. The internalisation of a fluorescent phospholipid analogue (NBD-PS) was measured in ATP-depleted parasites under several conditions. Knockdown of PfCSC1 did not have a significant effect on NBD-PS internalisation. However, upon exposure to hypotonic conditions or R/CLCs – both predicted to activate PfCSC1 – parasites expressing a normal level of PfCSC1 displayed a significant increase in NBD-PS internalisation, whereas the response of parasites in which PfCSC1 was knocked down was less pronounced. The data suggest that while PfCSC1 is capable of phospholipid scrambling, it is likely not always active under physiological conditions.

ID: 256 / **CP11.1: 2**
Contributed abstract
Conference Topics: Apicomplexa Biology, Bioinformatics, Genomics
Keywords: Apicomplexa, mRNA Export

Do *Plasmodium* and Other Apicomplexan Parasites have Parasite Specific mRNA Export?

Lakvin Fernando[1,2], Stuart Ralph[1,2]
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The formation of the nucleus is one of the most significant paradigm shifts in the evolution of species. The nucleus allows eukaryotes to transcribe with higher fidelity and better regulate the expression of their genes, resulting in highly specialised cells; the caveat being that eukaryotes must transport their RNA cargo into the cytoplasm to re-couple the partitioned transcription and translation. Most eukaryotes use an RanGTP-dependent system for nucleocytoplasmic transport including for the export of non-coding RNA. In addition, fungi and metazoans have evolved specialised RanGTP-independent pathways to export most Poly-A+ mRNA.

The mechanisms of mRNA export in protist parasites are relatively understudied. Proteins that have conserved sequences to proteins involved in RanGTP-independent mRNA export in humans and yeasts have been identified in *Plasmodium* spp. *Toxoplasma gondii*, and *Cryptosporidium* spp based on conserved sequences. However it is unclear if these parasites have mechanisms of mRNA export that are analogous to the RanGTP-independent metazoan mechanisms or if they have innovated parasite specific mechanisms of mRNA export.

I aim to determine if apicomplexan parasites have evolved parasite specific processes of mRNA export by identifying key molecules involved nucleocytoplasmic transport.

ID: 184 / **CP11.1: 3**
Contributed abstract
Conference Topics: Malaria, Molecular Biology
Keywords: New permeability pathways, CLAG3, nutrient uptake, sorbitol resistance

Characterising the impact of sorbitol resistance on nutrient uptake in *Plasmodium falciparum*

Nadine Djunaedi, Christopher MacRaild, Mitchell Trickey, Darren Creek
Monash Institute of Pharmaceutical Sciences, Australia

Malaria remains a global health burden and with ongoing resistance across all classes of antimalarials, advancing our understanding of *Plasmodium*'s biology could help identify novel therapeutic targets. During continuous *in vitro* culture, mutations commonly arise within the parasite genome, although these are often phenotypically silent. However, we have identified a sorbitol-resistant *Pf*3D7 strain that no longer undergoes haemolysis when exposed to isotonic concentrations of sorbitol, indicating a disruption in nutrient acquisition via new permeability pathways (NPPs) formed by parasites to facilitate enhanced nutrient uptake. In contrast to other sorbitol-resistant lines, this occurs without growth defects under standard culture conditions. Whole genome sequencing of clonal parasites identified a recombinant CLAG3 gene (CLAG3n) with a missense mutation at the recombination region. CLAG3 is a component of the RhopH complex, a trimeric protein complex essential for NPP formation. To characterise the phenotype of our CLAG3n line, we assessed parasite growth under normal and nutrient-deprived conditions and performed osmotic lysis assays using a range of solutes to evaluate changes in substrate selectivity. Additionally, we conducted proteome-wide analyses to investigate alterations in protein expression and aim to conduct localisation studies to determine the impact of the recombination and mutation on protein export and NPP function.

CP12.1: Sheep & Goats - 5 min talks

Time: Wednesday, 01/July/2026: 2:30pm - 2:45pm · *Location:* Lecture Theatre 2
Session Chair: Vern Bowles, The University of Melbourne
Session Chair: Nichola Calvani, The University of Sydney

ID: 180 / **CP12.1: 1**
Contributed abstract
Conference Topics: Drugs, Livestock Parasites, Veterinary Parasitology
Keywords: Haemonchus contortus, Caenorhabditis elegans, high-throughput screening, drug discovery, early anthelmintic discovery

High-throughput phenotypic screening of Medicines for Malaria Venture's Hit Generation Library 1 identifies new nematocidal chemotypes

Joseph Byrne[1], Aya Taki[1], Bill Chang[1], Benoit Laleu[2], Timothy Wells[2], Abdul Jabbar[1], Robin Gasser[1]
[1]Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Parkville, Victoria 3010, Australia; [2]Medicines for Malaria Venture (MMV), 1215 Geneva, Switzerland

Parasitic worms continue to exert major health and economic burdens on humans and livestock, while escalating resistance to existing anthelmintics highlights the urgent need for novel chemotypes with distinct modes of action to support integrated control strategies. We screened 139,916 compounds from the Medicines for Malaria Venture Hit Generation Library 1 against exsheathed third-stage larvae of *Haemonchus contortus*, with cross-species assessment in *Caenorhabditis elegans*. High-throughput infrared-based motility and developmental assays in 384-well format showed robust performance (mean Z′ = 0.799 ± 0.012; signal-to-background = 65.6 ± 9.8). From this screen, 272 primary hits (0.194%) were identified, of which 110 reproducibly inhibited larval motility and development. Among these, 39 compounds exhibited IC_{50} values <10 µM, and 33 induced complete developmental arrest at ≤12.5 µM, often with distinct morphological phenotypes. Four of the 39 compounds showed no detectable toxicity in HepG2 cells (CC_{50} and MC_{50} ≥20 µM), with ADME profiling available for a prioritsed subset. Integrating potency, selectivity and ADME properties enabled prioritisation of 16 compounds for advancement through medicinal chemistry. Collectively, these findings demonstrate that antimalarial-focused libraries can yield potent and selective nematocidal scaffolds, and highlight a scalable strategy for repurposing discovery libraries across various parasitic systems.

ID: 217 / **CP12.1: 2**
Contributed abstract
Conference Topics: Epidemiology, Livestock Parasites, Veterinary Parasitology
Keywords: Gastrointestinal nematodes, deep amplicon sequencing, Haemonchus contortus, dairy goats, Australia

A national survey of gastrointestinal nematodes in Australian dairy goats using faecal egg counts and deep amplicon sequencing

Endris Ali[1], Abdul Ghafar[1], Ghazanfar Abbas[1], Bahar E Mustafa[1], Sandra Baxendell[2], Elysia Ling[1], Charles Guaci[1], Ian Beveridge[1], Mark Stevenson[1], Abdul Jabbar[1]
[1]The University of Melbourne, Victoria, Australia; [2]Goat Veterinary Consultancies—goatvetoz, Keperra, Queensland, Australia

Gastrointestinal nematodes (GINs) are a major constraint to goat health, welfare and productivity worldwide and represent the highest-priority pathogens affecting goats in Australia. However, contemporary national-scale epidemiological data for Australian dairy goats are limited. This study quantified the prevalence, infection intensity, species diversity and determinants for GIN infections in Australian dairy goats. Faecal samples (n = 1,028) collected from 68 herds across Australia were analysed using a modified McMaster technique. Strongylid-positive samples (n = 500) underwent deep amplicon sequencing of the ITS-2 rDNA region for species identification. Prevalence estimates were adjusted for herd-level clustering, and predictors for strongylid faecal egg counts (FECs) were evaluated using linear mixed-effects models. Strongylids were detected in 91% of goats and in all herds, substantially exceeding the prevalence of *Trichuris* spp. (13%) and *Nematodirus* spp. (5%). Strongylid FECs were markedly overdispersed, with 30% of animals contributing 80% of total egg output. Age, climatic zone, anthelmintic treatment and coinfection with *Eimeria* were statistically significant (P < 0.05) determinants of strongylid FECs. Metabarcoding identified 11 species, dominated by *Haemonchus contortus*, *Teladorsagia circumcincta* and *Trichostrongylus colubriformis,* with distinct regional patterns. These findings demonstrate substantial infection pressure and species diversity, informing evidence-based parasite control for the Australian goat industry.

ID: 113 / **CP12.1: 3**
Contributed abstract
Conference Topics: Fasciolosis/Liver fluke
Keywords: Lymnaeid Snail, Invasive Species, Integrated Parasite Management, Liver fluke, Epidemiology

"First frost, last frost" Updating current knowledge on the seasonality of *Fasciola hepatica* in the Southern Tablelands of NSW

Priscilla Huynh[1], Roger A. Willoughby[2], Neil Young[3], Tanapan Sukee[3], Emily K. Francis[1], Nichola E. D. Calvani[1]
[1]University of Sydney, Australia; [2]Gunning Ag & Water Solutions, Gunning, NSW, Australia; [3]Melbourne Veterinary School, Faculty of Veterinary and Agricultural Sciences, The University of Melbourne, Vic, Australia

Fasciola hepatica larval development, infection, and presence of intermediate lymnaeid snail hosts require average daily temperatures >10°C. Livestock producers leverage the seasonal pause between the first and last frosts to guide treatment schedules. Whilst conducting a drug resistance trial in July 2024, numerous lymnaeid snails were discovered when average daily temperatures were <7°C, challenging the foundations underpinning these schedules. We set out to update knowledge on the seasonality of lymnaeid snail populations in the region to refine current *F. hepatica* risk periods and inform new integrated parasite management strategies.

Twelve sites across six farms endemic for *F. hepatica* were sampled bi-monthly during 2025. Collected snails were counted and morphologically speciated to determine the average abundance and diversity of susceptible hosts. Snails were visually inspected for *F. hepatica* larval infection, then confirmed with qPCR. One site significantly deviated from the historical seasonal

pattern, recording the study's highest abundance of lymnaeid snails (N = 240) during winter. The invasive *Pseudosuccinea columella* was most abundant in July at three sites. This study provides the first seasonal monitoring of the invasive *P. columella* and their increased abundance in winter suggests that the infective risk period for *F. hepatica* extends beyond the historical dormancy window.

CP13.1: Ticks, Mites, kissing bugs 5 min talks

Time: Wednesday, 01/July/2026: 2:30pm - 2:45pm · *Location:* Lecture Theatre 3
Session Chair: Katja Fischer, QIMR Berghofer
Session Chair: Xavier Barton, Murdoch University

ID: 270 / **CP13.1: 1**
Contributed abstract
Conference Topics: Ectoparasites, Invasive Species, Veterinary Parasitology
Keywords: invasive tick, Haemaphysalis longicornis, biosecurity

Phylogeography and genetic structure of HaemaphysalisIongicornis (Asian longhorned tick)

Zainab Umar Abdullahi[1], Dieter Bulach[2], Joanne M. Devlin[1], Glenn F. Browning[1]
[1]Asia-Pacific Centre for Animal Health, Melbourne Veterinary School, The University of Melbourne, Parkville, Australia; [2]National Centre for Antimicrobial Stewardship, The University of Melbourne, Parkville, Australia

The invasive tick *Haemaphysalis longicornis* is an important vector of veterinary and public health significance and has expanded beyond its native Asian range into Australia, Western Pacific Islands, and the USA. Despite its growing biosecurity importance, the global phylogeographic structure of this species remains unknown. This study investigated the evolutionary relationships and population structure of *H. longicornis* using 1,396 cytochrome c oxidase subunit I (COI). Sequences represented native populations from China, Japan, South Korea, and Russia, and invaded populations from Australia, New Zealand, Tonga, Pakistan, and the USA. A total of 166 haplotypes were identified, with high haplotype diversity (Hd = 0.88). Haplotype network and phylogenetic analyses identified two major lineages: a cosmopolitan lineage shared between native and invaded regions, and a second lineage restricted to Asia. Population genetic analyses demonstrated contrasting levels of genetic differentiation within and among countries, including high differentiation between Korea and the USA (FST = 0.73), while the lowest differentiation occurred between China and Korea (FST = 0.06). The cosmopolitan lineage displayed a star-like topology, suggesting demographic expansion and dispersal into invaded regions. These findings provide new insights into the invasion events, dispersal pathways, and evolutionary structure of *H. longicornis.*

ID: 161 / **CP13.1: 2**
Contributed abstract
Conference Topics: Ecology, Ectoparasites
Keywords: phenolgy, citizen science, ecology, Ixodidae

The Ticks You're Not Seeing: How Seasonality Shapes Tick Detection in Western Australia

Xavier Barton[1], Abdul Ghaffar Qamar[1], Jani Sormunen[2], Joseph Fontaine[3], Charlotte Oskam[1]
[1]School of Medical, Molecular and Forensic Sciences, College of Environmental and Life Sciences, Murdoch University; [2]Department of Biology, University of Turku; [3]School of Environmental and Conservation Sciences, College of Environmental and Life Sciences, Murdoch University

Ticks pose health risks to humans, companion animals and livestock through pathogen transmission, making knowledge of their seasonal activity vital for monitoring tick-borne disease. No studies have investigated the seasonal presence of questing tick species and life stages in Western Australia. This study presents preliminary findings from an ongoing phenological survey (October 2025 to October 2026) across four Swan Coastal Plain sites encompassing banksia woodland and pastoral land. Thirty-two fortnightly flagging sessions were conducted; flags were dragged and inspected at 10 m intervals with specimens morphologically identified to species and instar. All 1,859 ticks collected were *Amblyomma triguttatum*: larvae 88.2% (n = 1,639), nymphs 11.4% (n = 211) and adults 0.5% (n = 9). A seasonal shift was observed, with nymphs most prevalent in October to November 2025 and larval activity emerging in January 2026, peaking in February (n = 822). Site-level abundance ranged from 3.04 to 9.52 ticks per 100 m. Comparison with iNaturalist data (n = 227 observations, 2023 to 2025) revealed a spring-biased, adult-dominated reporting pattern, contrasting with flagging results where adults were largely absent and late-summer larval emergence was substantial. These findings highlight the value of combining standardised surveys and citizen science in tick phenology studies.

ID: 209 / **CP13.1: 3**
Contributed abstract
Conference Topics: Biochemistry, Ectoparasites, Host-parasite interactions, Immunology, Molecular Biology
Keywords: ticks, alpha-gal

Quantitative Assessment of α-Gal Production in Ixodes holocyclus Salivary Glands Using Indirect ELISA Methods

Emily Smith[1,2], Alexander Gofton[2], Raine Mercedes[2], Stephen Barker[3], Andrew Walker[1]
[1]Institute for Molecular Bioscience, UQ, Australia; [2]CSIRO; [3]School of Chemistry and Molecular Biology, UQ

Alpha-gal syndrome (AGS) is a tick bite-induced, IgE-mediated allergy to the carbohydrate galactose-α-1,3-galactose (α-Gal), characterised by delayed-onset hypersensitivity to mammalian meat and other mammal-derived products. AGS occurs globally

where ticks bite humans, with Australia experiencing some of the highest rates of AGS prevalence worldwide. Existing studies from North American and European tick species suggest that some ticks can produce α-Gal in their salivary glands, and that expression varies with feeding stage. However, comparable quantitative data are absent for the causative agent of AGS in Australia, the eastern paralysis tick, *Ixodes holocyclus*. Little is known about how much α-Gal *I. holocyclus* produces in its salivary glands, dynamics of α-Gal levels during blood feeding, or how host species influence α-Gal production.

To address this, indirect enzyme-linked immunosorbent assays (ELISAs) will be used to quantify α-Gal levels in *I. holocyclus* salivary glands across the feeding cycle and between different host species. By generating quantitative profiles of α-Gal abundance and integrating these with complementary localisation studies, this work aims to establish the first systematic assessment of α-Gal production dynamics in *I. holocyclus*. These data will provide critical insight into how tick biology and feeding behaviour shape α-Gal exposure and AGS risk in Australia.

The John Frederick Adrian Sprent Prize and Oration

3:30pm - 4:00pm
Location: Plenary Lecture Theatre
Session Chair: Aaron Jex, WEHI

P3: Elsevier Plenary Lecture Series International Journal for Parasitology (IJP) Invited Lecturer

Time: Thursday, 02/July/2026: 9:00am - 9:45am · *Location:* Plenary Lecture Theatre
Session Chair: Brian Cooke, James Cook University

ID: 277 / **P3: 1**
Invited speaker abstract

Don’t put that in your talk! The importance of life cycles in parasitology research.

Christopher Goodman
The University of Melbourne, Australia

Many parasites have elaborate life cycles with multiple stages and strict host requirements. The biological questions posed by these life cycles are fascinating, but their complexity can be overwhelming and showing every detail sends audiences reaching for their phones. As a result, life cycles are frequently overlooked, even though they are fundamental to questions of why parasites live the way they do, how they adapt to changing environments, and whether we can exploit life cycle traits to eradicate disease.

We aim to answer some of these questions by studying the mosquito stages of the malaria life cycle. To survive in the insect host, malaria parasites must adapt to a radically different physical and immunological environment. They do this by rapidly and irreversibly progressing through distinct morphological forms and significantly altering their metabolic processes. Faced with these extreme changes, the parasite population shrinks dramatically, and the surviving parasites grow very slowly. Somewhat surprisingly, parasites complete their obligate sexual reproduction during this period of stress and parasite death. Recent advancers in our understanding of the biological processes and genetic impacts of mosquito-stage development highlight significant vulnerabilities that can be exploited to disrupt disease transmission. We’ve identified anti-malarial compounds that can be delivered across the mosquito life-stages to directly kill parasites with minimal selection for resistance, and drug-resistant parasites with little or no ability to survive under the metabolic demands of the mosquito stages. We’re also exploring new genetic tools than can be exploit the obligate sexual stages to spread through and modify entire parasite populations.

Surprisingly, we discovered significant gaps in our knowledge of the well-studied malaria parasite life cycle. Being able to fill these gaps is an important benefit of developing new anti-malarial strategies and highlights the practical importance of thoroughly understanding parasite life cycles. While the details of these new anti-parasite strategies are malaria specific, we believe that many other parasite life cycles share these vulnerabilities and can be targeted using similar approaches tailored to specific parasite biology.

CP14: Top Rated Contributed Abstracts 15 min talks

Time: Thursday, 02/July/2026: 9:45am - 10:30am · *Location:* Plenary Lecture Theatre
Session Chair: Danielle Stanisic, Institute for Biomedicine and Glycomics, Griffith University

ID: 120 / CP14: 1
Contributed abstract
Conference Topics: Ectoparasites, Epidemiology, Immunology, One Health
Keywords: Mammalian meat allergy, epidemiology, tick bite, alpha-gal syndrome

Tick-Induced Mammalian Meat Allergy in Australia: National Prevalence and Geographic Distribution from Laboratory Surveillance, 2014-2024

Alexander W. Gofton[1,2], Emily Smith[1,3,4], Paul Campbell[5], Carl Kennedy[6], Karl Baumgart[7], Lucinda Wallman[8], Stephen C. Barker[9], Sheryl van Nunen[2,10,11], Andrew A. Walker[3,4]
[1]CSIRO Health and Biosecurity, Brisbane, Australia; [2]TiARA (Tick-induced Allergies Research and Awareness), Australia; [3]Institute for Molecular Bioscience, The University of Queensland, Brisbane, Australia; [4]Australian Research Council Centre of Excellence for Innovations in Peptide and Protein Science, The University of Queensland, Brisbane, Australia; [5]QML Pathology, Brisbane, Australia; [6]Sullivan & Nicolaides Pathology, Brisbane, Australia; [7]Douglass Hanly Moir Pathology, Sydney, Australia; [8]Laverty Pathology, Sydney, Australia; [9]School of Chemistry and Molecular Biology, The University of Queensland, Brisbane, Australia; [10]National Allergy Centre of Excellence, Australia; [11]Faculty of Medicine and Health, The University of Sydney, Sydney, Australia

Mammalian meat allergy (MMA) is an IgE-mediated allergic condition triggered by sensitisation to galactose-α-1,3-galactose (α-Gal) following tick bite. First described in Australia in 2007, MMA is now recognised globally, yet its national burden in Australia has never been systematically characterised. We addressed this gap by analysing 11 years of national α-Gal specific IgE testing data (2014–2024), encompassing over 16,000 tests from 14,000 individuals. Our findings reveal MMA as a substantial and rapidly growing health concern, with case detection increasing 22% annually since 2020. The geographic distribution of cases closely mirrors the range of *Ixodes holocyclus*, with extreme spatial clustering within this endemic zone: just nine discrete regions account for over half od the national MMA disease burden. This tight coupling between vector ecology and disease distribution underscores the central role of *I. holocyclus* in driving MMA in Australia, with only minor contributions from other tick species. Among serially tested patients, α-Gal specific IgE levels declined over time in the vast majority of individuals, supporting the utility of repeat testing for clinical monitoring. This study provides the first comprehensive national epidemiological assessment of MMA in Australia and establishes baseline metrics for ongoing surveillance of this emerging tick-borne allergic condition.

ID: 198 / CP14: 2
Contributed abstract
Conference Topics: Apicomplexa Biology, Cell Biology, Drugs, Immunology, Malaria, Vaccines
Keywords: falciparum, antimalarial, vaccine, plasmepsin, prevention

A late liver-stage chemical vaccine for malaria

Ryan Steel[1,2], Yu Cheng Chua[1,2,3], Waail Abdalla[1], Robyn McConville[1,2], Amelia Ford[1,2], Sabrina Caiazzo[1,2], Eva Hesping[1,2], Daniel Fernandez-Ruiz[3,4], Lauren Holz[3], William Heath[3], John McCauley[5], David Olsen[5], Justin Boddey[1,2]
[1]Walter and Eliza Hall Institute, Australia; [2]Department of Medical Biology, University of Melbourne; [3]The Peter Doherty Institute, University of Melbourne; [4]University of New South Wales; [5]Merck & Co., Inc., USA

Late-arresting sporozoite vaccines against *Plasmodium falciparum* achieve high efficacy but pose manufacturing and intravenous delivery challenges. We describe an alternative chemo-attenuation strategy that exploits first-in-class antimalarials targeting parasite aspartyl proteases plasmepsin IX and X. A single low-dose infection with virulent *Plasmodium berghei* sporozoites, delivered intravenously or by mosquito bite, followed by cure with a dual plasmepsin IX/X inhibitor prevented blood-stage infection by generating chemo-attenuated liver merozoites (CALM) that are incapable of erythrocyte invasion. CALM vaccination conferred sterile protection in mice for up to 2 years. Protective immunity involved both antibodies and CD8+ T cells recognising a spectrum of *Plasmodium* antigens including CSP, SERA1, RPL6, GAP50, RNT, PHIST, S20 and RBP. Studies in humanised mice confirmed plasmepsin IX/X inhibition similarly prevents *Plasmodium falciparum* liver-to-blood transition, demonstrating conservation of the drug's late liver-stage mechanism across species. By enabling controlled liver-stage arrest without complex genetic attenuation or irradiation, plasmepsin IX/X-targeting drugs provide a practical, broadly applicable pathway to whole-parasite chemovaccination. This approach may convert seasonal mosquito exposure into progressive immune education if long-acting injectable (LAI) formulations prove feasible. These findings lay the foundation for clinical evaluation of CALM vaccination including via mosquito-bite immunisation. Aspirationally, LAI CALM could transform malaria prevention programs at scale.

ID: 172 / CP14: 3
Contributed abstract
Conference Topics: Bioinformatics, Ectoparasites, Genomics, Livestock Parasites, Molecular Biology, Veterinary Parasitology
Keywords: flystrike, Australian sheep blowfly, Genome sequencing

Towards Sustainable Flystrike Control: A Chromosomal-Level Genome and Population Genomics of *Lucilia cuprina dorsalis*

Shilpa Kapoor[1,2], Amrita Vijay[2], Balu Balan[2], Louise Baker[2], Laura Wines[3], Vernon M. Bowles[1], Aaron R. Jex[1,2], Clare A. Anstead[1]
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The Australian sheep blowfly, *Lucilia cuprina dorsalis*, is a significant ectoparasite of sheep responsible for flystrike, leading to substantial production losses, animal injury, and mortality. Current flystrike control strategies rely heavily on surgical mulesing and insecticides; however, these approaches present ongoing welfare, sustainability, and efficacy challenges. Despite the

economic and biological importance of this species, genomic resources for *L. c. dorsalis* remain limited, constraining our understanding of its biology and population dynamics. To address this gap, we employed an integrated genomics approach combining Oxford Nanopore Technologies (ONT) long-read sequencing, Illumina short-read sequencing, and Omni-C proximity ligation to generate a chromosomal-level genome assembly. Additionally, comprehensive long- and short-read RNA sequencing was used to construct a high-resolution de novo transcriptome. Together, these datasets provide a robust foundation for genome annotation and functional characterization. This multi-omics framework provides new insights into the genetic architecture, evolutionary history, and key biological processes of *L. c. dorsalis*. Importantly, the chromosomal-level assembly enables population genomics analyses across Australian blowfly populations, facilitating investigation of genetic diversity, structure, and dispersal. These resources establish a critical platform for advancing genomic research and supporting the development of improved and sustainable flystrike management strategies, bridging fundamental genomics with applied sheep health outcomes.

CP17: Epidemiology & Diagnostics 15 min talk

Time: Thursday, 02/July/2026: 11:00am - 11:15am · *Location:* Lecture Theatre 3
Session Chair: Deepani Fernando, QIMR Berghofer
Session Chair: Luke Hall, St Vincent's Hospital Sydney

ID: 143 / **CP17: 1**
Contributed abstract
Conference Topics: Diagnostics, Helminthology, Molecular Biology, Strongyloides
Keywords: Strongyloidiasis, Diagnostics, CRISPR, SHERLOCK

Development of CRISPR-based diagnostic tools for the detection of Strongyloidiasis

Skye MacGregor[1], Matthew Watts[2], Malcolm Jones[1,3], Rogan Lee[2], Vito Colella[4], Richard Bradbury[5], Thomas Egwang[6], Darren Gray[7], Hong You[1,3]

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Strongyloidiasis is a much-neglected disease caused by the soil-transmitted helminth *Strongyloides stercoralis*, afflicting millions globally- predominantly low-income tropical regions as well as the remote Aboriginal communities of northern Australia. Current diagnostic tests for Strongyloidiasis are neither sufficiently sensitive nor field-friendly for use in low-endemic and resource-poor settings, and reliable point-of-care (POC) diagnostic tools are urgently needed for disease mapping and monitoring of control efforts.

CRISPR technologies have enabled the development of a powerful new class of rapid, ultra-sensitive, cost-effective POC diagnostics for viruses/cancers. For the first time, we developed CRISPR-based assays for the detection of Strongyloidiasis, validated using human and animal stool samples collected from *S. stercoralis* endemic regions. The assays were demonstrated to be highly specific, with no cross-reactivity observed with an array of bacteria/fungi/parasite species. They also showed a sensitivity comparable to qPCR (the emerging gold standard), but are more field-friendly, with significantly reduced need for specialised equipment and expertise, requiring only a simple portable heat-block and UV detection. We also developed duplex CRISPR-based assays for simultaneous detection of other major helminth infections including *Schistosoma mansoni* and hookworm (*Necator americanus*), to better investigate helminth co-infections. This CRISPR-based platform offers a promising, field-ready next-generation approach to Strongyloidiasis diagnostics.

S6: Drugs & Drug Resistance Symposium sponsored by Institute for Biomedicine and Glycomics, Griffith University

Time: Thursday, 02/July/2026: 11:00am - 11:20am · *Location:* Lecture Theatre 1
Session Chair: Jacinta Macdonald, Griffith University
Session Chair: Rohith Kutty, Griffith University

ID: 101 / **S6: 1**
Invited speaker abstract

Malaria drug discovery and target identification – from gene editing to AI tools

Jacinta Macdonald[1], Wisam Dawood[1], Gillian Fisher[1], Tina Skinner-Adams[1,2], Katherine Andrews[1,2]
[1]Institute for Biomedicine and Glycomics, Griffith University, Queensland, Australia.; [2]School of Environment and Science, Griffith University, Nathan, Queensland, Australia.

Malaria causes significant morbidity and mortality, with 282 million cases and 610,000 deaths in 2024. The past decade has seen progress towards malaria eradication, however recent trends indicate that improvements have plateaued, partly due to parasite drug-resistance and treatment failure. To combat parasite drug resistance, new drugs with different modes of action to current antimalarials are needed. We have identified novel antiplasmodial compounds from synthetic and natural product libraries and using machine learning/AI tools. This includes the indoloquinolizidine alkaloid natural product alstonine (*Pf*IC_{50} of 0.18 µM, Selectivity Index (SI) >1,000), novel 1,3,4-oxadiazoles (e.g., **3** with *Pf*IC_{50} 0.16 µM, SI 162) and primary hydroxamates (e.g., ACY-738 with *Pf*IC_{50} 0.08 µM, SI 314). In this presentation, an overview will be given on strategies employed to identify these compounds and to understand their antiplasmodial action, including phenotypic analyses and using CRISPR/Cas9 mediated approaches to investigate putative targets. Elucidating the mechanism of action of antiplasmodial compounds can help identify novel druggable targets, inform downstream drug development and aid in improving our understanding *Plasmodium* biology.

S7: Horses & Cows 1 Symposium

Time: Thursday, 02/July/2026: 11:00am - 11:20am · *Location:* Lecture Theatre 2
Session Chair: Abdul Jabbar, The University of Melbourne
Session Chair: Narelle Dybing, Murdoch University

ID: 278 / **S7: 1**
Invited speaker abstract

Rethinking deworming: resistance, risk, and responsible control of canine hookworms

Swaid Abdullah
The University of Queensland, Australia

Canine hookworms, predominantly *Ancylostoma caninum*, remain the most prevalent intestinal nematode of dogs in Australia, with infection dynamics strongly influenced by host age, management practices, and geographic location. Current anthelmintic control relies on three major drug classes - benzimidazoles (febantel, fenbendazole), macrocyclic lactones (moxidectin, milbemycin oxime), and tetrahydropyrimidines (pyrantel). However, an increasing number of clinical cases are characterised by persistent infections refractory to standard treatment regimens.

Recent investigations from our group provide compelling evidence that many of these cases are associated with reduced efficacy, and in some instances resistance, to fenbendazole and pyrantel. These findings raise concerns regarding the emergence of multiple anthelmintic drug resistance (MADR) in Australian *A. caninum* populations. Epidemiological trends indicate a disproportionate representation of affected dogs originating from breeding and training facilities, where intensive deworming and high environmental contamination likely create strong selection pressure for resistant parasite populations.

The growing movement of dogs from such facilities into the general community presents a significant risk for the dissemination of resistant hookworm strains. This concern is further amplified by recent reports of MADR *A. caninum* in the United States, underscoring the global relevance of this emerging threat. Adoption of evidence-based, risk-driven parasite control strategies is therefore critical to minimise unnecessary anthelmintic use and slow resistance selection.

This presentation will discuss current advances in the diagnosis of anthelmintic resistance in canine hookworms, encompassing in vitro assays, in vivo efficacy studies, and molecular approaches targeting resistance-associated markers. In addition, I will highlight the urgent need for coordinated national surveillance and reporting frameworks to detect and respond to treatment failure. Finally, the development of integrated, multidisciplinary guidelines engaging veterinarians, parasitologists, epidemiologists, and animal welfare organisations will be proposed as a strategic priority to safeguard the long-term efficacy of anthelmintics and ensure sustainable parasite control in dogs.

CP17.1: Epidemiology & Diagnostics 10 min talks

Time: Thursday, 02/July/2026: 11:15am - 12:05pm · *Location:* Lecture Theatre 3
Session Chair: Deepani Fernando, QIMR Berghofer
Session Chair: Luke Hall, St Vincent's Hospital Sydney

ID: 147 / CP17.1: 1
Contributed abstract
Conference Topics: Diagnostics, Helminthology, Molecular Biology
Keywords: CRISPR, helminth infections, nucleic acid detection, simplified DNA extraction, point-of-care diagnostics

A Field-Deployable CRISPR-Cas12/13 Diagnostic Platform Integrated with Rapid DNA Extraction for Helminth Detection

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[1]Infection and Inflammation Program, QIMR Berghofer Medical Research Institute, Brisbane, Queensland, Australia; [2]UQ Centre Clinical Research, The University of Queensland, Brisbane, Queensland, Australia; [3]Cancer Research Program, QIMR Berghofer Medical Research Institute, Brisbane, Queensland, Australia; [4]School of Veterinary Science, The University of Queensland, Gatton, Queensland, Australia

Helminth infections, particularly schistosomiasis and soil-transmitted helminthiases, impose a severe global health burden, infecting more than a quarter of the world population, with a disproportionate effect on those in extreme poverty. Current diagnostic tests for worm infections are neither sufficiently sensitive nor field-friendly for use in resource-limited or low-endemic settings, leading to underestimation of true infection rates. Ultrasensitive, field-friendly, low-cost point-of-care diagnostics are urgently needed to better control these diseases.
Our group has established a highly sensitive CRISPR-Cas12/13-based platform for femtogram-level nucleic acid detection of multiple helminth infections. We also developed portable, lyophilized one-pot CRISPR detection to enable field testing, reducing reliance on cold-chain transportation and making it easier to perform. To further address the DNA sample preparation bottleneck in field applications, we developed a rapid extraction method that yields high-quality DNA for use with our CRISPR-based schistosomiasis diagnostic tool. Our method is faster and supports scalable, cost-effective field screening, compared with conventional DNA extraction.
To demonstrate the clinical utility of this integrated system, we are currently validating the assay for helminth detection using a large cohort of human samples from Uganda. This scalable, field-deployable approach holds significant potential to advance global NTD control and surveillance efforts.

ID: 224 / CP17.1: 2
Contributed abstract
Conference Topics: Malaria, Microscopy
Keywords: Artificial intelligence; Giemsa stain; light microscopy

Rapid Multi-Species Malaria Parasite Detection Using Deep Learning

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[1]UNSW, Australia; [2]Imperial College London

Giemsa-stained blood smear microscopy is the gold standard for detecting malaria parasites, but it is time-consuming and limited for storage and reference. To address this, we developed PlasmoCount, a deep learning tool for accurate, automated counting of intracellular parasites and digital archiving support. Principally, we have achieved a substantial reduction in PlasmoCount's processing time allowing for evaluation of a single image in under 3 seconds (reduced from 40). In addition, we have updated the tool so that it can now detect blood-stage infections from multiple species of human-infective and experimental rodent-infective *Plasmodium* parasites. Combined with a suite of other updates, including advanced cell differentiation and use at different magnifications, these augmentations broaden the distribution of input data our model can accommodate and radically advance its speed whilst maintaining its high classification accuracy (99.8%). Finally, we provide an offline, on-device version of the standardised framework designed for smartphones, including iOS and Android operating systems. By making use of imported images or image capture via a smartphone camera, PlasmoCount 2.0 markedly improves malaria parasite smear-based detection and provides a reproducible means to assess parasite infections either in routine laboratory work or as a future aid in clinical or field diagnosis.

ID: 235 / CP17.1: 3
Contributed abstract
Conference Topics: Epidemiology, Genomics, Host-parasite interactions, Malaria
Keywords: Asymptomatic, Clinical malaria, Population genetics, Surveillance

Population genetics of *P. falciparum* clinical and asymptomatic infections at low transmission

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Countries close to malaria elimination are reporting significant resurgence following periods of declining prevalence. Reduced transmission decreases opportunities for genetic recombination and generates geographically isolated hotspots of infection, resulting in lower diversity and increased population structure. With reduced exposure, naturally acquired immunity wanes, potentially rendering human populations more susceptible to outbreaks. But paradoxically, many low-transmission countries also record high prevalence of asymptomatic infections. We hypothesised that these asymptomatic cases are associated with immunologically familiar, locally circulating strains whereas clinically infectious parasites are potentially imported. We analysed *Plasmodium falciparum* samples from a period of low transmission (2012) prior to resurgence (2016) in East Sepik, Papua New Guinea (PNG), and from a low-transmission setting with ongoing occupational exposure in Mondulkiri, Cambodia, by sequencing a validated genome-wide single nucleotide polymorphism (SNP) barcode and immune evasion antigen marker (varcode). Parasite lineages underlying clinical infections in PNG were clonal and distinct from circulating asymptomatic isolates, suggesting potential importation and outbreak caused by immunologically unfamiliar parasites. Infections in Cambodia however indicate a more complex dynamic between parasite strain and host factors. These results emphasise how surveillance reliant on just clinical infections inadequately reflects control success and must account for asymptomatic malaria for sustainable reduction and elimination.

ID: 134 / **CP17.1: 4**
Contributed abstract
Conference Topics: Epidemiology, Microscopy, Protozoa
Keywords: Dientamoeba fragilis, Cyst stage, Historical slides

Dientamoeba fragilis cysts and precysts in historic slide collections and a review of cyst formation among the Parabasalia

Luke Hall[1,2], Joel Barratt[3], John Ellis[2], Damien Stark[1]
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Transmission is a basic aspect of intestinal parasite's biology that is poorly understood for *Dientamoeba fragilis*. Early historical reports reflecting the absence of a cyst are often cited as a central argument in debates supporting the lack of a *D. fragilis* cyst. Despite *D. fragilis* cysts being described since Dobell's original description, their existence is not universally accepted. Here, Dobell's, Wenyon's, and Hoare's collection of historical faecal smears stored at the Natural History Museum (London), dating back to the 1890s and the early 1900s, were examined for forms consistent with modern descriptions of *D. fragilis* cysts, and an example was found in one slide. Such rare forms were also detected during examination of stained faecal smears archived in the parasite reference laboratory collection at the United States Centers for Disease Control and Prevention (CDC). Considering published literature on the subject of *D. fragilis* cysts and the broader picture of cyst formation across diverse members of Parabasalia, we recommended that future investigations on *D. fragilis* transmission consider mounting evidence for the role of a true cyst despite its rarity in human faecal specimens. The factors leading to cyst formation and further characteristics of this life cycle stage require further study.

ID: 151 / **CP17.1: 5**
Contributed abstract
Conference Topics: Diagnostics, Protozoa, Zoonoses
Keywords: Babesiosis, Babesia microti, Near-infrared spectroscopy, NIRS, Non-invasive diagnosis

Rapid and non-invasive detection of *Babesia microti* parasites using near-infrared spectroscopy and machine learning

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[1]Institute of Biomedicine and Glycomics, Griffith University, Gold Coast, Queensland; [2]School of the Environment, University of Queensland, St Lucia, Queensland

Babesiosis, caused by intraerythrocytic parasites of the genus *Babesia*, is an emerging zoonotic threat with a global distribution. Babesiosis ranges from asymptomatic to fulminant disease occurring predominantly in immunocompromised hosts, with fatality rates up-to 20%. Diagnosis traditionally relies on microscopic examination of blood smears; however, up-to 20% of infections are sub-microscopic, risking transmission via blood transfusion, organ transplantation, and the ixodid tick vector. While molecular and immunodiagnostic methods exhibit improved sensitivity, they are costly, time-consuming, technically complex, and invasive. Near-infrared spectroscopy (NIRS) utilises near-infrared electromagnetic energy (350–2500 nm) to generate spectral signatures reflective of specific chemical changes in a biological sample. When coupled with machine learning algorithms, diagnostic features can be extracted, allowing sample classification. NIRS has been successfully applied as a non-invasive malaria diagnostic in mice and humans; however analogous studies have not been performed with *Babesia* spp. This study evaluated the ability of NIRS to non-invasively detect *B. microti* in mice. The sensitivity and specificity of non-invasive detection was compared to invasive detection (blood spots). By situating NIRS alongside established molecular detection methods, our goal is to lay the groundwork for a rapid, reagent-free diagnostic that complements existing assays while enabling non-invasive babesiosis surveillance and enhanced donor screening.

CP15: Drugs & Drug Resistance 2 - 10 min talks sponsored by Institute for Biomedicine and Glycomics, Griffith University

Time: Thursday, 02/July/2026: 11:20am - 11:50am · *Location:* Lecture Theatre 1
Session Chair: Jacinta Macdonald, Griffith University
Session Chair: Rohith Kutty, Griffith University

ID: 223 / **CP15: 1**
Contributed abstract
Conference Topics: Drugs, Malaria, Microscopy
Keywords: peptides, membranes, antimicrobial peptide, nile red, laurdan

Anti-plasmodial peptide induces polarity and fluidity changes in host and parasite membranes without translocation

Sarah Woodcock[1], Jiwon Lee[2], Angus Rae[2], Daryl Webb[2], Melanie Rug[2], Nicole Lawrence[3], Brendan McMorran[1]
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Platelet Factor 4 Derived Internalisation Peptide (PDIP), based on the antimicrobial peptide-like domain of human PF4, exhibits activity against *Plasmodium*. PDIP rapidly kills parasites by penetrating *Plasmodium*-infected erythrocytes and destroying the digestive vacuole. Why PDIP penetrates only infected but not healthy erythrocytes and destroys only the digestive vacuole is unclear. We hypothesised that differences in lipid composition between infected erythrocyte and parasite membranes alter membrane polarity and fluidity, thereby facilitating PDIP's differential interactions.

To test this, we used the membrane dyes Nile Red and Laurdan, which report polarity and fluidity through emission-wavelength shifts quantified as ratiometric indices. These ratios were measured for uninfected and *Plasmodium*-infected erythrocyte membranes, and the intracellular parasite, with and without PDIP treatment. We found comparable fluidity between infected and uninfected erythrocyte membranes. Polarity differed significantly, ranking from most to least: intracellular parasite > uninfected > and infected erythrocyte membranes. These polarity differences may contribute to PDIP's ability to selectively enter infected cells and suggested that parasites contain more polar lipids that may facilitate PDIP's activity. PDIP treatment significantly increased polarity and reduced fluidity for all membrane types. This indicates a non-specific interaction of the peptide independent of translocation that alters membrane properties non-destructively.

ID: 187 / **CP15: 2**
Contributed abstract
Conference Topics: Drugs, Malaria
Keywords: Plasmodium falciparum, antimalarial development, digestive vacuole, haemoglobin digestion

The *Plasmodium falciparum* digestive vacuole is the site of action for second-generation bis-triazines and related antimalarial candidates

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Widespread resistance to all current antimalarials threatens the control and eradication of malaria. Second-generation bis-triazines display low nanomolar potency and fast-killing asexual *P. falciparum*. However, the novel mechanism of action (MOA) remains unknown.

I*n vitro* combination drug-pulse assays using various inhibitors were performed to identify potential modulators of bis-triazine activity. We also included two antimalarial candidates currently under development with the Medicines for Malaria Venture (MMV) with some structural similarity to the bis-triazines. E64d, a cysteine protease inhibitor, bafilomycin A1, a V-ATPase inhibitor and chloroquine all caused antagonism of trophozoite-stage activity across the bis-triazine analogues and MMV candidates (between 2 and 10-fold increases in IC_{50}). All three activity modulators are known to localise to the digestive vacuole and indicates potential involvement of the haemoglobin digestion pathway in the MOA of these series.

3-hour ring-stage survival assays with an artemisinin-resistant clinical isolate and a *Pf*3D7 line genetically modified to induce knockdown of essential falcipain-3 resulted in decreased activity (up to 20-fold increases in IC_{50}) for both MMV candidates and one bis-triazine analogue. The current lead bis-triazine analogue, however, observed no change or slight hypersensitisation. Uninterrupted haemoglobin digestion appears to be vital for these compounds to maintain their fast-killing activity.

ID: 188 / **CP15: 3**
Contributed abstract
Conference Topics: Apicomplexa Biology, Biochemistry, Drugs, Malaria
Keywords: Plasmodium falciparum, antimalarial, mitochondrial electron transport chain, strobilurin, drug resistance

Characterising Novel Mitochondrial Electron Transport Chain Inhibitors in Apicomplexan Parasites

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Malaria remains one of the most devastating infectious diseases globally. Resistance to frontline antimalarials continues to compromise control efforts, highlighting the urgent need for novel therapeutic strategies. We previously identified the strobilurin compound MMV1794211 as a highly potent antiplasmodial agent with low-picomolar activity against blood-stage *Plasmodium falciparum*. Biochemical and enzymatic analyses demonstrated that MMV1794211 targets Complex III of the parasite mitochondrial electron transport chain (mtETC), a clinically validated antimalarial target. Our recent structure-activity

relationship studies have identified key chemical elements underlying its potency and enabled the synthesis of derivative compounds with favourable antiplasmodial activity and enhanced selectivity for parasites over human cells. *In vitro* evolution experiments generated parasites carrying a single mutation in cytochrome *b* conferring high-level resistance to MMV1794211. Drug sensitivity profiling of the mutant revealed pan-strobilurin resistance; however, no cross-resistance was observed with established antimalarials or other mtETC inhibitors currently under investigation. Ongoing work includes assessing the fitness costs associated with strobilurin-resistance mutations and developing derivative compounds that remain active in resistant parasites. Collectively, our study provides a comprehensive evaluation of strobilurins as a chemically distinct class of antiplasmodial agents and highlights their promise for future therapeutic development.

CP16: Horses & Cows - 10 min talks

Time: Thursday, 02/July/2026: 11:20am - 11:50am · *Location:* Lecture Theatre 2
Session Chair: Abdul Jabbar, The University of Melbourne
Session Chair: Narelle Dybing, Murdoch University

ID: 133 / **CP16: 1**
Contributed abstract
Conference Topics: Bioinformatics, Diagnostics, Livestock Parasites, Protozoa, Veterinary Parasitology
Keywords: Tritrichomonas foetus, bovine trichomonosis, long-read sequencing, Oxford Nanopore Technologies, venereal pathogens

Simultaneous Detection of Bovine Venereal Pathogens in Bulls Using Long-Read Sequencing

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Bovine Trichomonosis, caused by the protozoan parasite *Tritrichomonas foetus,* poses significant economic threats to the global cattle industry. In Australia, disease control relies heavily on accurate diagnosis followed by culling infected bulls. However, conventional diagnostic methods, including culture and quantitative PCR (qPCR), are limited by the complex microbial environment of the bull prepuce, which leads to false-negative and positive results. These arise from *T. foetus* low abundance and cross-reactive trichomonad species.

To address these limitations, we developed and tested a diagnostic test using Oxford Nanopore Technologies (ONT) long-read sequencing to simultaneously detect multiple venereal-transmitted organisms, *T. foetus* and *Campylobacter fetus* subsp. *venerealis*. Using artificially spiked DNA samples and field samples, our pipeline improved sensitivity and specificity, achieving detection limits for *T. foetus* down to 0.4 ng and identifying 50.0% more positives (n =9) than qPCR (n =6) across samples from persistently infected bulls. Simultaneously detecting *C. fetus* subsp. *venerealis* down to 0.04 ng, lower than its 0.1 ng qPCR limit.

These findings highlight the potential of ONT long-read sequencing as a robust, single-test alternative for accurately detecting *T. foetus* and *C. fetus subsp. venerealis* in bulls. Its adoption would enhance disease surveillance and help safeguard the Australian cattle industry.

ID: 110 / **CP16: 2**
Contributed abstract
Conference Topics: Immunology, Proteomics, Veterinary Parasitology
Keywords: Cyathostomin, equine, saliva, encysted larvae

Exploration of equine saliva proteins as a source of immune biomarkers that may reflect the mucosal response to a gastrointestinal worm infection.

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[1]Federation University, Australia; [2]Monash Proteomics and Metabolomics Platform, BDI, Monash University, Clayton, Australia

Cyathostomin infections caused by small strongyle worms are among the most prevalent and clinically significant parasitic diseases affecting horses worldwide. Although considerable research has focused on identifying host–parasite interaction markers in serum and faecal samples, the salivary proteome remains largely unexplored. Saliva represents a non-invasive biofluid that may enable monitoring of infection status and immune responses, while providing access to both host and parasite derived proteins.

In this study, the salivary proteome of four naturally infected horses was analysed at two timepoints using mass spectrometry. Two preparation approaches were evaluated to determine the feasibility of saliva for parasite protein detection: direct digestion of neat saliva using S-Trap protocol, and on-bead enrichment method employing hydrophilic interaction liquid chromatography (HILIC) beads to concentrate polar and extracellular vesicle-associated proteins. Samples were analysed by data-independent acquisition (DIA) on an Orbitrap Astral mass spectrometer, searched in Spectronaut using DirectDIA, and analysed in the DIA Analyst platform.

Across all samples, 2,535 proteins (2,448 groups) were identified. Among these, 34 *Cylicocyclus nassatus* proteins were detected with more than one unique peptide, including 28 proteins with no detectable homology to the host proteome. These findings demonstrate the potential of equine saliva for detecting parasite and host immune proteins.

ID: 243 / **CP16: 3**
Contributed abstract
Conference Topics: Livestock Parasites, Veterinary Parasitology
Keywords: Gastrointestinal nematodes, Haemonchus spp, Cooperia spp, faecal egg count, larval culture

Gastrointestinal parasite prevalence in dairy cattle in subtropical Queensland: A paddock-based faecal sampling study across commercial farms

Md Anowarul Haque[1], Benjamin Wood[1], Ricardo J. Soares Magalhães[1,2], Shahab Ranjbar Nezhad Isfahani[1], Matthew Ball[3], Swaid Abdullah[1]

[1]The University of Queensland, Australia; [2]Children's Health and Environment Program, UQ Children's Health Research Centre, The University of Queensland, Australia; [3]Virbac, Australia

Gastrointestinal (GI) nematodes are a major constraint to dairy productivity and heifer growth globally and are of particular concern in subtropical Queensland. In this study, we collected fresh faecal samples from the paddock of 43 dairy farms, categorised by age into six groups: calves, weaners, heifers, springers, milking cows, and dry cows. Faecal egg counts (FEC) were performed using the modified McMaster technique, and species identification was conducted through larval culture, with morphological characterization of third-stage (L_3) larvae. All 43 farms had at least one age group positive for GI parasites, indicating widespread endemic infection throughout the region. The overall parasite prevalence was 78.7%, with infection burden varying by age: heifers had the highest FEC (mean EPG of 226.7; 26 to 553), followed by weaners (235.7; 31 to 651), and milking cows (75.8; 5 to 180). Calves were mostly uninfected. Egg counts did not differ significantly between farms (Kruskal–Wallis, $p > 0.05$) but varied strongly within age groups ($H=107.5$, $p<0.001$), with a clear age-dependent gradient ($p < 0.001$). *Haemonchus* spp. (47.2%) and *Cooperia* spp. (43.1%) dominated, comprising ~90% of larvae, underscoring the need for regular, age-based faecal monitoring to guide targeted parasite control on dairy farms in subtropical Queensland.

CP16.1: Horses & Cows - 5 min talks

Time: Thursday, 02/July/2026: 11:50am - 12:05pm · *Location:* Lecture Theatre 2
Session Chair: Abdul Jabbar, The University of Melbourne
Session Chair: Narelle Dybing, Murdoch University

ID: 231 / CP16.1: 1
Contributed abstract
Conference Topics: Diagnostics, Epidemiology, Genomics, Molecular Biology, Strongyloides, Veterinary Parasitology
Keywords: Strongyloides westeri, Thoroughbred foals, qPCR, Australia, Strongyloides species

Molecular epidemiology of *Strongyloides westeri* in Australian Foals

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Strongyloides westeri is an intestinal threadworm of foals (≤16 weeks) that can cause dermatitis, diarrhoea and respiratory signs depending on parasite burden and transmission route. Despite its clinical relevance, epidemiological data on infections in Australian foal populations are limited. Additionally, traditional diagnostic methods such as faecal flotation techniques may have lower diagnostic sensitivity, highlighting the need for improved molecular diagnostic methods. This study aims to develop and validate a quantitative PCR (qPCR) assay for the detection and quantification of *S. westeri* in faecal samples from Australian foals. Following McMaster faecal egg count (FEC), DNA extracted from samples will be screened for *Strongyloides* spp. using a qPCR assay targeting the 18S ribosomal RNA region. The positive amplicons will be submitted for sanger DNA sequencing. Sequence analysis will be undertaken using Geneious Prime and alignments made with GenBank references, followed by species identity confirmation using phylogenetic analysis. Although experimental work is ongoing, it is anticipated that the qPCR assay will have greater sensitivity and accuracy compared to the traditional FEC. This will be the first study to provide molecular epidemiological data of *S. westeri* in Australian foals and support sustainable equine health management.

ID: 178 / CP16.1: 2
Contributed abstract
Conference Topics: Bioinformatics, Education/Outreach, Epidemiology, Livestock Parasites, Veterinary Parasitology, Zoonoses
Keywords: Tick-borne diseases Knowledge, attitudes and practices (KAP), Cattle producers

Knowledge, Attitudes, and Practices of Cattle Farmers Regarding Ticks and Tick-Borne Diseases in South-Western Western Australia

Abdul Ghaffar Qamar[1], Amanda Duarte Barbosa[1], Joshua W Aleri[2], Tracey Kreplins[3], Charlotte Louise Oskam[1]
[1]Murdoch University, Perth Western Australia, Australia; [2]The University of Queensland, Australia; [3]Department of Primary Industries and Regional Development, Western Australia

Ticks and tick-borne diseases (TTBDs) are an emerging threat to human and animal health and livestock productivity. This study assessed knowledge, attitudes and practices (KAP) regarding TTBDs among cattle producers in Western Australia. A 44-item questionnaire (covering farm demographics, knowledge of tick-borne diseases, attitudes towards prevention and control, and on-farm management practices) was utilised to survey cattle producers in the region. The survey was distributed through regional biosecurity groups, field visits, and online platforms. Among 108 producers participated, 83 were included in the final analysis. Overall knowledge was low, no respondent (0/83) correctly answered all six knowledge questions, and the mean score was less than 1 out of 6. Most participants recognised the adverse effects of ticks on cattle health (81.3%), however, awareness of zoonotic risks (18.8%) and diseases such as bovine theileriosis (BATOG) (31.3%) was limited. Only 26.5% of the respondents reported favourable attitudes towards tick prevention and control. Management practices were variable, with 30.7% classified as poor, 47.6% as moderate, and 21.5% as optimal. Acaricides were commonly used (63.8%). This study reveals a critical low knowledge among cattle producers in WA and inconsistent control practices. Improved on-farm tick management including appropriate control methods, and disease awareness is required.

ID: 127 / CP16.1: 3
Contributed abstract

Conference Topics: Parasites of companion animals, Wildlife parasitology
Keywords: Gastrointestinal parasites, Prevalence study, Horse Health, Helminths, Fecal examination

Prevalence of different gastrointestinal parasite horse infections in Riyadh, Saudi Arabia

Wafa Almegrin, Wadha Alsahli, Afaf Alhazzaa, Rawan Alotaibi, Dalal Alotaibi, Rania Aldosari
Princess Nourah bint Abdulrahman University, Saudi Arabia

Gastrointestinal parasites are a significant health concern for horses, affecting their overall health and performance. Detecting intestinal parasites in horses is crucial for maintaining their health and preventing disease outbreaks, which can lead to significant economic and performance-related losses. This research provides essential data that can inform better management practices and parasite control strategies, ultimately enhancing horse health and productivity. This study was conducted to estimate the prevalence of different gastrointestinal parasites in horses in Riyadh, Saudi Arabia, and to investigate the relationship between infection rates and the horses' age, sex, and species. A total of 113 fecal samples from horses were gathered and examined using NaCl flotation and direct fecal smear techniques to detect gastrointestinal parasites. The results showed that among the 113 samples examined, 44 (38.93%) were found positive for various gastrointestinal parasites. The detected parasites included *Entrobius* spp. (10.6%), *Eimeria* spp. (6.19%), *Anoplocephala* spp. (2.65%), *Parascaris equorum* (1.76%), *Ascaris* spp. (1.76%), and *Gastrodiscus* spp. (0.88%). Additionally, 29.2% of the infections were attributed to unidentified oocysts, larvae, or eggs. These findings suggest that gastrointestinal parasites are common in horses in Riyadh, Saudi Arabia.

CP15.1: Drugs & Drug Resistance 2 - 5 min talks sponsored by Institute for Biomedicine and Glycomics, Griffith University

Time: Thursday, 02/July/2026: 11:50am - 12:15pm · *Location:* Lecture Theatre 1
Session Chair: Jacinta Macdonald, Griffith University
Session Chair: Rohith Kutty, Griffith University

ID: 271 / CP15.1: 1
Contributed abstract
Conference Topics: Apicomplexa Biology, Drugs, Malaria
Keywords: antimalarial, drugs, malaria

Investigating the potential of robenidine analogues as antiplasmodial compounds

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The treatment of malaria, an infectious disease caused by *Plasmodium* parasites, relies on numerous chemotherapies that are hampered by drug resistance. New antimalarial drugs with mechanisms of action different to currently used drugs are required to combat *Plasmodium* drug resistance. Robenidine is an antiparasitic drug with modest activity against *P. falciparum* (50% growth inhibitory concentration; IC_{50} 0.76 μM) and unknown mode of action. To aid in the identification of new antimalarial drug candidates, a library of robenidine analogues was assessed for improved activity and selectivity for *P. falciparum* over mammalian cells. Multiple compounds with hit and early-lead activity and selectivity were identified, including NCL123 and NCL146, which demonstrated *P. falciparum* IC_{50} values <0.1 μM and selectivity indices of >100. While *in vivo* studies with NCL123 and NCL146 demonstrated that further optimisation is needed to facilitate cures in mice infected with *P. berghei* ANKA, both compounds were well-tolerated and structural activity relationships have been useful in identifying avenues to improve this activity. Preliminary mode of action studies with NCL146 also suggest that this compound may have an unexploited mode of action associated with lipid biosynthesis, membrane function and cellular trafficking.

ID: 206 / CP15.1: 2
Contributed abstract
Conference Topics: Apicomplexa Biology, Cell Biology, Drugs, Malaria, Protozoa
Keywords: proguanil, malaria, Plasmodium, field isolates, slow action activity

The malaria drug proguanil demonstrates slow action *in vitro* activity against *P. falciparum* field isolates from Uganda.

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Proguanil is a used in combination with atovaquone to prevent and treat malaria. Activity of proguanil is thought to be due to its ability to potentiate atovaquone activity and the potent antiplasmodial activity of its *in vivo* metabolite, cycloguanil. *Plasmodium falciparum* resistance to cycloguanil is known to be due to mutations in the dihydrofolate reductase, but studies on proguanil resistance are lacking given it was essentially considered a prodrug. We overturned this dogma showing that proguanil has slow action activity against *P. falciparum* laboratory lines *in vitro* (*Pf*3D7 IC_{50} 110 nM)[1]. Here, we investigated thirteen culture-adapted *P. falciparum* isolates collected in eastern Uganda with low *ex vivo* sensitivity to proguanil[2]. The *in vitro* sensitivity of the culture-adapted isolates to proguanil was assessed using 48 h, 72 h and 96 h growth-inhibition assays. While proguanil resistance was not confirmed in these studies, proguanil demonstrated slow-action activity against field isolates with 96 h IC_{50} values of approximately 80-430 nM. These data extend our understanding of proguanil action to more clinically relevant field isolates, adding to findings that indicate that the antiplasmodial activity of the proguanil-atovaquone combination may be more complicated than previously thought.

ID: 205 / **CP15.1: 3**
Contributed abstract
Conference Topics: Bioinformatics, Cell Biology, Drugs, Genomics, Malaria, Molecular Biology, Proteomics
Keywords: Malaria, Plasmodium falciparum, Antimalarial drug resistance, Atovaquone–proguanil, CRISPR-Cas9 gene editing

Investigating putative target/s of the malaria drug proguanil identified using solvent-induced protein precipitation

Rohith Kutty[1], Wisam Dawood[1], Gillian Fisher[1], Jacinta Macdonald[1], Patrick Tumwebaze[1], Yijia Ji[3], Joshua Morrow[3], Andrew Riches[2], John Ryan[2], Ghizal Siddiqui[3], Darren Creek[3], Tina Skinner-Adams[1], Katherine Andrews[1]
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Malaria remains a persistent global health threat leading to around 600,000 deaths annually. Resistance of Plasmodium parasites to most available drugs is significantly impacting prevention and control efforts. Combating resistance requires a clear understanding of drug mode of action, which remains incomplete for several current options. One such drug combination is atovaquone-proguanil. Atovaquone targets the cytochrome bc1 complex, and proguanil, originally developed as a prodrug, is converted in vivo to the dihydrofolate reductase (DHFR) inhibitor cycloguanil. While proguanil has been shown to potentiate the activity of atovaquone in vitro, for decades proguanil was thought to lack intrinsic activity. However, we showed that proguanil has a potent slow-acting in vitro antiplasmodial activity that is distinct from the folate metabolism pathway1. To try to identify the target/s of proguanil's slow-action activity, we employed solvent-induced protein precipitation combined with mass spectrometry to assess protein stability in the presence of proguanil. Here, data will be presented on one of several candidate proteins stabilized by proguanil. Conditional knockdown is being performed in wildtype Plasmodium falciparum using TetR-DOZI and CRISPR-Cas9 Guide RNA constructs. Future work will include confirmation of knockdown and phenotypic assays with proguanil to assess changes in sensitivity to this drug.

ID: 216 / **CP15.1: 4**
Contributed abstract
Conference Topics: Livestock Parasites, Veterinary Parasitology
Keywords: Blowfly, Lucilia cuprina, flystrike, RNAi, compound screening

Discovery and Validation of Novel Drug Targets for Sustainable Blowfly Control and Improved Sheep Welfare

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Department of Veterinary Biosciences, Melbourne Veterinary School, Faculty of Science, The University of Melbourne

The Australian sheep blowfly, *Lucilia cuprina*, is the principal cause of flystrike (cutaneous myiasis) in sheep, incurring losses of ~AU$320 million annually and inflicting severe welfare impacts in sheep. Current treatment includes insecticides and surgical mulesing; however, heavy reliance on insecticides has accelerated resistance, while mulesing draws strong public opposition due to animal welfare concerns—undermining both ethics and economic outcomes. Sustainable alternatives will be explored through the discovery and validation of novel blowfly-specific molecular targets identified using population genetic data from blowfly populations across Australia. RNA interference (RNAi) will be used to silence top molecular targets and protein modelling, and larval bioassays will be used for identifying and testing top candidate compounds. This project is expected to provide validated targets and candidate compounds for *L. cuprina* as the basis for novel larvicides to overcome existing insecticide resistance and reduce dependence on mulesing.

ID: 141 / **CP15.1: 5**
Contributed abstract
Conference Topics: Veterinary Parasitology
Keywords: Gastrointestinal parasites; anthelmintic resistance; goats; sheep; Sri Lanka; systematic review

A systematic review of epidemiology, anthelmintic resistance and economic impact of gastrointestinal parasites in Sri Lankan small ruminants

Suraj Walimunige[1], Dulari Thilakarathne[2], Abdul Ghafar[1], Ghazanfar Abbas[1], Endris Ali[1], Anil Kalupahana[2], Jayanthe Rajapakshe[2], Robin Gasser[1], Abdul Jabbar[1]
[1]University of Melbourne, Australia; [2]University of Peradeniya, Sri Lanka

Gastrointestinal parasitic infections (GIP) are a major constraint to small ruminant production in Sri Lanka, yet a comprehensive synthesis of their epidemiology and anthelmintic resistance (AR) is lacking. This systematic review synthesised evidence on GIP, AR, and key knowledge gaps. A search of Web of Science, PubMed, Scopus, and CAB Abstracts (1971–January 2025), cross-checked with Google Scholar, identified 34 studies. Study quality was assessed using the Joanna Briggs Institute (JBI) prevalence checklist, and apparent prevalence was adjusted to true prevalence using the Rogan–Gladen estimator. In goats, prevalence ranged from 74–78%, while sheep showed seasonal prevalence of 84% (dry) and 92% (wet), with higher true prevalence after diagnostic adjustment. Predominant nematodes in goats were *Haemonchus contortus* (90%), *Oesophagostomum columbianum (88%), Trichostrongylus colubriformis (76%)*, and *Strongyloides* spp. (72.5%). In sheep, *Haemonchus contortus*, *Toxocara* spp., and *Trichuris* spp. were common. AR was documented against benzimidazoles and levamisole, with *Haemonchus* spp. consistently implicated. Estimated economic losses in goats were LKR 230 million, based on limited data. Studies were heterogeneous in diagnostics, limiting comparability. These findings highlight critical gaps in parasite prevalence, species distribution, and AR, and underscore the need for coordinated, standardised national surveillance for sustainable parasite control in Sri Lanka.

CP17.2: Epidemiology & Diagnostics 5 min talks

Time: Thursday, 02/July/2026: 12:05pm - 12:15pm · *Location:* Lecture Theatre 3
Session Chair: Deepani Fernando, QIMR Berghofer
Session Chair: Luke Hall, St Vincent's Hospital Sydney

ID: 272 / **CP17.2: 1**
Contributed abstract
Conference Topics: Diagnostics, Epidemiology, Protozoa
Keywords: surveillance, soil-transmitted helminths, intestinal protozoa

Validation of a Wastewater Surveillance Protocol for Soil-Transmitted Helminths and Intestinal Protozoan Parasites

Emily Grahn[1], Daphne Maneniaru[1], David Sellars[1], Kirstin Ross[2], Constantin Constaninoiu[3], Richard S Bradbury[1]
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Intestinal parasitic diseases remain a significant public health burden globally. There is an increasing need for a versatile, sensitive, and cost-effective mechanism for near-elimination and post-elimination surveillance systems for population-wide. Traditional faecal sampling from individuals across large populations is expensive, intrusive, and impractical. Wastewater-based epidemiology is a population-level surveillance tool.

A series of multiplex qPCRs was chosen to detect common parasites of public health importance. Limits of detection and specificity for human-infecting species were determined. Influent wastewater from an urban treatment plant was spiked with a known concentration of cysts, eggs, and larvae of the target parasites (*Giardia duodenalis*, *Ascaris lumbricoides*, *Trichuris trichiura*, *Necator americanus*) and processed using three concentration protocols. DNA was extracted using Powersoil Pro (Qiagen) kits, and four Taqman multiplex qPCRs were performed, including human DNA and internal amplification control targets.

The diagnostic qPCRs chosen demonstrated high sensitivity and specificity. Wastewater application of the multiplex was successful. Initial results indicate that centrifugation or sieve selection prior to DNA extraction was necessary to detect parasitic DNA in wastewater samples. Processing raw wastewater or wastewater sediments proved ineffective.

This work establishes a methodological basis for implementing wastewater-based population-level surveillance for soil-transmitted helminths and intestinal protozoa.

ID: 192 / **CP17.2: 2**
Contributed abstract
Conference Topics: Diagnostics, Ecology, Protozoa
Keywords: Acanthamoeba, parasitic sinusitis, rhinosinusitis.

Incidental finding of amoeba on nasal cavity of an immunocompetent patient. A case report and review of the literature.

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[1]Microbiology registrar, John Hunter Hospital; NSW Health Pathology.; [2]Anatomical pathologist, ACT Pathology, Canberra Health Services.; [3]Microbiologist and Infectious Diseases physician, ACT Pathology, Canberra Health Services

A 71-year-old man was found incidentally to have a crusted lesion on the floor of the nose during resection of a pituitary tumour. Microscopy showed an inflammatory infiltrate with ovoid cells, staining positive with PAS and silver raising concern for a free-living amoeba (FLA). However, multiplex PCR for *Acanthamoeba* species, *Balamuthia mandrillaris* and *Naegleria fowleri* was negative. There was no clinicoradiological evidence of meningoencephalitis. We reviewed the biology, clinical features and diagnosis of FLA infections.

Amongst 26 cases of rhinosinusitis, *Acanthamoeba* species were implicated in 25 and *Entamoeba histolytica*in one. The average age of patients was 43 mostly males. 11 patients died. Histopathology was used for diagnosis in 21 cases, PCR in 7. In two cases both were utilised. Culture was used in 1 case. All Acanthamoeba cases were associated with immunosuppression; HIV (17), CLL and solid organ transplant (3), haematopoietic stem cell transplant (1) and one hypogammaglobulinaemia.

Our asymptomatic case represents a dilemma, without literature to guide management in an incidental finding of amoeba in histopathological specimen especially after breach of the nasal mucosa for biopsy. FLA are ubiquitous and are found as carriers in the nose of volunteers. Disease likely affects immunosuppressed patients with compatible inoculation route.

CP20: Zoonoses & One Health 15 min talks

Time: Thursday, 02/July/2026: 1:30pm - 2:00pm · *Location:* Lecture Theatre 3
Session Chair: Darren Gray, QIMR Berghofer
Session Chair: Jessica Scott, James Cook University

ID: 236 / **CP20: 1**
Contributed abstract
Conference Topics: Genomics, Helminthology, One Health, Parasites of companion animals, Parasites of dogs, Strongyloides, Veterinary Parasitology, Zoonoses
Keywords: Strongyloides, hookworms, dogs, zoonoses, neglected tropical disease

Genetic diversity and transmission dynamics of soil-transmitted helminths in humans and dogs in Vanuatu

Molly Waldron[1], Lucas Huggins[1], Patsy A. Zendejas-Heredia[1], Sze Fui Hii[1], Ian Douglas[2], Nick Sangster[2], Clare Anstead[1], Paolo Bareng[3], Prudence Rymill[4], Fasihah Taleo[5], Susana Vaz Nery[3], Vito Colella[1]
[1]The University of Melbourne, Australia; [2]Vets Beyond Borders, Australia; [3]Kirby Institute, UNSW, Australia; [4]Ministry of Health, Vanuatu; [5]World Health Organization, Vanuatu

Soil-transmitted helminths (STHs) cause some of the most prevalent neglected tropical diseases worldwide. Despite this, our understanding of diversity across hosts and extent of animal contributions to human infections remains limited. We screened 2,285 humans and 148 dogs from Vanuatu for STHs of public health importance using multiplex qPCR. A subset (n=148 humans, 148 dogs) were sequenced using a nanopore-based approach to characterise the 'nemabiome'. Given evidence for zoonotic transmission of *Ancylostoma ceylanicum and Strongyloides stercoralis*, we also sequenced a large region of the cox1 gene in these species to assess haplotype diversity and infer zoonotic STH transmission dynamics. Overall, qPCR detected >1 STH in 48.5% of humans and 97.3% of dogs. Specifically, a prevalence of 8.9% and 64.9% were observed for *A. ceylanicum* and 3.0% and 47.3% for *Strongyloides* spp. in humans and dogs, respectively. Nemabiome analysis revealed a greater diversity of intestinal parasites than that detected by qPCR in both hosts. These findings indicate a high abundance of STHs across Vanuatu, supporting the need for mass-drug administration. Molecular data suggest canine-human transmission may be occurring, however further sequencing at nuclear and mitochondrial loci will resolve transmission dynamics and inform the need for control approaches that include animal reservoirs.

ID: 181 / **CP20: 2**
Contributed abstract
Conference Topics: Epidemiology, Genomics, Helminthology, Livestock Parasites, Parasites of dogs, Veterinary Parasitology, Zoonoses
Keywords: Echinococcus granulosus, hydatid cyst, G3 genotype, mitochondrial genome, Pakistan

Unraveling the whole mitochondrial genome of the highly endemic G3 genotype of Echinococcus granulosus from Pakistan: an emerging epidemiological concern

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Introduction

Echinococcus granulosus sensu stricto (s.s.) is a zoonotic parasite that infects a wide spectrum of hosts. A substantial public health concern, this cestode causes socioeconomic burdens to resource-limited pastoral communities globally. The G1 and G3 genotypes of E. granulosus s.s. are associated with human and animal infection, with G1 predominating. However, an opposite trend has been observed in Pakistan, where the G3 genotype is more common.

Materials and Methods

In the present study, genomic DNA was extracted from hydatid cysts obtained from cattle in Multan, Pakistan and processed for whole-genome sequencing (Illumina NovaSeq). Complete mitochondrial genomes were assembled and population genetic analysis was performed and compared to those from public databases.

Results

The results indicated that the G3 genotype of E. granulosus s.s. is highly prevalent among the cattle of Pakistan. Using whole mitogenome data (13,610 bp), when compared to the G1 reference sequence, G3 exhibited substantial variation at 48 diagnostic positions, indicating a distinct mitochondrial lineage. Genetic variability indices revealed high haplotype diversity and low nucleotide diversity.

Conclusions

This study reports the first complete mitogenome of the G3 genotype of E. granulosus s.s. from Pakistan, providing a foundation for future genetic analyses from other regions of the world.

CP19: Immunology 2 - 15 min talks

Time: Thursday, 02/July/2026: 1:30pm - 2:15pm · *Location:* Lecture Theatre 2
Session Chair: Alicja (Ala) Tabor, The University Of Queensland
Session Chair: Hannah Siddle, The University of Queensland

ID: 132 / **CP19: 1**
Contributed abstract
Conference Topics: Host-parasite interactions, Malaria
Keywords: Plasmodium falciparum, PfEMP1, Cerebral Malaria, System Serology, Machine Learning

Investigating Antibodies that Protect from Cerebral Malaria in Kenyan Children

Yuchi Ji[1], Isobel S Walker[1], Niamh Meagher[1], Wina Hasang[1], Sidney Ogolla[2], David Midem[3], J Alexandra Rowe[4], Stanley Otoboh[4], Elizabeth H Aitken[1,5], James W Kazura[6], Stephen J Rogerson[1]

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Cerebral malaria, a severe manifestation of *Plasmodium falciparum* infection, results from PfEMP1-mediated sequestration of infected erythrocytes in the brain microvasculature. PfEMP1 also promotes rosetting, and antibodies against specific domains are linked to protection and reduced disease severity. In this study, plasma from Kenyan children with cerebral or uncomplicated malaria collected at hospital enrolment, six weeks post-infection, and six months post-infection was used to characterise targets and features of antibody responses. Antibody classes, subclasses, and engagement of Fcγ receptors and complement C1q were assessed using 28 PfEMP1 antigens previously associated with severe malaria, along with five merozoite and one sporozoite antigens by multiplex immunoassay. Machine learning and tensor decomposition analyses revealed that Fc-mediated features, including FcγRIIIa and FcγRIIIb engagement with rosetting DBLα and EPCR-binding CIDRα1 domains, were higher in children with uncomplicated malaria compared to those with cerebral malaria. Subsequent linear mixed-effect modelling shows that these antibody responses were sustained over time. These findings offer insights into the development of malaria therapeutic interventions to reduce cerebral malaria morbidity and mortality in children.

ID: 159 / **CP19: 2**
Contributed abstract
Conference Topics: Immunology, Malaria
Keywords: malaria, Plasmodium vivax, functional antibodies, antigens, protective immunity

Functional antibodies to *Plasmodium vivax* merozoite antigens are associated with protection against clinical malaria

Chau Quang[1,2], Takaaki Yuguchi[3], Eizo Takashima[3], Ivo Mueller[1,2], Rhea Longley[1,2,4]

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No prophylactic vaccines currently exist for *Plasmodium vivax*, the predominant cause of malaria outside Africa. As natural infection with *P. vivax* induces antibodies with broad specificity, the key targets of protective immunity remain unclear. Identifying immune correlates of protection against *P. vivax* is critical to inform vaccine development.

We characterised functional antibody responses and evaluated their association with subsequent malaria in a PNG cohort of 185 children aged 1-3yo, using incidence rate ratios (IRR) from a negative binomial generalised estimating equation whereby IRR<1 indicates reduced infection risk. Blood samples were collected at enrolment, with active surveillance for clinical malaria and *P. vivax* infection by PCR every 2 weeks for 16 months. Total IgG, IgG1, IgG3, Fc receptor binding and C1q fixation to seven *P. vivax* merozoite antigens (Pv12/Pv32/Pv41/Pv50/PvEXP1/PvMSP10/PvRBP2b) were quantified using multiplex immunoassays.

PvMSP10 generated robust functional antibody responses that were significantly associated with protection to *P. vivax* infection, including IgG, IgG1, IgG3 (IRR: 0.3), and binding to phagocytic FcgRIIA and cytotoxic FcgRIIIA (IRR: 0.35). Pv32, Pv41, Pv50 and PvEXP1 induced similar protective antibody responses but to a weaker extent. This suggests that PvMSP10 can stimulate protective immunity, highlighting functional antibodies as a correlate of protection to *P. vivax* infection.

ID: 138 / **CP19: 3**
Contributed abstract
Conference Topics: Host-parasite interactions, Immunology, Malaria, Vaccines
Keywords: Malaria, CD8, Vaccine, Memory

Human *Plasmodium falciparum* sporozoite effector-memory CD8$^+$ T cells exhibit epitope-specific activation and display distinct T cell receptor clustering

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Malaria, caused by *Plasmodium falciparum* (Pf), remains a major global health burden. Despite strong evidence for a critical role for CD8$^+$ T cells in protective immunity, and the importance of the liver-stage as a target, the specific mechanisms by which CD8$^+$ T cells target particularly *Plasmodium*-infected liver cells remain unclear, particularly in humans. To investigate this, we studied human *Pf* sporozoite (SPZ)-specific memory CD8$^+$ T cells using an *in vitro* antigen-presenting cell co-culture model. Repeated stimulation with *Pf* SPZ drove differentiation into highly activated effector-memory T cells expressing CD137, IFN-γ, and perforin. T cell receptor sequencing revealed distinct clusters, indicating clonal expansion of *Pf*-specific T cells.

When stimulated with known liver-stage epitopes (CSP, TRAP, and LSA-1), only the circumsporozoite protein-related antigen precursor (CRA) epitope GLLGNVSTV induced strong activation. This epitope triggered upregulation of activation markers (CD137, IFN-γ) and cytotoxic molecules (perforin, granzyme A, and granzyme B), demonstrating robust recognition by *Pf*-specific memory T cells. This study advances our understanding of human *Pf* SPZ memory $CD8^+$ T cell responses and provides a novel approach for dissecting antigen specificity and memory development which would inform the design of more effective malaria vaccines.

CP18: Cells, Molecules & Genes 3 - 10 min talks

Time: Thursday, 02/July/2026: 1:30pm - 2:40pm · *Location:* Lecture Theatre 1
Session Chair: Shilpa Kapoor, The University of Melbourne
Session Chair: Balu Balan, Walter and Eliza Hall Institute

ID: 201 / **CP18: 1**
Contributed abstract
Conference Topics: Bioinformatics, Genomics, Helminthology, Host-parasite interactions, Livestock Parasites, Veterinary Parasitology
Keywords: Chemosensation, Transcriptomics, Microfluidics, GPCR

In vitro studies support early *Ascaris* infection driven by specific chemotactic attraction to host liver

Pradip Roy[1,2], Joy Liu[2], Peter Thurgood[3], Amrita Vijay[1,2], Louise Baker[1,2], Balu Balan[1,2], Khashayar Khoshmanesh[3], Aaron Jex[1,2]

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Ascaris spp. infect approximately 804 million people, causing substantial malnutrition, stunting and morbidity in school-aged children in low-income populations, exceeding 750,000 Disability Adjusted Life-Years annually. Current single-dose oral anthelmintics offer no sustained protection nor interruption of transmission. Interventions that prevent infection are needed. During early infection, *Ascaris larvae* migrate from the host intestine to the liver and lung before maturation in the small intestine. This process is thought to be passively directed by host blood flow; however, multiple studies suggest active chemotaxis. Here, we use larval agar migration assays and microfluidic chemotaxis arenas to demonstrate that freshly-hatched *Ascaris suum* larvae exhibit liver-specific chemotaxis *in vitro*, including increased migration distance, speed, and directional movement toward liver gradients, and reduced turning behaviours, relative to lung or RPMI controls. These responses coincided with specific transcriptional up-regulation of chemotaxis receptors, signalling markers and metabolic pathways in liver relative to lung or RPMI. Our findings provide strong evidence of organ-specific, active chemotaxis in *Ascaris* infection, with implications for other major human helminthiases, and identify conserved chemosensory pathways as promising therapeutic targets. Disrupting chemosensory-guided navigation could prevent larval migration, offering a novel strategy to combat ascariasis.

ID: 237 / **CP18: 2**
Contributed abstract
Conference Topics: Biochemistry, Cell Biology, Genomics, Molecular Biology, Protozoa
Keywords: Giardia duodenalis, RNA binding proteins, Post-transcriptional Regulation, CRISPR, RNA interactome capture, Encystation

The Early-Diverging Eukaryote Giardia Reveals the Origins of Eukaryotic Post-Transcriptional Regulatory Networks

Balu Balan[1,3], Alex Lam[1,3], Amrita Vijay[1,3], Myo Nuang[1,5], Swapnil Tichkule[1,8], Esther Bandala Sanchez[1,3], Sachin Khurana[1,3], Waruni Abeysekera[1,3], Samantha J. Emery-Corbin[1,7], Jarrod J. Sandow[1], David Zhu[1], Ahmad Wardak[1], Jacob Munro[1,3], Pradip Roy[1,3], Brendan Robert E. Ansell[1,3], Peter Czabotar[1,3], Andrew I. Webb[1,3], Marija Darmacian[1,3], Gordon K Smyth[1,3], Olivia Rissland[6], Staffan G. Svärd[4], Aaron R. Jex[1,2,3]

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RNA-binding proteins (RBPs) regulate splicing, RNA silencing, and translational repression in eukaryotes. Many are conserved from yeast to humans but are absent or rudimentary in prokaryotes, suggesting an early emergence of “eukaryotic-innovative” RBPs. We hypothesised that these RBPs arose long before yeast and are retained in Giardia, an early-diverging eukaryote. To test this, we built a phylogenomic atlas of RBP families across the tree of life and analysed domain topology, domain co-occurrence, and intrinsically disordered regions (IDRs) to define their architectural evolution. We then characterised the Giardia RBPome using domain- and structure-informed annotation integrated with transcriptomics, proteomics, and RNA–protein interactome capture to identify canonical and non-canonical (“moonlighting”) RBPs. Direct RNA targets and regulatory networks of representative eukaryotic-innovative RBPs, including PUF, DDX3X, EIF4A, and PGK, were resolved using enhanced CLIP-seq and RBP immunoprecipitation. Functional significance was assessed by CRISPRi-based genetics, and condensate behaviour was tested using phase-separation assays *in vitro* and *in vivo*. Our analyses show that multiple eukaryotic-innovative RBPs are already present in Giardia, with simplified but functional architectures, conserved RNA–protein networks, regulatory phenotypes, and condensate-like behaviour, establishing Giardia as a minimal model for the earliest evolution of eukaryotic post-transcriptional control.

ID: 170 / **CP18: 3**
Contributed abstract
Conference Topics: Biochemistry, Bioinformatics, Drugs, Protozoa, Proteomics
Keywords: Kinase, Drug discovery, Giardia, Functional genetics, Biophysics, Biochemistry

Chemoproteomics Identifies a Druggable Kinase in the Parasitic Protist *Giardia duodenalis*

Alex Lam[1], Louise Baker[1], Balu Balan[1], Toby Dite[2], Dylan Multari[3], Vineet Vaibhav[1], Mengbo Li[1], Guillaume Lessene[1], Isabelle Lucet[1], Andrew Thompson[1], Aaron Jex[1], Samantha Emery-Corbin[3]
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Giardia duodenalis is a gastrointestinal parasite causing ~200 million symptomatic infections annually, disproportionately in lower socioeconomic tiers and children. Chemotherapeutic interventions are limited to nitroheterocyclic antibiotics such as metronidazole. However, high doses are toxic and drug-resistant treatment failures occur in up to 20% of cases, highlighting the urgency of novel and safer chemotherapeutics. Here, we target the disproportionate kinome of *G. duodenalis* with small-molecule inhibitors to reveal novel antigiardials and their molecular targets for next generation antiparasitics.

Using "Click" chemistry, we immobilised a potent drug-like kinase inhibitor to azide-agarose supports and identified a high-affinity kinase domain-containing protein (GiK5) as the putative target for this inhibitor. We validated recombinant GiK5-inhibitor engagement through differential scanning fluorimetry, native mass spectrometry and the fluorescent ADP-glo assay. Further, we conducted a multiplexed CRISPR-interference knockdown which showed lower GiK5 abundance at the protein level contributed to slower parasite growth, suggesting the likely essentiality of this protein in the parasite.

We reveal this likely druggable kinase in *G. duodenalis* and this workflow incentivises high-throughput, target-centric screening campaigns for structure-guided drug-discovery, as well as repurposing clinically-approved kinase inhibitors for chemotherapeutic interventions against this parasite.

ID: 156 / **CP18: 4**
Contributed abstract
Conference Topics: Bioinformatics, Genomics, Helminthology, Veterinary Parasitology
Keywords: Haemonchus contortus, apoptosis, intrinsic pathway, structural modelling, nematodes, post-transcriptional regulation

Genome-wide reconstruction of the intrinsic apoptosis pathway in *Haemonchus contortus*

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Programmed cell death (apoptosis) is a fundamental process in metazoans, extensively characterised in vertebrates but comparatively understudied in invertebrates beyond the model organisms *Caenorhabditis elegans* and *Drosophila melanogaster*. Here, we present the first reconstruction of the intrinsic apoptosis pathway in the parasitic nematode *Haemonchus contortus*, a blood-feeding pathogen of ruminants responsible for substantial global production losses. Using *C. elegans* proteins as a reference, we integrated genome-wide homology searches with structural modelling and developmental transcriptomic and proteomic analyses to identify apoptosis regulators in *H. contortus*. Homologues of all canonical pathway components were identified, including CEP-1, EGL-1, CED-9, CED-4 and CED-3, together with modulators such as DRE-1 and PUF-8. Structural analyses revealed conservation of key interaction complexes (CED-9:CED-4 and CED-4:CED-3), whereas EGL-1 and CEP-1 retained critical structural domains despite marked sequence divergence. Transcriptomic profiling showed that *Hc-ced-9* and *Hc-ced-3* are constitutively expressed across developmental stages, whereas *Hc-cep-1* and *Hc-egl-1* display stage-specific transcription. Proteomic data confirmed the presence of *Hc*-CED-9, *Hc*-CED-4 and *Hc*-CED-3 in at least one life stage but did not detect *Hc*-EGL-1 or *Hc*-DRE-1. Discordances between transcript and protein abundance, particularly for *Hc*-EGL-1, suggest post-transcriptional regulation. Future efforts are needed to elucidate the apoptosis pathway across members of the phylum Nematoda.

ID: 157 / **CP18: 5**
Contributed abstract
Conference Topics: Bioinformatics, Genomics, Helminthology
Keywords: Intrinsic apoptosis, Nematoda, BCL-2 family proteins, Caspase–adaptor signalling, Evolutionary diversification

Elucidating the intrinsic apoptosis pathway in nematodes: fundamental and applied implications

Marc Kaethner[1], Neil D. Young[1], Erinna F. Lee[2,3], W. Douglas Fairlie[2,3,4,5], Brad E. Sleebs[1,6], Bill C. H. Chang[1], Robin B. Gasser[1]
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Intrinsic apoptosis is a form of programmed cell death that governs development, tissue homeostasis and stress responses in animals. In nematodes, the pathway was first genetically defined in the free-living nematode *Caenorhabditis elegans*, yet how it has diversified and operates across the phylum, which encompasses parasites of humans and animals spanning clades I–V, remains poorly resolved. Here, we synthesise genomic, structural and functional data to establish a framework for intrinsic

apoptosis in nematodes. Although the core CED-9-CED-4-CED-3 module is retained, its developmental deployment and regulatory processes remain largely uncharacterised beyond *C. elegans*. Unlike the corresponding BCL-2-regulated apoptotic pathway in vertebrates, *C. elegans* lacks a canonical BAX/BAK-driven mitochondrial permeabilisation system, and whether comparable mitochondrial amplification mechanisms operate in other nematodes remains unclear, particularly in species encoding multiple CED-9/BCL-2-like proteins. This alternative regulatory configuration, coupled with structural divergence of nematode CED-9 and CED-4-like proteins from their vertebrate orthologues, suggests a distinctive evolutionary trajectory for apoptotic regulation in nematodes. By integrating biological with emerging structural insights, we define a foundation for studying apoptosis across clades I–V and assess its potential for anthelmintic target discovery.

ID: 155 / **CP18: 6**
Contributed abstract
Conference Topics: Bioinformatics, Genomics, Helminthology, Host-parasite interactions, Immunology, Molecular Biology
Keywords: schistosomiasis, single-cell spatial transcriptomics, granuloma, fibrosis

Mapping fibrotic microenvironments: Single-cell resolution spatial profiling of *Schistosoma mansoni*-induced tissue fibrosis

Veronika Toth[1], Prakrithi Pavithra[1], Zherui Xiong[1], Chaoyi Li[1], Malcolm Jones[1,2], Quan Nguyen[1,2], Hong You[1,2]
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Schistosomiasis-induced fibrosis, driven by host immune response to trapped eggs, is the principal cause of pathology and schistosomiasis-related morbidity. While praziquantel effectively clears adult parasites, no therapies exist to target egg or egg-induced tissue fibrosis. To address gaps in fibrosis-related cellular mechanisms and identify novel antifibrotic targets, we used single-cell spatial transcriptomics with a 5000-gene mouse panel to map the molecular architecture of hepatic and intestinal fibrotic regions in *Schistosoma mansoni*-infected mice. Results showed that schistosome-induced granulomas are highly organized and overlap with fibrotic regions in both tissues at 8 weeks post-infection. Differential expression analysis identified 1175 genes in 534k liver cells and 807 genes in 259k intestine cells significantly altered between healthy control and infected samples. These DEGs are strongly associated with cytokine and interleukin signaling, TGF-β pathway, and extracellular matrix organization. The cell types enriched in infected regions included macrophages, collagen-producing cells (liver-activated hepatic stellate cells, intestine-activated fibroblasts), eosinophil-like cells, T, B, and plasma cells. Furthermore, cell-cell interaction analysis revealed strong interactions between macrophages and collagen-producing cells in infected tissues. While many identified ligand-receptor pairs are organ-specific, a core signature of 5 pairs (including *Col1a2/Tgm2-Itgb1*) was shared across tissues, representing potential pan-tissue therapeutic targets for schistosome-induced fibrosis.

ID: 225 / **CP18: 7**
Contributed abstract
Conference Topics: Biochemistry, Bioinformatics, Cell Biology, Genomics, Microscopy, Molecular Biology, Protozoa, Proteomics
Keywords: Splicing, Evolution, Giardia, Multiomics, Bioinformatics

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

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Alternative splicing is a major mediator of eukaryotic gene expression, operating co-transcriptionally and post-transcriptionally to influence cellular function, development, and differentiation. During alternative splicing, *trans*-acting factors interact with *cis*-elements within pre-mRNA transcripts to produce divergent proteoforms. In eukaryotes, most*trans*-acting factors work in conjunction as the spliceosome, a dynamic ribonucleoprotein complex comprised of catalytic RNAs and hundreds of proteins. Previous studies sought to comprehend the intricate mechanisms of the spliceosome within a simplistic system, utilising the model organism *Saccharomyces cerevisiae*. However, there exists a eukaryote more basal than yeast – *Giardia duodenalis,* an intron-poor enteric parasite, evolved over 500 million years earlier. Our bioinformatic analyses indicated that splicing proteins in *Giardia* are minimal, both in number and structure. This raises the question: In deeply-branching eukaryotes such as *Giardia,* does splicing occur spliceosomally or via a more primordial method? To understand the system of splicing in *Giardia*, we conducted an *in vitro* splicing assay involving both conventional and chimeric intron-containing transcripts. In parallel, we established a nuclear proteome to locate and validate the suite of *Giardia* splicing proteins. By implementing a multiomic approach, our study offers a glimpse into the origins and evolution of alternative splicing following the inception of eukaryotic life.

CP20.1: Zoonoses & One Health 1 - 10 min talks

Time: Thursday, 02/July/2026: 2:00pm - 2:30pm · *Location:* Lecture Theatre 3
Session Chair: Darren Gray, QIMR Berghofer
Session Chair: Jessica Scott, James Cook University

ID: 191 / **CP20.1: 1**
Contributed abstract
Conference Topics: Biodiversity, Ectoparasites
Keywords: Mosquito biodiversity, Landscape, Climate

Exploring spatiotemporal shifts on mosquito diversity in Perth, Western Australia.

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Mosquitoes are the most significant vectors of human and animal diseases. Worldwide, there are over 3,500 extant species, each with varying ecological behaviours. Consequently, mosquito populations are dynamic, shifting over time and space. As a result, the risk of disease transmission is also dynamic, as it relies on the prevalence and abundance of known competent vectors. Therefore, understanding how mosquito populations shift is essential to accurately assess mosquito-borne disease transmission risk.

To investigate spatiotemporal effects on mosquito populations, 4,469 mosquitoes (representing 15 species) were collected from 10 locations within the Perth metropolitan area between 2022 and 2024. Climate variables (rainfall and temperature) and landscape coverage (within 2.5 km) were obtained for each location and trapping event. Generalised linear models determined the influence of spatiotemporal variables on mosquito population diversity indices (Hill's numbers) and species abundance.

Mosquito population diversity was not significantly influenced by landscape coverage; instead, diversity was driven by climate variables. Mosquito species exhibited diverse temperature and rainfall preferences, indicating that competent vectors for endemic mosquito-borne diseases are prevalent year-round in Perth. Our findings underscore the importance of integrating climate-driven and land-use data into mosquito surveillance and control programs to improve predictions of mosquito-borne disease transmission risk.

ID: 230 / **CP20.1: 2**
Contributed abstract
Conference Topics: Ectoparasites, Epidemiology, One Health, Veterinary Parasitology, Wildlife parasitology, Zoonoses
Keywords: Cuora flavomarginata, Tick-borne pathogens, Francisella spp., One Health, Wildlife parasitology

Uncovering One Health Risks: High Prevalence of *Francisella* spp. in Tick-Borne Pathogens of *Cuora flavomarginata* in Taiwan

Yi-Lun Tsai[1,2], Alinda Lok-Yan Yong[1], Chi-Hsin Tsou[1], Tien-His Chen[3]
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Cuora flavomarginata, an endemic terrestrial turtle in Asia and a Class I protected species in Taiwan, remains understudied in terms of parasite diversity and zoonotic risk. This study investigated endo- and ectoparasites, with a One Health focus on tick-borne pathogens. Samples were collected from 12 turtles in Shizi Township, yielding 154 ticks and 6 fecal samples. All ticks were identified as *Amblyomma geoemydae* using morphological and molecular approaches. A total of 51 tick DNA samples were screened by PCR for *Francisella* spp., *Rickettsia* spp., *Coxiella burnetii*, and *Ehrlichia* spp., while fecal samples were examined for endoparasites. *Francisella* spp. showed a notably high detection rate (86.3%), followed by *Rickettsia* spp. (27.5%) and *C. burnetii* (2.0%); *Ehrlichia* spp. were not detected, and no endoparasites were found. The high prevalence of *Francisella* spp. suggests that reptile-associated ticks may contribute to its environmental maintenance. Given the unclear transmission pathways of tularemia in Taiwan, these findings highlight a previously underrecognized component in its ecology. This study underscores the importance of integrating wildlife, vector, and pathogen surveillance to better understand zoonotic disease risks within a One Health framework.

ID: 122 / **CP20.1: 3**
Contributed abstract
Conference Topics: Ectoparasites, Epidemiology, Host-parasite interactions, One Health, Wildlife parasitology, Zoonoses
Keywords: Bats; ticks; tick-borne pathogens; Australasia; conservation; One Health

Bat-Associated Ticks and Tick-Borne Pathogens in Australasia: Implications for Conservation and One Health

Mehran Khan, Abdul Ghafar, Bahar E Mustafa, Ian Beveridge, Abdul Jabbar
The University of Melbourne, Australia

Bats are key ecological species and recognised reservoirs of diverse pathogens, yet their role in tick–host–pathogen systems in Australasia remains poorly understood. This study systematically synthesises current knowledge on bat-associated ticks and tick-borne pathogens (TBPs) and identifies critical gaps. Following PRISMA guidelines, literature was retrieved from major databases, including studies reporting ticks on bats or within their environments, and those investigating TBPs. Risk of bias was assessed using an AXIS-based framework. Of 512 studies screened, 35 met the inclusion criteria. Fourteen typical and nine incidental tick species were reported from bats, predominantly in Australia and New Guinea. Only one bacterial pathogen, *Rickettsia japonica*, was detected in the typical bat-associated tick *Argas dewae*. The non-typical tick *Ixodes holocyclus* was associated with paralysis in bats, while *A. dewae* may infest other hosts, including humans. Most studies relied on opportunistic sampling and morphological identification, with minimal use of molecular tools and limited investigation of

pathogen diversity or bat health impacts. These findings highlight major gaps in surveillance, molecular characterisation, and pathogen detection, underscoring the need for integrated One Health approaches to better understand bat–tick–pathogen dynamics in Australasia.

CP19.1: Immunology 2 - 10 min talks

Time: Thursday, 02/July/2026: 2:15pm - 2:45pm · *Location:* Lecture Theatre 2
Session Chair: Alicja (Ala) Tabor, The University Of Queensland
Session Chair: Hannah Siddle, The University of Queensland

ID: 109 / **CP19.1: 1**
Contributed abstract
Conference Topics: Immunology, Malaria
Keywords: Fc-afucosylation, VAR2CSA, ADCC, ADNP

Afucosylated VAR2CSA-specific IgG reduce risks of placental malaria

HongHua Ding[1], Oscar H. Lloyd Williams[1], Maria Saeed[1], Wina Hasang[1], Bruce D. Wines[2], Mary Lopez-Perez[3], Maria del Pilar Quintana[3], Lars Hviid[3], Adam Wheatley[4], Mwayiwawo Madanitsa[5,6], Victor Mwapasa[7], Kamija S. Phir[7,8], Feiko O. ter Kuile[6], Nichollas Scott[4], Elizabeth H. Aitken[1,4], Stephen J. Rogerson[1,9]

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Antibody Fc regions have important roles in clearance of Plasmodium falciparum infected erythrocytes. One such role is engagement with Fcg receptors on host leukocytes. In the case of FcgRIIIa and b, this interaction is greatly enhanced when the IgG glycan is afucosylated. In this study, we used a fucose-sensitive enzyme-linked immunosorbent assay, FEASI, to assess afucosylation in IgG specific for placental malaria protein VAR2CSA from n=139 malaria-exposed pregnant Malawian women, correlating these data with mass spectrometry-based analysis. Furthermore, we measured the effect of afucosylation on Fc-mediated leukocyte functions, using both plasma and a monoclonal antibody, PAM2.8, with varying levels of afucosylation.

Results showed significantly higher levels of VAR2CSA-specific IgG afucosylation in women with no placental malaria measured by FEASI ($p < 0.0001$), which correlated strongly with mass spectrometry analysis ($R = 0.8$, $p < 0.0001$). In addition, highly afucosylated IgG mediated significantly greater neutrophil phagocytosis of antigen coated beads and induction of NK cell degranulation by IEs.

Afucosylated IgG to VAR2CSA, measured by FEASI or mass spectrometry, was a correlate of protection from placental malaria, and afucosylated IgG activated NK cells and neutrophils. Naturally acquired or therapeutic afucosylated IgG antibody could have a role in protection from malaria infection.

ID: 221 / **CP19.1: 2**
Contributed abstract
Conference Topics: Biochemistry, Cell Biology, Drugs, Host-parasite interactions, Immunology
Keywords: Therapeutic, Inflammatory disease, Hookworm, Recombinant proteins

Necator americanus recombinant protease inhibitors as novel therapeutics for inflammatory disease

Connor McHugh, Suchandan Sikder, Kim Miles, Sophie Gisder, Yoshimi Peck, Maggie Veitch, Maxine Smith, Stephanie Ryan, Darren Pickering, Roland Ruscher, Paul Giacomin, Alex Loukas
Australian Institute of Tropical Health and Medicine, JCU

Helminth infections, whether experimental or naturally acquired, are increasingly recognised as potent modulators of human immunity, with protective effects across a range of inflammatory diseases. Much of this activity is driven by excretory/secretory proteins (ESPs), complex mixtures of bioactive molecules that act at the host–parasite interface to reshape immune responses and suppress inflammation. Despite this, the therapeutic use of live helminths remains limited due to safety concerns, complex life cycles, and variable host responses. Consequently, focus has shifted toward isolating individual ESPs as more tractable, drug-like candidates. To address this, we generated a recombinant library spanning the secretomes of both larval and adult stages of the human hookworm *Necator americanus*. This enabled systematic screening across *in vitro* and *in vivo* assays to identify proteins with immunoregulatory activity. To date, two distinct protease inhibitors have emerged, each displaying pronounced anti-inflammatory effects. Their independent identification via separate screening strategies underscores the library's versatility as a discovery platform. Their protease inhibitory activity has been confirmed *in vitro*, consistent with established roles for helminth-derived inhibitors in modulating host inflammatory pathways. Work is now focused on defining their mechanisms of action in vivo and assessing their potential as pre-clinical therapeutic candidates.

ID: 166 / **CP19.1: 3**
Contributed abstract
Conference Topics: Immunology, Malaria, Vaccines
Keywords: vaccine, boosting, antibody, malaria, children

Repeated malaria vaccine booster doses in children shapes protective antibody responses

Alexander Harris[1,2], Liriye Kurtovic[1,2,3], Adam Thomas[1,2], Bruce Wines[1,3], Alassane Dicko[4], Jean-Bosco Ouedrogo[4], Daniel Chandramohan[4], Brian Greenwood[4], NCT04319380 Clinical trial team[4], James Beeson[1,2,3,5]
[1]Burnet Institute, Melbourne, Australia; [2]School of Translational Medicine, Monash University, Melbourne, Australia; [3]Department of Infectious Diseases, University of Melbourne, Melbourne, Australia; [4]RTS,S SMC clinical trial NCT04319380 team; [5]Department of Microbiology, Monash University, Melbourne, Australia

Effective malaria interventions are essential to reduce disease burden in children. Combining RTS,S vaccination with seasonal antimalarial 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. Protective antibodies were shown to peak following primary vaccination but became progressively lower with each annual booster. It is unknown what drives poor antibody responses to boosters, which likely vary between different antibody isotypes and antigenic targets, and if host factors such as viral co-infections and micronutrient deficiencies are implicated in this suboptimal response.

To address these knowledge gaps, we evaluated plasma samples from young children (n=1,929) in West Africa who received RTS,S and subsequent boosters with or without seasonal chemoprevention over four years as part of a phase-III clinical trial. We found that repeated vaccine doses had differential effects on antibody responses, which varied by the antigenic region of the vaccine. We also investigated the impact of host factors on these responses. By improving our understanding of the immune response to repeated booster doses, this work informs optimal RTS,S implementation strategies to improve vaccine efficacy and longevity.

CP20.2: Zoonoses & One Health 5 min talks

Time: Thursday, 02/July/2026: 2:30pm - 2:45pm · *Location:* Lecture Theatre 3
Session Chair: Darren Gray, QIMR Berghofer
Session Chair: Jessica Scott, James Cook University

ID: 196 / **CP20.2: 1**
Contributed abstract
Conference Topics: Epidemiology, Genomics, One Health, Parasites of dogs
Keywords: Hookworm infections, Necator americanus, Ancylostoma, Papua New Guinea

Ancylostoma ceylanicum Hookworm in Papua New Guinea, 2020

Jessica Scott[1], Daniel Pelowa[2], Wanye Melrose[1], Jeffrey Warner[1], Catherine Rush[1]
[1]James Cook University, Townsville, Australia; [2]Balimo District Hospital, Balimo, Western Province, Papua New Guinea

Hookworm infection remains a significant public health burden in Papua New Guinea (PNG), and whether *Ancylostoma ceylanicum* occurs within local populations in PNG remains unknown. This cross-sectional study, conducted in Balimo, Western Province, in January 2020, investigated hookworm prevalence and species distribution among 122 community members using microscopy and quantitative PCR (qPCR). Sanger sequencing of the internal transcribed spacer region was used to determine the *Ancylostoma* species present in qPCR-positive samples. Overall, hookworm prevalence was 54.9%, with qPCR demonstrating greater sensitivity than microscopy (52.5% vs. 22.9%). *Necator americanus* was the dominant species, detected in 52.5% of participants. Four cases (3.3%) of the zoonotic species *A. ceylanicum* were identified, all as co-infections with *N. americanus*. No *A. duodenale* was detected. The findings provide the first molecular evidence of local *A. ceylanicum* infections in a rural PNG community. Given the overall high burden of hookworm and zoonotic potential of *A. ceylanicum,* sustainable control in this setting may require a One Health approach addressing both human and animal reservoirs.

ID: 262 / **CP20.2: 2**
Contributed abstract
Conference Topics: Diagnostics, Helminthology, Molecular Biology, One Health, Zoonoses
Keywords: canine parasites, hookworm, Tonga, One Health, molecular diagnostics, Ancylostoma ceylanicum, Ancylostoma caninum

Molecular Detection and Species Identification of Hookworm in Free-Roaming Dogs in Tonga, with Identification of Zoonotic Ancylostoma ceylanicum

Kate Harder[1], K Naden[2], D Maneniaru[3], H Zhao[3], C Joone[3], C Constantinoiu[3], R Bradbury[3]
[1]Environmental and Animal Sciences, Unitec New Zealand, Auckland New Zealand; [2]Otago Polytechnic, New Zealand; [3]James Cook University, Townsville, Australia

Dogs can act as reservoirs for parasites of veterinary and zoonotic importance, yet data from Pacific Island nations remain limited. This study presents preliminary molecular screening results from 179 canine faecal samples collected across five villages in Tongatapu, Tonga, and one village on Vava'u, Tonga to investigate gastrointestinal parasite occurrence and spatial variation. Faecal samples were analysed using molecular methods targeting common canine parasites, with microscopy comparisons to these findings underway. Overall, Ancylostoma caninum was the most frequently detected parasite, identified in approximately 80% of samples, followed by Giardia (~35%), Ancylostoma ceylanicum (~25%), and Strongyloides spp. (~20%). Parasite prevalence varied among villages. A. caninum prevalence exceeded 70% in several locations and was highest in Vava'u and Kolovai. Notably, A. ceylanicum, a zoonotic hookworm of public health concern, reached approximately 60% prevalence in Fua'amotu. Giardia detection was also variable, reaching approximately 50% in Tokomololo and over 50% in Kolonga. Patterns suggest substantial spatial heterogeneity in parasite exposure and transmission risk. Due to small sample

size in villages, this must be noted as a limitation and considered for future studies. These preliminary findings indicate a high burden of gastrointestinal parasitism in dogs in Tonga, including zoonotic species relevant to One Health.

ID: 115 / **CP20.2: 3**
Contributed abstract
Conference Topics: Protozoa, Veterinary Parasitology, Zoonoses
Keywords: anaemia, haemotrophic, zoonosis

The parasites that jumped ship

Johann Schröder, Joan B Lloyd, Una M Ryan, Caroline L Jacobson
Murdoch University, Australia

They can be seen in a Giemsa-stained blood smear. For more than a century, they've been written about and then written off because, mostly, they seemed harmless. They have been found in a variety of livestock and other animals. They behave like blood parasites and even use arthropods as vectors.

They are not as harmless as they seem. Their principal pathogenic effect is haemolytic anaemia. Outbreaks have been described in sheep flocks, with infections severe enough to cause clinically evident ill-thrift, haemolytic anaemia, and mortality. A disquieting discovery has been the zoonotic risk they pose to people in close contact with infected animals – livestock, companion animals, synanthropic rodents.

They cannot be grown in culture. They were classified in two protozoal genera. Transmission electron microscopy raised doubts about their *bona fides*. Nucleic acid extraction and amplification lifted the veil, revealing their true identity and starting the analysis of their phylogenetic evolution

CP18.1: Cells, Molecules & Genes 3 - 5 min talks

Time: Thursday, 02/July/2026: 2:40pm - 2:45pm · *Location:* Lecture Theatre 1
Session Chair: Shilpa Kapoor, The University of Melbourne
Session Chair: Balu Balan, Walter and Eliza Hall Institute

ID: 254 / **CP18.1: 1**
Contributed abstract
Conference Topics: Bioinformatics, Cell Biology, Host-parasite interactions, Malaria, Microscopy
Keywords: malaria, Plasmodium falciparum, MSP, AMA1, immune evasion

Investigating the function of and impact of polymorphism in the malaria vaccine candidate *Plasmodium falciparum* merozoite surface protein 2.

Kaitlin Turland[1], Dimithu Angage[2], Isabelle Henshall[3], Robin Anders[2], Michael Foley[2], Melanie Ridgway[1], Danny Wilson[1]
[1]Adelaide University, Adelaide, Australia; [2]LaTrobe University, Melbourne, Australia; [3]Bernard-Nocht Institute of Tropical Medicine, Hamburg, Germany

The significant global burden of malaria, caused by *Plasmodium* spp., warrants novel treatment and prevention strategies. *Plasmodium falciparum* merozoite surface protein 2 (PfMSP2) has been a target of blood stage malaria vaccines, which have reached phase I/2b clinical trials. Strain-specific polymorphisms complicate the development of this antigen as a vaccine candidate; most PfMSP2 genes can be classified into two main alleles, FC27 and 3D7. Bioinformatic comparison indicates that MSP2 of Laverania species maintain conserved and intrinsically disordered property regions, and tend to have repeat structures that most closely resemble *P. falciparum* 3D7-like alleles. We successfully knocked out PfMSP2 in both FC27- and 3D7-like parasites, which did not influence blood stage growth but did increase potency of antibodies targeting another vaccine candidate, PfAMA1. The *ama1* genotypes of parasites with 3D7- and FC27-like *msp2* do not significantly differ, indicating the dimorphism of PfMSP2 is not significantly influenced by PfAMA1 diversity. Ongoing work is applying microscopy and gene editing techniques to understand the role of PfMSP2 in potentiating antibodies to AMA1. This work will define the impact of *msp2* diversity on antibodies targeting other antigens and will help inform merozoite vaccine development.

CP23: Zoonoses & One Health 2 - 15 min talk

Time: Thursday, 02/July/2026: 3:30pm - 3:45pm · *Location:* Lecture Theatre 3
Session Chair: Catherine Gordon, QIMR Berghofer
Session Chair: Fasil Shiferaw, QIMR Berghofer

ID: 222 / **CP23: 1**
Contributed abstract
Conference Topics: Epidemiology, Protozoa, Zoonoses
Keywords: Cryptosporidium, Giardia, molecular epidemiology, public health surveillance, zoonosis, outbreak detection

Large-scale molecular epidemiological survey of *Giardia* and *Cryptosporidium* in Victoria, Australia (2020–2024) reveals novel subtypes and outbreak-associated lineage

Marielle Babineau[1,2], Anson Koehler[2], Michelle Sait[3], Karolina Mercoulia[3], Sally Dougall[4], Jane McAllister[4], Evelyn Wong[4], Norelle Sherry[1,3], Robin Gasser[2], Ben Howden[1,3,5]

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Cryptosporidium and *Giardia* are major causes of gastrointestinal illness globally. In Australia, cryptosporidiosis is notifiable, yet molecular characterisation is not routinely performed, limiting detection of outbreaks and zoonotic transmission. In 2024, Australia recorded a 273% increase in cryptosporidiosis notifications, the third-highest rise globally. Here, we conducted an epidemiological investigation of *Cryptosporidium* and *Giardia* in faecal samples from patients with gastroenteritis in Victoria between 2020 and 2024. Samples underwent *SSU* and *gp*60 sequencing for *Cryptosporidium* and *tpi* sequencing for *Giardia*, and parasite load was estimated. Of 2,330 samples, 225 were *Cryptosporidium*-positive and nine *Giardia*-positive. One *Giardia* isolate was sub-assemblage AI, two AII, and six assemblage B. Seven species and 24 subtypes were identified, including eight novel subtypes. *Cryptosporidium hominis* predominated (85%), followed by *C. parvum* and *C. meleagridis*. Six *C. hominis* subtypes were detected, three linked to 11 recreational water-associated outbreaks in 2024; IaA12R3 and IeA11G3T3 were most frequent, with concordance analysis suggesting 52 additional cases. Multiple *C. parvum* subtypes were identified, including two linked to childcare and camp outbreaks, and novel human-infective subtypes of *C. occultus*, *C. fayeri*, and *C. meleagridis* were detected. These findings highlight *Cryptosporidium* diversity in Victoria and the value of molecular surveillance for public health.

CP21: Cells, Molecules & Genes 4 - 15 min talks

Time: Thursday, 02/July/2026: 3:30pm - 4:00pm · *Location:* Lecture Theatre 1
Session Chair: Andrew Walker, The University of Queensland
Session Chair: Natasha Sharma, The University of Melbourne

ID: 202 / **CP21: 1**
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 Vijay[1], Alexander Gofton[2], Quentin Gouil[3], Shilpa Kapoor[4], Swapnil Tichkule[1,5], Balu Balan[1], Louise Baker[1], Stefano Gaiarsa[6], Clare A Anstead[4], Ala Tabor[7], Nathan Lo[8], Stephen Barker[9], Jan Riemer[10], Fabrizia Stavru[11], Davide Sassera[12,13], Peter Czabotar[1], Tony Papenfuss[1], Aaron Jex[1,4]

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Ixodes holocyclus (the Australian eastern paralysis tick) is a medically and veterinary important ectoparasite that produces potent neurotoxins, holocyclotoxins, causing rapidly ascending flaccid paralysis in companion animals, livestock and humans, often fatally. Despite its importance, the molecular basis of toxin production, host specificity and survival remains poorly understood because genomic and transcriptomic resources are limited. We generated the first chromosomal-scale genome for *I. holocyclus* using Oxford Nanopore long reads, Illumina short reads and Hi-C, and annotated genes with a hybrid *de novo* transcriptome, resolving alternative splicing with long- and short-read alignments. We scanned UTRs and upstream regions of complete genes for conserved regulatory motifs, including putative promoters. Comparative genomics, including synteny and phylogenomic placement, was performed, and ticks from 32 eastern Australian sites were sequenced to examine genomic diversity and its links to ecological adaptation and vector capacity. The 1.9 Gb assembly contains 13 chromosome-level scaffolds, 66% repetitive elements and 93.3% BUSCO completeness. Annotation identified a high-confidence gene set including protein-coding genes. Synteny with *I. scapularis* and *I. ricinus* revealed conserved supergene blocks. Together, these resources advance tick biology and support targeted control strategies against tick-borne diseases.

ID: 207 / **CP21: 2**
Contributed abstract
Conference Topics: Ectoparasites, Genomics
Keywords: Ixodes ricinus, salivary glands, ovarian tissue, blood-feeding, ectoparasites

Long-read–supported gene modelling illuminates feeding, immune, and developmental biology in the European castor bean tick, *Ixodes ricinus*.

Amrita Vijay[1], Balu Balan[1], Louise Baker[1], Stefano Gaiarsa[2], Quentin Gouil[3], Pradip Roy[1], Alexander Gofton[4], Alessandra Cafiso[5], Ala Tabor[6], Nathan Lo[7], Olivier Plantard[8], Jan Riemer[9], Fabrizia Stavru[10], Clare A Anstead[11], Peter Czabotar[1], Tony Papenfuss[1], Davide Sassera[12,13], Aaron Jex[1,11]

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The European castor bean tick, *Ixodes ricinus*, is a major blood-feeding ectoparasite and vector of Lyme disease, tick-borne encephalitis and babesiosis. Reducing tick-borne disease requires an improved understanding of tick molecular biology across tissues and feeding stages. We applied long-read, full-length mRNA sequencing to refine gene models in the published *I. ricinus* genome and to profile salivary glands and ovaries across blood-feeding phases. Curated functional annotation was used to interpret pathways central to ectoparasitism. We defined 30,454 gene models, including 28,515 protein-coding genes. Functional curation provided insight into chemosensation, hematophagy, immune tolerance, reproduction and fecundity. Salivary glands showed feeding-associated metabolic rewiring, vesicle biogenesis, and secretome remodelling, consistent with anti-clotting, vasodilatory, anti-inflammatory, and immunomodulatory functions that counter host defences. Ovarian transcriptomes revealed early reprogramming of cell-fate pathways, cytoskeletal organisation, extracellular matrix dynamics, and immune regulation, consistent with priming for fertilisation and embryogenesis. This transcriptomic blueprint provides a high-resolution annotation resource for the *I. ricinus* genome and delineates tissue- and stage-specific programmes that underpin feeding and reproduction, providing a platform for mechanistic studies and improved tick control strategies.

CP22: Horses & Cows 2 - 10 min talks

Time: Thursday, 02/July/2026: 3:30pm - 4:20pm · *Location:* Lecture Theatre 2
Session Chair: Alicja (Ala) Tabor, The University Of Queensland
Session Chair: Sara Taylor, QIMR Berghofer

ID: 213 / **CP22: 1**
Contributed abstract
Conference Topics: Ectoparasites, Host-parasite interactions, Livestock Parasites, Veterinary Parasitology
Keywords: Haemaphysalis longicornis, artificial membrane feeding, tick biology, vector competence, Theileria orientalis

A host-free in vitro membrane feeding system for an emerging tick vector, *Haemaphysalis longicornis*

Abdul Ghafar[1], Bahar E Mustafa[1], Charles G Gauci[1], Ian Beveridge[1], Robin B Gasser[1], Ard M Nijhof[2,3], Abdul Jabbar[1]

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Haemaphysalis longicornis is a parthenogenetic three-host tick of increasing veterinary and public health importance and is the principal vector of *Theileria orientalis* in Australasia. Experimental research on this species has been constrained by reliance on live vertebrate hosts, limiting scalability, standardisation and ethical feasibility. This study established a reproducible, host-free in vitro membrane feeding system for adult and nymphal stages. Ticks were fed using a silicone membrane-based system under controlled laboratory conditions. Field-collected adult females were evaluated across six independent experiments for attachment, engorgement, oviposition, egg hatchability and bloodmeal-to-egg conversion efficiency. Nymphal feeding performance was assessed across five experiments. Adult feeding was robust, with 67% (35/52) attachment and 74.3% (26/35) engorgement. Engorged females reached a mean weight of 161 mg and produced a mean egg mass of 67 mg (40% conversion efficiency), with >92% hatchability. Nymphs showed consistently high performance, with 88.4% (76/86) attachment, 97.4% (74/76) engorgement and 90.5% (67/74) moulting success. Engorgement typically occurred within 3–5 days post-attachment, and moulting within 2–4 weeks post-detachment. This system enables controlled, ethical and scalable experimentation and provides a powerful platform for studies of tick physiology, vector competence, acaricide screening and pathogen–vector interactions.

ID: 200 / CP22: 2
Contributed abstract
Conference Topics: Livestock Parasites, Veterinary Parasitology
Keywords: Cattle tick, Rhipicephalus australis, artificial tick feeding, in vitro feeding, artificial membrane feeding

First Report of Consecutive Artificial Membrane Feeding of All Life Stages of *Rhipicephalus australis*

Bahar E Mustafa[1], Abdul Ghafar[1], Charles G. Gauci[1], Swaid Abdullah[2], Ian Beveridge[1], Ard M. Nijhof[3,4], Abdul Jabbar[1]
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Ticks are obligate haematophagous ectoparasites, and species within the *Rhipicephalus microplus* complex are responsible for substantial economic losses to the cattle industry. Traditionally, laboratory rearing of ticks has relied on live animal hosts; however, increasing animal welfare concerns have driven the development of artificial tick feeding systems (ATFS). Here, we report the first successful consecutive artificial feeding of all life stages of the Australian cattle tick, *Rhipicephalus australis*, using a silicone membrane-based system. Success was achieved through optimisation of membrane thickness, incorporation of olfactory stimuli and strict contamination control, enabling continuous *in vitro* feeding of this one-host tick species. Using larvae aged 2-13.5 weeks, all developmental stages demonstrated good attachment and engorgement rates. This study represents the first demonstration of consecutive feeding of all life stages of *R. australis* without the use of live animal hosts. The developed ATFS provides a good platform for further investigating tick biology and tick–pathogen interactions under controlled conditions, as well as for evaluating acaricides/vaccine candidates. However, the limitations of this system include reduced oviposition and increased mortality indicating the need for further optimisation. Overall, this system supports the principles of the 3Rs (Replacement, Reduction and Refinement) in tick and tick-borne disease research.

ID: 193 / CP22: 3
Contributed abstract
Conference Topics: Livestock Parasites, Veterinary Parasitology
Keywords: Cattle tick, Rhipicephalus australis, microbiome, amplicon sequencing, 16S rRNA gene

Decoding the Microbiome of the Australian Cattle Tick (*Rhipicephalus australis*): Stage-Specific Diversity and Symbiotic Associations

Bahar E Mustafa[1], Abdul Ghafar[1], Lianet Abuin-Denis[2], Endris A. Ali[1], Mehran Khan[1], Swaid Abullah[3], Ian Beveridge[1], Charles G. Gauci[1], Alejandro Cabezas-Cruz[2], Ard M. Nijhof[4,5], Abdul Jabbar[1]
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Ticks are among the most important vectors of pathogens affecting livestock. *Rhipicephalus australis* (the Australian cattle tick) transmits several economically significant pathogens, including *Anaplasma* and *Babesia* spp. Increasing evidence suggests that the tick microbiome influences pathogen acquisition, persistence and transmission, thereby shaping vectorial capacity. This study characterised bacterial communities across larval, nymphal and adult stages of *R. australis* collected from cattle farms in Queensland, Australia. Following surface decontamination and DNA extraction, 16S rRNA gene sequencing was performed using the Illumina NextSeq 1000 platform, with downstream analyses conducted in QIIME 2 and R. A diverse bacterial community was identified, including *Arsenophonus, Acinetobacter, Brevibacterium, Coxiella, Corynebacterium, Serratia, Stenotrophomonas, Escherichia–Shigella* and *Staphylococcus*. Several taxa were consistently detected across all life stages, suggesting conserved or potentially symbiotic associations while others were stage-specific, indicating dynamic shifts in microbial community composition during tick development. Microbial network analyses further revealed distinct, stage-specific interaction patterns. These findings provide the first comprehensive insights into life stage-associated microbiome variation in *R. australis* and highlight the potential role of microbial communities in tick biology and pathogen transmission. This work establishes a foundation for microbiome-informed strategies to improve Australian cattle tick control and enhance livestock health and productivity.

ID: 195 / CP22: 4
Contributed abstract
Conference Topics: Livestock Parasites, Veterinary Parasitology
Keywords: Cattle tick, Rhipicephalus australis, microbiome, artificial tick feeding, amplicon sequencing

First Characterisation of the Microbiome of Artificial Membrane Fed *Rhipicephalus australis*

Bahar E Mustafa[1], Abdul Ghafar[1], Lianet Abuin-Denis[2], Mehran Khan[1], Swaid Abullah[3], Ian Beveridge[1], Charles G. Gauci[1], Alejandro Cabezas-Cruz[2], Ard M. Nijhof[4,5], Abdul Jabbar[1]
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Ticks are important vectors of pathogens affecting both humans and animals. For example, cattle ticks transmit *Anaplasma* and *Babesia* spp., causing substantial economic losses to the global cattle industry. Artificial tick feeding systems (ATFS) are increasingly used as alternatives to animal-based feeding to study tick biology, pathogen transmission and control strategies. However, their impact on the tick microbiome remains poorly understood. This study investigated the microbiome and microbial co-occurrence networks of the Australian cattle tick (*Rhipicephalus australis*) reared under ATFS conditions to establish baseline insights. Ticks were collected across multiple life stages, from larvae to engorged females, surface decontaminated, and processed for DNA extraction. Microbial profiling was conducted using 16S rRNA gene sequencing on the Illumina NextSeq 1000 platform, with downstream analyses performed in QIIME 2 and R. Artificially fed ticks harboured diverse microbial communities, with variation observed across life stages. Network analyses revealed distinct microbial interaction patterns, suggesting structured community dynamics under *in vitro* conditions. These findings indicate that ATFS

environments, including the blood meal, membrane system, and absence of host-derived immune factors, may influence microbial community composition. Such effects should be considered when interpreting biological processes and vector competence in artificially fed ticks.

ID: 168 / **CP22: 5**
Contributed abstract
Conference Topics: Epidemiology, Livestock Parasites, Molecular Biology, Veterinary Parasitology
Keywords: Theileria orientalis, Ikeda, Cattle, Australia

Not just where the ticks are: Insights into *Theileria orientalis* Ikeda in Australia

Emily Onizawa[1,2], Steve Walkden-Brown[1], Cheryl Jenkins[2]
[1]University of New England, Australia; [2]EMAI, Department of Regional NSW, Australia

Theileria orientalis Ikeda has been a significant cause of disease in the Australian cattle industry for over a decade. To investigate how widespread *T. orientalis* Ikeda is across Australia, particularly in regions in and outside of known ranges of the vectors and in the absence of clinical disease, we conducted testing on herds of homebred adult cattle in New South Wales and Queensland. Molecular testing of 526 blood samples from 49 properties was performed to detect and quantify different *T. orientalis* genotypes present within these herds. Previous studies have shown that in clinical cases of theileriosis, the Ikeda genotype is detected in 88% of samples.

Preliminary findings suggest that *T. orientalis* Ikeda may be more widespread than previously thought, including in areas outside the known ranges of the tick vectors. These detections in herds that have not experienced clinical disease indicate that presence of the organism may be a necessary but not sufficient cause of disease on its own. These results help build a clearer understanding of clinical theileriosis in Australia and highlight the value of ongoing active surveillance. Future work to include additional Australian states is planned.

CP23.1: Zoonoses & One Health 2 - 10 min talks

Time: Thursday, 02/July/2026: 3:45pm - 4:25pm · *Location:* Lecture Theatre 3
Session Chair: Catherine Gordon, QIMR Berghofer
Session Chair: Fasil Shiferaw, QIMR Berghofer

ID: 114 / **CP23.1: 1**
Contributed abstract
Conference Topics: Epidemiology, Protozoa, Zoonoses
Keywords: Cryptosporidium parvum, 18S rRNA, gp60, zoonotic, MLST

Multi locus sequence typing (MLST) supports zoonotic transmission of *Cryptosporidium parvum* in Western Australia

Dinamithra Gedara Sugandika Samanmali Bulumulla[1], Una Ryan[1], Amanda Ash[1], Amanda D Barbosa[1], Joshua Aleri[2]
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Cryptosporidium is a major enteric zoonotic protozoan parasite with a wide host range, causing gastrointestinal disease in humans and animals. Livestock, particularly calves, serve as an important zoonotic reservoir for *Cryptosporidium parvum*, the main zoonotic species. In the present study, 300 cattle faecal samples (200 calves and 100 adult cattle) from Western Australia (WA) were screened for *Cryptosporidium* by PCR and sequence analysis at the18S rRNA locus. *Cryptosporidium parvum* positives were analysed using multi locus sequence typing (MLST) of five polymorphic loci including the hypervariable *gp*60 gene and compared to previously typed human *C. parvum* samples from WA.

Results revealed that *C. parvum* was the only species detected in both calves and adult cattle with three *C. parvum* gp60 subtype families identified; IIaA18G3R1, IIaA19G4R1 and IIaA20G3R1. MLST analysis demonstrated limited genetic diversity among cattle and human *C. parvum* isolates, with most isolates clustering within a single dominant clade. The intermixing of host-derived isolates supports potential zoonotic transmission of *C. parvum.*

ID: 215 / **CP23.1: 2**
Contributed abstract
Conference Topics: Diagnostics, Epidemiology, Molecular Biology, One Health, Protozoa, Zoonoses
Keywords: Cryptosporidium, Giardia, Dairy goats, Molecular epidemiology, Australia

Zoonotic *Cryptosporidium* and *Giardia* in Australian dairy goats: a national molecular epidemiological study

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[1]The University of Melbourne, Melbourne, Victoria, Australia; [2]Goat Veterinary Consultancies – goatvetoz, Brisbane, Queensland, Australia

Cryptosporidium and *Giardia* are globally important enteric protozoa with broad host ranges and significant One Health implications. However, molecular epidemiological data in Australian goats remain limited. This study investigated the molecular prevalence, species composition and genotype diversity of *Cryptosporidium* and *Giardia* in Australian dairy goats. A cross-sectional survey (2023–2024) was conducted involving 386 goats (4 weeks–12 months old) from 61 dairy herds across six states. DNA samples were analysed using nested PCR and Sanger sequencing for species and assemblage identification. *Cryptosporidium* was detected in 6.5% of samples and *Giardia* in 9.3%. Three *Cryptosporidium* species were identified: *Cryptosporidium xiaoi*, *C. ubiquitum*, and *C. muris*, with the latter two recognised as zoonotic. *Giardia duodenalis* assemblages AI and E were detected, including five novel assemblage E variants. *C. muris* and a zoonotic *Giardia* sub-assemblage AI are reported for the first time in Australian goats, alongside two novel *C. xiaoi* genotypes. Although overall prevalence was low, the detection of zoonotic genotypes highlights the role of young goats as reservoirs for infection. The absence of *C. parvum*, a

major zoonotic species globally, suggests distinct regional transmission dynamics. These findings provide baseline molecular data to inform surveillance, risk assessment and One Health management strategies in Australia.

ID: 176 / **CP23.1: 3**
Contributed abstract
Conference Topics: Epidemiology, Livestock Parasites, Protozoa, Veterinary Parasitology, Zoonoses
Keywords: Cryptosporidium spp., Giardia duodenalis, pigs, molecular epidemiology, zoonotic risk

Uncovering zoonotic protozoa in pigs: molecular epidemiology of *Cryptosporidium* spp. and *Giardia duodenalis* in Victoria, Australia

Jianuo（Gavin} Xu, Anson V. Koehler, Ghazanfar Abbas, Robin B. Gasser, Abdul Jabbar
Melbourne Veterinary School, Faculty of Science, The University of Melbourne, Victoria, Australia

Cryptosporidium spp. and *Giardia duodenalis* are globally significant enteric protozoa affecting livestock health, productivity and public health through their zoonotic potential. In pigs, these infections can impair growth performance and welfare, yet molecular epidemiological data in Australian production systems remain limited. This study aimed to determine the prevalence, distribution and genetic diversity of *Cryptosporidium* spp. and *G. duodenalis* in pigs across Victoria. A cross-sectional survey was conducted using 626 faecal samples collected from pigs of different age groups across 69 commercial farms. Samples were screened using nested PCR, and amplicons were subjected to Sanger sequencing. Phylogenetic analyses were conducted to identify species, assemblages/subtypes and determine their genetic relationships. Preliminary findings have identified *Cryptosporidium scrofarum*, *C. suis* and the zoonotic species *C. ubiquitum*, as well as *G. duodenalis* assemblages A and E. These results indicate the presence of both host-adapted and zoonotic genotypes in Victorian pig populations. This study will generate the first comprehensive molecular epidemiological dataset for these protozoa in Victorian pigs, providing critical insights into infection dynamics and zoonotic risk. The findings will inform evidence-based parasite control, strengthen biosecurity strategies and support sustainable productivity in the Australian pork industry.

ID: 116 / **CP23.1: 4**
Contributed abstract
Conference Topics: Epidemiology, Helminthology, One Health, Strongyloides
Keywords: soil-transmitted helminth, Schistosoma, Strongyloides, epidemiology

Expanded molecular evidence of soil-transmitted helminth and *Schistosoma spp.* infections in Myanmar schoolchildren: a qPCR update

Catherine Gordon, Eindra Aung, Natasha Collinson, Darren Gray
QIMR Berghofer, Australia

Building on our previous report of high prevalence of soil-transmitted helminth (STH) infections among Myanmar schoolchildren (*Aung et al.*, *Infectious Diseases of Poverty*, 2022), we conducted additional molecular screening of archival stool samples from the same cohort in Phyu Township, Bago Region, to investigate additional helminth infections. We also report finding of other helminths by Kato-Katz in the previous study that were not previously published. Stool samples utilised in this study were collected in 2016 and the DNA extracted in 2017 and kept stored at -20°C until further molecular characterisation in this study in 2025. Using quantitative PCR (qPCR), we detected *Schistosoma* DNA in two of 264 samples, *Strongyloides stercoralis* DNA in twelve, and *Ancylostoma ceylanicum* in eleven. Although sequencing of the *Schistosoma*-positive samples was unsuccessful, the molecular evidence aligns with other recent reports suggesting emerging or cryptic transmission of schistosomiasis in Myanmar. The epidemiology of schistosomiasis in the region remains poorly defined, highlighting the need for targeted snail surveys, environmental DNA (eDNA) monitoring, and host sampling to confirm transmission foci. This study demonstrates the added value of molecular diagnostics for complementing traditional parasitological methods and guiding surveillance and control strategies in areas of emerging endemicity.

CP21.1: Cells, Molecules & Genes 4 - 10 min talks

Time: Thursday, 02/July/2026: 4:00pm - 4:20pm · *Location:* Lecture Theatre 1
Session Chair: Andrew Walker, The University of Queensland
Session Chair: Natasha Sharma, The University of Melbourne

ID: 255 / **CP21.1: 1**
Contributed abstract
Conference Topics: Genomics, Helminthology, Parasites of companion animals
Keywords: filarial nematode; taxonomy; comparative genomics

Chromosome-contiguous nuclear genome of the zoonotic filarial parasite *Dirofilaria asiatica* (Spirurida: Onchocercidae)

Neil Young, Vito Colella, Yuanting Zheng, Anson Koehler, Tao Wang, Sunita Sumanam, Ushani Atapattu, Robin Gasser
The University of Melbourne, Australia

Parasitic nematodes of the family Onchocercidae have co-evolved with vertebrate hosts for millions of years. Although morphology has traditionally underpinned species identification, many taxa are cryptic and difficult to distinguish, limiting accurate diagnosis. Clinically important genera include *Dirofilaria*, which causes heartworm disease in dogs and occasional zoonotic infections in humans. However, substantial gaps remain in our understanding of other *Dirofilaria* species and genotypes and their impacts on host health. This study aimed to generate a comprehensive morphological and molecular resource for a newly identified *Dirofilaria* species. Using long-read PacBio and Hi-C sequencing, we assembled and characterised both mitochondrial and nuclear genomes. The nuclear genome comprises four autosomes and one sex-linked scaffold, encoding 9,658 genes. Comparative analyses with related filarial nematodes revealed conserved chromosomal structure alongside lineage-specific rearrangements. We identified 881 predicted excretory/secretory proteins enriched in immune-related pathways such as proteolysis, lysosomal function and antigen presentation. Notably, 26% of these proteins were unique, many associated with host–parasite interactions, immune evasion and metabolic adaptation. This genome fills a key gap in filarial resources and supports advances in epidemiology, host adaptation studies and diagnostic development.

ID: 167 / **CP21.1: 2**
Contributed abstract
Conference Topics: Bioinformatics, Fasciolosis/Liver fluke, Helminthology, Host-parasite interactions, Veterinary Parasitology
Keywords: Fasciola hepatica, intermediate host, histopathology, microCT, spatial transcriptomics

A spatial–molecular framework for studying host–parasite interactions in a freshwater snail

Natasha Sharma[1], Tanapan Sukee[1], Jay Black[2], Jiadong Mao[3], Bonnie Webster[4], Winston Ponder[5], Robin Gasser[1], Anson Koehler[1], Neil Young[1]
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Understanding how biological processes and interactions unfold within intact organisms requires approaches that link anatomical organisation with spatially resolved molecular information. Although spatially integrated analyses have transformed vertebrate biology, comparable frameworks remain limited for many non-model invertebrates despite their ecological and biomedical importance. Freshwater snails represent a particularly informative system, functioning both as key components of aquatic ecosystems and as intermediate hosts for numerous parasitic organisms. Here we establish an integrated spatial–molecular analytical framework for investigating biological processes within a lymnaeid snail using infection with the liver fluke *Fasciola hepatica* as a model host–parasite system. The approach combines whole-organism three-dimensional imaging using X-ray microcomputed tomography with serial histopathology, bulk RNA sequencing and spatial transcriptomic analysis. Integrating these complementary datasets enables parasite distribution to be examined within the anatomical structure of the host while simultaneously linking tissue pathology with spatially resolved gene transcription patterns. Beyond the present host–parasite system, this framework provides a foundation for investigating a broad range of biological processes in freshwater snails, including environmental responses, neurobiology and host–parasite interactions.

CP21.2: Cells, Molecules & Genes 4 - 5 min talks

Time: Thursday, 02/July/2026: 4:20pm - 4:30pm · *Location:* Lecture Theatre 1
Session Chair: Andrew Walker, The University of Queensland
Session Chair: Natasha Sharma, The University of Melbourne

ID: 128 / **CP21.2: 1**
Contributed abstract
Conference Topics: Ecology, Genomics
Keywords: Simulium damnosum, Population structure, Agro-ecological zones, Onchocerciasis, Transmission

Genetic structuring of *Simulium damnosum* across Ghana's agro-ecological zones: implications for *Onchocerca volvulus* transmission dynamics

Millicent Opoku[1,2,3], Neha Sirwani[1,2], Kwadwo K. Frempong[3], Sampson Otoo[3], Sedou Naniogou[4], Philomena Jackson[3], Millicent S. Afatodzie[3], Sellase Pi-Bansa[3], Joseph H. N. Osei[5], Franklin Ayisi[3], Sarah M. Dogbe[3], Abena A. Nyarko[3], Warwick N. Grant[2], Daniel A. Boakye[3,4], Shannon M. Hedtke[1,2]

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Understanding the population structure of *Simulium damnosum*, the blackfly vector of *Onchocerca volvulus*, is fundamental to predicting onchocerciasis transmission risk and optimising intervention strategies. We investigated the genetic structure of *S. damnosum. S. damnosum* specimens were collected from 24 localities across Ghana via human landing catches. Following genomic DNA extraction and short read-sequencing, genome-wide nuclear SNPs were generated and analysed using DAPC and ADMIXTURE to delineate ancestry clusters. Five genetically distinct clusters were identified, broadly corresponding to Ghana's major agro-ecological zones: two savannah clusters (GS/SS-A and GS/SS-B), a Coastal Savannah (CS), and two Semi-Deciduous Forest clusters (SDF-W and SDF-E). The unexpected subdivision of the SDF ecozone into two divergent lineages, with the Bosomase population forming a genetically isolated western unit, suggests that *O.volvulus* transmission dynamics may differ markedly across this zone despite shared ecological classification. Populations at ecozone boundaries showed elevated admixture (gene flow), indicating contact zones where vector populations and parasite strains may intermix. These results indicate that *S. damnosum* populations in Ghana should not be treated as a single panmictic unit for epidemiological modelling or parasitological surveillance. Parasite-vecto compatibility studies stratified by genetic cluster are warranted to fully characterise transmission risk across Ghana's heterogeneous landscape.

ID: 251 / **CP21.2: 2**
Contributed abstract
Conference Topics: Molecular Biology, One Health, Strongyloides, Veterinary Parasitology, Wildlife parasitology, Zoonoses
Keywords: Strongyloides fuelleborni, Non-human primates, Zoonosis, Captive wildlife animals

The molecular identification of *Strongyloides fuelleborni* from non-human primates (Bornean orangutans, chimpanzees and Red-Shanked Douc Langurs) in Khon Kaen Zoo, Thailand.

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Strongyloidiasis, caused by the nematodes *Strongyloides stercoralis* and *S. fuelleborni*, is a neglected tropical disease affecting millions of people in tropical and subtropical areas. Non-human primates (NHPs) are typically natural hosts of *S. fuelleborni* but this parasite may spread to humans by zoonotic transmission. In this study, the presence of *Strongyloides* infections in NHPs in Khon Kaen Zoo, Thailand, was tested for using the agar plate culture technique. Three out of nine species of NHPs harbored *S. fuelleborni* (33.3%). The overall prevalence from three infected species was 45.5% (5 of 11 animals). DNA was amplified from larval stages of *S. fuelleborni* and a portion of the 18S ribosomal RNA gene was sequenced. These sequences were identical to previously published for *S. fuelleborni*. The parasitological and molecular data obtained in this study confirmed the presence of *S. fuelleborni* among captive NHPs (Bornean orangutans, chimpanzees, and red-shanked douc langurs) in Khon Kaen Zoo in Thailand. There is therefore high transmission potential of *S. fuelleborni* from NHP reservoir hosts to humans and the subsequent development of human strongyloidiasis.

2026 Annual Conference of the Australian Society for Parasitology Inc.

29 June – 2 July, 2026 Mantra on View, Surfers Paradise, Gold Coast Australia

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Bento, Inês	232	CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
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Bischof, Rob	110	CP16 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 2)
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Bott, Nathan J.	265	CP6.2 (Tue, 2026/6/30 14:25-14:45; Lecture Theatre 3)
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Bradbury, Richard S	272	CP17.2 (Thu, 2026/7/2 12:05-12:15; Lecture Theatre 3)
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Browne, Daniel J	211	CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2)
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Dowd, Cynthia S.	158	CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1)
Draper, Sarah	211	CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2)
Duncan, Samuel	260	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
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G. Svärd, Staffan	237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
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Gauci, Charles	231, 213, 124, 131, 193, 195, 200	CP16.1 (Thu, 2026/7/2 11:50-12:05; Lecture Theatre 2), CP22 (Thu, 2026/7/2 15:30-16:20; Lecture Theatre 2), CP10 (Wed, 2026/7/1 11:00-11:15; Lecture Theatre 3), CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3), CP22 (Thu, 2026/7/2 15:30-16:20; Lecture Theatre 2), CP22 (Thu, 2026/7/2 15:30-16:20; Lecture Theatre 2), CP22 (Thu, 2026/7/2 15:30-16:20; Lecture Theatre 2)
Geoghegan, Niall	249	CP1.1 (Tue, 2026/6/30 12:05-12:15; Lecture Theatre 1)

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Giacomin, Paul	125, 199, 221	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2), CP9.1 (Wed, 2026/7/1 11:35-12:05; Lecture Theatre 2), CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Giannangelo, Carlo	153, 173, 187	CP1.1 (Tue, 2026/6/30 12:05-12:15; Lecture Theatre 1), CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1), CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1)
Gisder, Sophie	221	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Alexander Gofton	, , , ,	TBS (Tue, 2026/6/30 13:30-13:35; Lecture Theatre 1), CP4 (Tue, 2026/6/30 13:35-13:50; Lecture Theatre 1), CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1), CP4.2 (Tue, 2026/6/30 14:40-14:50; Lecture Theatre 1), CP4Q (Tue, 2026/6/30 14:50-15:00; Lecture Theatre 1)

Gofton, Alexander	209, 202, 207, 120	CP13.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 3), CP21 (Thu, 2026/7/2 15:30-16:00; Lecture Theatre 1), CP21 (Thu, 2026/7/2 15:30-16:00; Lecture Theatre 1), CP14 (Thu, 2026/7/2 9:45-10:30; Plenary Lecture Theatre)
Gonzalez, Ben	248	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Gonzalez-Miguel, Javier	239	CP4 (Tue, 2026/6/30 13:35-13:50; Lecture Theatre 1)
Good, Michael	165, 151	CP9.2 (Wed, 2026/7/1 12:05-12:15; Lecture Theatre 2), CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Goodman, Christopher	277	P3 (Thu, 2026/7/2 9:00-9:45; Plenary Lecture Theatre)
Goodman, Dean	233, 244, 246	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1), CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1), CP4.2 (Tue, 2026/6/30 14:40-14:50; Lecture Theatre 1)
Catherine Gordon	, , ,	SW (Mon, 2026/6/29 9:00-16:00; Lecture Theatre 2), CP23 (Thu, 2026/7/2 15:30-15:45; Lecture Theatre 3), CP23.1 (Thu, 2026/7/2 15:45-16:25; Lecture Theatre 3), CP23Q (Thu, 2026/7/2 16:25-16:35; Lecture Theatre 3)
Gordon, Catherine	276, 102, 139, 116	S3 (Tue, 2026/6/30 11:00-11:20; Lecture Theatre 3), CP3.1 (Tue, 2026/6/30 11:35-11:55; Lecture Theatre 3), CP3.2 (Tue, 2026/6/30 11:55-12:15; Lecture Theatre 3), CP23.1 (Thu, 2026/7/2 15:45-16:25; Lecture Theatre 3)
Gouil, Quentin	202, 207	CP21 (Thu, 2026/7/2 15:30-16:00; Lecture Theatre 1), CP21 (Thu, 2026/7/2 15:30-16:00; Lecture Theatre 1)

Grahn, E	262	CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3)
Grahn, Emily	272	CP17.2 (Thu, 2026/7/2 12:05-12:15; Lecture Theatre 3)
Grant, Warwick N.	121, 128	CP3 (Tue, 2026/6/30 11:20-11:35; Lecture Theatre 3), CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Grassi, Natalie K	259	CP10.2 (Wed, 2026/7/1 11:45-12:15; Lecture Theatre 3)
Darren Gray	, , , , , , , ,	S3 (Tue, 2026/6/30 11:00-11:20; Lecture Theatre 3), CP3 (Tue, 2026/6/30 11:20-11:35; Lecture Theatre 3), CP3.1 (Tue, 2026/6/30 11:35-11:55; Lecture Theatre 3), CP3.2 (Tue, 2026/6/30 11:55-12:15; Lecture Theatre 3), S3Q (Tue, 2026/6/30 12:15-12:30; Lecture Theatre 3), CP20 (Thu, 2026/7/2 13:30-14:00; Lecture Theatre 3), CP20.1 (Thu, 2026/7/2 14:00-14:30; Lecture Theatre 3), CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3), CP20Q (Thu, 2026/7/2 14:45-15:00; Lecture Theatre 3)
Gray, Darren	102, 139, 143, 116	CP3.1 (Tue, 2026/6/30 11:35-11:55; Lecture Theatre 3), CP3.2 (Tue, 2026/6/30 11:55-12:15; Lecture Theatre 3), CP17 (Thu, 2026/7/2 11:00-11:15; Lecture Theatre 3), CP23.1 (Thu, 2026/7/2 15:45-16:25; Lecture Theatre 3)
Gray, Darren J	276	S3 (Tue, 2026/6/30 11:00-11:20; Lecture Theatre 3)
Gray, Rachael	261	CP10.2 (Wed, 2026/7/1 11:45-12:15; Lecture Theatre 3)
Green, Jennifer	150	CP2.1 (Tue, 2026/6/30 11:45-12:15; Lecture Theatre 2)
Greenwood, Brian	166	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)

Grievink, Sanne	123	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
Guaci, Charles	217	CP12.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 2)
Gunawan, Gunawan	245	
Gyamfi, Emmanuel	220	CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
Häberli, Cécile	162	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Haining, Jessica	131	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)
Hall, Luke	134	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Luke Hall	, , ,	CP17 (Thu, 2026/7/2 11:00-11:15; Lecture Theatre 3), CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3), CP17.2 (Thu, 2026/7/2 12:05-12:15; Lecture Theatre 3), CP17Q (Thu, 2026/7/2 12:15-12:30; Lecture Theatre 3)
Hamersma, Ashley	248	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Hansman, Grant	269	S5 (Wed, 2026/7/1 13:30-13:50; Lecture Theatre 3)
Haque, Md Anowarul	243	CP16 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 2)
Harder, Kate	262	CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3)
Harman, Madison	248	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Harris, Alexander	166	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Christopher Hart	, ,	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1), CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1), CP8Q (Wed, 2026/7/1 12:15-12:30; Lecture Theatre 1)

Hart, Christopher	119, 137	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1), CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1)
Hartel, Gunter	218	CP13 (Wed, 2026/7/1 13:50-14:30; Lecture Theatre 3)
Hartmann, Arik	248	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Hasang, Wina	132, 109	CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2), CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Heath, William	198, 211	CP14 (Thu, 2026/7/2 9:45-10:30; Plenary Lecture Theatre), CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2)
Hedtke, Shannon	121, 128	CP3 (Tue, 2026/6/30 11:20-11:35; Lecture Theatre 3), CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Henshall, Isabelle	254	CP18.1 (Thu, 2026/7/2 14:40-14:45; Lecture Theatre 1)
Hermans, Ian F	211	CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2)
Hesping, Eva	198	CP14 (Thu, 2026/7/2 9:45-10:30; Plenary Lecture Theatre)
Hetzel, Manuel	235	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Hii, Sze Fui	236	CP20 (Thu, 2026/7/2 13:30-14:00; Lecture Theatre 3)
Hodgkinson, Jane	160	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Holz, Lauren	211, 198	CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2), CP14 (Thu, 2026/7/2 9:45-10:30; Plenary Lecture Theatre)
Hongsrichan, Nuttanan	251	CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Howden, Ben	222	CP23 (Thu, 2026/7/2 15:30-15:45; Lecture Theatre 3)
Hufschmid, Jasmin	131	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)

Huggins, Lucas	258, 148, 236	CP2 (Tue, 2026/6/30 11:30-11:45; Lecture Theatre 2), CP2.1 (Tue, 2026/6/30 11:45-12:15; Lecture Theatre 2), CP20 (Thu, 2026/7/2 13:30-14:00; Lecture Theatre 3)
Hutton, Tia	218	CP13 (Wed, 2026/7/1 13:50-14:30; Lecture Theatre 3)
Huynh, Long	240, 249	CP1 (Tue, 2026/6/30 11:45-12:05; Lecture Theatre 1), CP1.1 (Tue, 2026/6/30 12:05-12:15; Lecture Theatre 1)
Huynh, Priscilla	113	CP12.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 2)
Hviid, Lars	109	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
I. Stanisic, Danielle	208	CP9.1 (Wed, 2026/7/1 11:35-12:05; Lecture Theatre 2)
I. Webb, Andrew	237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Inobaya, Marianette	102	CP3.1 (Tue, 2026/6/30 11:35-11:55; Lecture Theatre 3)
Inpankaew, Tawin	258	CP2 (Tue, 2026/6/30 11:30-11:45; Lecture Theatre 2)
J. Emery-Corbin, Samantha	237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
J. Fairweather, Stephen	212	CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
J. Sandow, Jarrod	237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Abdul Jabbar	, , ,	S7 (Thu, 2026/7/2 11:00-11:20; Lecture Theatre 2), CP16 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 2), CP16.1 (Thu, 2026/7/2 11:50-12:05; Lecture Theatre 2), S7Q (Thu, 2026/7/2 12:05-12:30; Lecture Theatre 2)

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Jackson, Philomena	128	CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Jacobson, Caroline L	115	CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3)
Jansen, Robert S.	135	CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
Jasni, Nur Zakirah	216	CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
Jefferson, Natalie	112	CP10.2 (Wed, 2026/7/1 11:45-12:15; Lecture Theatre 3)
Jenkins, Cheryl	168	CP22 (Thu, 2026/7/2 15:30-16:20; Lecture Theatre 2)

Aaron Jex	, , , ,	BMM (Tue, 2026/6/30 9:45-10:30; Plenary Lecture Theatre), Tribute (Wed, 2026/7/1 9:00-9:10; Plenary Lecture Theatre), BOM (Wed, 2026/7/1 9:10-9:45; Plenary Lecture Theatre), Sprent (Wed, 2026/7/1 15:30-16:00; Plenary Lecture Theatre), AGM (Wed, 2026/7/1 16:00-18:15; Plenary Lecture Theatre)
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Ji, Yijia	205	CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
Ji, Yuchi	132	CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2)
Jiz, Mario	102	CP3.1 (Tue, 2026/6/30 11:35-11:55; Lecture Theatre 3)
Ellis Joch	, ,	CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1), CP11.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 1), CP11Q (Wed, 2026/7/1 14:45-15:00; Lecture Theatre 1)
Joch, Ellis	271	CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
Joglekar, Alok	146	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
Johnson, Michael	145, 146	CP9.2 (Wed, 2026/7/1 12:05-12:15; Lecture Theatre 2), CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)

Jones, Lisa	100	CP7 (Wed, 2026/7/1 9:45-10:30; Plenary Lecture Theatre)
Jones, Malcolm	105, 143, 147, 155	CP3.2 (Tue, 2026/6/30 11:55-12:15; Lecture Theatre 3), CP17 (Thu, 2026/7/2 11:00-11:15; Lecture Theatre 3), CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3), CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Joone, C	262	CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3)
Jore, Matthijs	123	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
Julien, Jean-Philippe	123	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
K Smyth, Gordon	237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Kaethner, Marc	157	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Kaius-Ome, Maria	177	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
Kalupahana, Anil	140, 141	CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1), CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
Kapoor, Shilpa	148, 172, 216, 202	CP2.1 (Tue, 2026/6/30 11:45-12:15; Lecture Theatre 2), CP14 (Thu, 2026/7/2 9:45-10:30; Plenary Lecture Theatre), CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1), CP21 (Thu, 2026/7/2 15:30-16:00; Lecture Theatre 1)
Shilpa Kapoor	, ,	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1), CP18.1 (Thu, 2026/7/2 14:40-14:45; Lecture Theatre 1), CP18Q (Thu, 2026/7/2 14:45-15:00; Lecture Theatre 1)
Karl, Stephan	174	CP3.2 (Tue, 2026/6/30 11:55-12:15; Lecture Theatre 3)

Karunarathne, Janani	238	CP5.1 (Tue, 2026/6/30 14:00-14:30; Lecture Theatre 2)
Kattenberg, Johanna H.	235	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Kazura, James W	132, 235	CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2), CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Keiser, Jennifer	162	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Kelly, Ashton	204	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2)
Kelly, Ashton M	211	CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2)
Kennedy, Carl	120	CP14 (Thu, 2026/7/2 9:45-10:30; Plenary Lecture Theatre)
Kennedy, Karina	192	CP17.2 (Thu, 2026/7/2 12:05-12:15; Lecture Theatre 3)
Khan, Mehran	122, 193, 195	CP20.1 (Thu, 2026/7/2 14:00-14:30; Lecture Theatre 3), CP22 (Thu, 2026/7/2 15:30-16:20; Lecture Theatre 2), CP22 (Thu, 2026/7/2 15:30-16:20; Lecture Theatre 2)
Khieu, Virak	276	S3 (Tue, 2026/6/30 11:00-11:20; Lecture Theatre 3)
Khim, Nimol	235	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Khoshmanesh, Khashayar	201	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Khoury, David	226	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
Khurana, Sachin	237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Kimkamkaew, Siriwan	251	CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
King, Maggie	211	CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2)
King, Tanya	110	CP16 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 2)
Kinsella, Mike	248	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)

Kirkwood, Nicolle	150	CP2.1 (Tue, 2026/6/30 11:45-12:15; Lecture Theatre 2)
Klafke, Guilherme	250	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1)
Kniha, Edwin	258	CP2 (Tue, 2026/6/30 11:30-11:45; Lecture Theatre 2)
Koehler, Anson	222, 215, 167, 255, 152, 257, 162	CP23 (Thu, 2026/7/2 15:30-15:45; Lecture Theatre 3), CP23.1 (Thu, 2026/7/2 15:45-16:25; Lecture Theatre 3), CP21.1 (Thu, 2026/7/2 16:00-16:20; Lecture Theatre 1), CP21.1 (Thu, 2026/7/2 16:00-16:20; Lecture Theatre 1), CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3), CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3), CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Koinari, Melanie	174	CP3.2 (Tue, 2026/6/30 11:55-12:15; Lecture Theatre 3)
Kooij, Taco	123, 135	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1), CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
Kreplins, Tracey	178	CP16.1 (Thu, 2026/7/2 11:50-12:05; Lecture Theatre 2)
Kreutzfeld, Oriana	274, 206	S1 (Tue, 2026/6/30 11:05-11:45; Lecture Theatre 1), CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
Kristan, Mojca	118	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
Kuligowski, Michael	239, 228	CP4 (Tue, 2026/6/30 13:35-13:50; Lecture Theatre 1), CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Kumar, Chaya A	242	CP13 (Wed, 2026/7/1 13:50-14:30; Lecture Theatre 3)
Kumar, Manoharan	125	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2)

Kurtovic, Liriye	227, 166	CP5.1 (Tue, 2026/6/30 14:00-14:30; Lecture Theatre 2), CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Kutty, Rohith	205	CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
Rohith Kutty	, , ,	S6 (Thu, 2026/7/2 11:00-11:20; Lecture Theatre 1), CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1), CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1), S6Q (Thu, 2026/7/2 12:15-12:30; Lecture Theatre 1)
Laleu, Benoit	180	CP12.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 2)
Lam, Alex	170, 225, 237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1), CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1), CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Lam, Kwong Sum	126	CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1)
Laman, Moses	177, 235	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2), CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Lan, Chunling Blue	142	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1)
Lantero Escolar, Elena	154	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1)
Laoprom, Nonglak	251	CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Laurence, Mike	260	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Lautu-Gumal, Dulcie	235	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Lawrence, Nicole	223	CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1)
Le, Jennifer	153, 187	CP1.1 (Tue, 2026/6/30 12:05-12:15; Lecture Theatre 1), CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1)

Lee, Chiyun	146	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
Lee, Cythia	227	CP5.1 (Tue, 2026/6/30 14:00-14:30; Lecture Theatre 2)
Lee, Erinna F.	156, 157	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1), CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Lee, Jiwon	223	CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1)
Lee, Rogan	150, 143	CP2.1 (Tue, 2026/6/30 11:45-12:15; Lecture Theatre 2), CP17 (Thu, 2026/7/2 11:00-11:15; Lecture Theatre 3)
Lehane, Adele	219, 185, 188, 214	CP1 (Tue, 2026/6/30 11:45-12:05; Lecture Theatre 1), CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1), CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1), CP11.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 1)
Lehnert, Kristina	273	P1 (Tue, 2026/6/30 15:30-16:15; Plenary Lecture Theatre)
Leonard, Rachel	188	CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1)
Lessene, Guillaume	170	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Li, Chaoyi	155	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Li, Mengbo	170	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Li, Qiuyuan	158	CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1)
Li, Yunchuan	224	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Liang, Ruijia	219, 188	CP1 (Tue, 2026/6/30 11:45-12:05; Lecture Theatre 1), CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1)
Liebman, Katherine M.	164	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1)

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Ligda, Panagiota	258	CP2 (Tue, 2026/6/30 11:30-11:45; Lecture Theatre 2)
Lim, Allyn	179	CP6.2 (Tue, 2026/6/30 14:25-14:45; Lecture Theatre 3)
Lim, Nicole	266	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
Ling, Elysia	217	CP12.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 2)
Liu, Darren	226	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
Liu, Joy	201	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Liu, Xiangning	158	CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1)
Liyanage, Tharaka	131, 231	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3), CP16.1 (Thu, 2026/7/2 11:50-12:05; Lecture Theatre 2)
Lloyd, Joan B	115	CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3)
Lloyd Williams, Oscar H.	109	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
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Locke, Emily	227	CP5.1 (Tue, 2026/6/30 14:00-14:30; Lecture Theatre 2)
Longley, Rhea	238, 159	CP5.1 (Tue, 2026/6/30 14:00-14:30; Lecture Theatre 2), CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2)
Longo, Ana	248	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)

Longobardi, Mariangela	123	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
Lopez-Perez, Mary	109	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Loukas, Alex	125, 199, 221	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2), CP9.1 (Wed, 2026/7/1 11:35-12:05; Lecture Theatre 2), CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Loukopoulos, Panayotis	131	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)
Lucentini, Livia	131	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)
Lucet, Isabelle	170	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Luque, Daniel	145	CP9.2 (Wed, 2026/7/1 12:05-12:15; Lecture Theatre 2)
Lymber, Alan	191	CP20.1 (Thu, 2026/7/2 14:00-14:30; Lecture Theatre 3)
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M. Athar Ali, Rana	163	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
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MacRaild, Christopher	184	CP11.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 1)
Madan, Sonakshi	235	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Madanitsa, Mwayiwawo	109	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Madi Salloum, Priscila	210	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)
Maia, Carla	258	CP2 (Tue, 2026/6/30 11:30-11:45; Lecture Theatre 2)
Maier, Alexander G.	126	CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1)
Makani, Venkata Krishna Kanth	146	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
Makota, Victor	212	CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
Manage, Lenin	186	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Maneniaru, D	262	CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3)
Maneniaru, Daphne	272	CP17.2 (Thu, 2026/7/2 12:05-12:15; Lecture Theatre 3)
Mantila, Daisy	177	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
Mao, Jiadong	167	CP21.1 (Thu, 2026/7/2 16:00-16:20; Lecture Theatre 1)
Marcal Antunes, Ines	151	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Marchant, Jonathan	275	P2 (Tue, 2026/6/30 16:15-17:00; Plenary Lecture Theatre)
Marina, Rina	245	
Martelli, Paolo	112	CP10.2 (Wed, 2026/7/1 11:45-12:15; Lecture Theatre 3)
Martin, Elyse	248	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)

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Mationg, Mary Lorraine	102	CP3.1 (Tue, 2026/6/30 11:35-11:55; Lecture Theatre 3)
Matoba, Naoki	118	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
McAllister, Jane	222	CP23 (Thu, 2026/7/2 15:30-15:45; Lecture Theatre 3)
McCann, Kirsty	235, 177	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3), CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
McCauley, John	198, 154	CP14 (Thu, 2026/7/2 9:45-10:30; Plenary Lecture Theatre), CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1)
McCluskey, Adam	271	CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
McConville, Robyn	198	CP14 (Thu, 2026/7/2 9:45-10:30; Plenary Lecture Theatre)
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McHugh, Connor	125, 199, 221	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2), CP9.1 (Wed, 2026/7/1 11:35-12:05; Lecture Theatre 2), CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
McHugh, Emma	240	CP1 (Tue, 2026/6/30 11:45-12:05; Lecture Theatre 1)
McMorran, Brendan	266, 223	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2), CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1)

McNeilly, Tom N.	260	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Meagher, Niamh	132	CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2)
Meggiolaro, Maira Nascimento	150	CP2.1 (Tue, 2026/6/30 11:45-12:15; Lecture Theatre 2)
Mehmood, Naunain	181	CP20 (Thu, 2026/7/2 13:30-14:00; Lecture Theatre 3)
Meijering, Erik	224	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Melrose, Wanye	196, 197	CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3), CP3.2 (Tue, 2026/6/30 11:55-12:15; Lecture Theatre 3)
Mensah, Ernest	121	CP3 (Tue, 2026/6/30 11:20-11:35; Lecture Theatre 3)
Mercedes, Raine	209	CP13.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 3)
Mercoulia, Karolina	222	CP23 (Thu, 2026/7/2 15:30-15:45; Lecture Theatre 3)
Michie, Kate	220	CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
Midem, David	132	CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2)
Miguel, Javier Gonzalez	228	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Miles, Kim	125, 221	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2), CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
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Modak, Joyanta	203	CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
Morganti, Giulia	131	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)
Morrow, Joshua	205	CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)

Mota, Maria	232	CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
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Mukutmoni, Mandira	253	CP3.1 (Tue, 2026/6/30 11:35-11:55; Lecture Theatre 3)
Multari, Dylan	170	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Munro, Jacob	237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Muqaddas, Hira	181	CP20 (Thu, 2026/7/2 13:30-14:00; Lecture Theatre 3)
Murdoch, Jem	241	CP5.1 (Tue, 2026/6/30 14:00-14:30; Lecture Theatre 2)
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Mwapasa, Victor	109	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Naden, K	262	CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3)
Nagaraja, Haleagrahara	125	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2)

Naniogou, Sedou	128	CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Nataraj, Gita	242	CP13 (Wed, 2026/7/1 13:50-14:30; Lecture Theatre 3)
Nath, Tilak Chandra	253	CP3.1 (Tue, 2026/6/30 11:35-11:55; Lecture Theatre 3)
Naung, Myo T.	177	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
Navarro, Severine	105	CP3.2 (Tue, 2026/6/30 11:55-12:15; Lecture Theatre 3)
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Nguyen, Anne	145	CP9.2 (Wed, 2026/7/1 12:05-12:15; Lecture Theatre 2)
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Nguyen, Nghi	162	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Nguyen, Quan	204, 155	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2), CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Nguyen, Tam H	218	CP13 (Wed, 2026/7/1 13:50-14:30; Lecture Theatre 3)
Nguyen, Thuy	258	CP2 (Tue, 2026/6/30 11:30-11:45; Lecture Theatre 2)
Nguyen, William	188	CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1)
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Nisbet, Alasdair	260	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Novotny, David	224	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Nowak, Barbara F.	194	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)

Nuang, Myo	237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Nyarko, Abena A.	128	CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Ockenhouse, Chris	227	CP5.1 (Tue, 2026/6/30 14:00-14:30; Lecture Theatre 2)
Ogolla, Sidney	132	CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2)
O'Grady, Jakub	218	CP13 (Wed, 2026/7/1 13:50-14:30; Lecture Theatre 3)
O'Handley, Ryan	233	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
Ojeda-Rojas, Maria	248	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Olsen, David	198	CP14 (Thu, 2026/7/2 9:45-10:30; Plenary Lecture Theatre)
Olsen, David B.	154	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1)
Ome-Kaius, Maria	235	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Onizawa, Emily	168	CP22 (Thu, 2026/7/2 15:30-16:20; Lecture Theatre 2)
Ono, Masahiro	146	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
Opare, Joseph	121	CP3 (Tue, 2026/6/30 11:20-11:35; Lecture Theatre 3)
Opi, D. Herbert	227	CP5.1 (Tue, 2026/6/30 14:00-14:30; Lecture Theatre 2)
Opoku, Millicent	121, 128	CP3 (Tue, 2026/6/30 11:20-11:35; Lecture Theatre 3), CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Osei, Joseph H. N.	121, 128	CP3 (Tue, 2026/6/30 11:20-11:35; Lecture Theatre 3), CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Oskam, Charlotte	247, 161	CP7 (Wed, 2026/7/1 9:45-10:30; Plenary Lecture Theatre), CP13.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 3)

Oskam, Charlotte Louise	178	CP16.1 (Thu, 2026/7/2 11:50-12:05; Lecture Theatre 2)
Ossipow, Suzy	139	CP3.2 (Tue, 2026/6/30 11:55-12:15; Lecture Theatre 3)
Otieno, Lucas	227	CP5.1 (Tue, 2026/6/30 14:00-14:30; Lecture Theatre 2)
Otoboh, Stanley	132	CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2)
Otoo, Sampson	128	CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Ouedrogo, Jean-Bosco	166	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Page, Stephen W.	271	CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
Painter, Gavin F	211	CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2)
Papenfuss, Tony	202, 207	CP21 (Thu, 2026/7/2 15:30-16:00; Lecture Theatre 1), CP21 (Thu, 2026/7/2 15:30-16:00; Lecture Theatre 1)
Passamonti, Fabrizio	131	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)
Pathirana, Erandi	186	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Pattinson, David J	211	CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2)
Paun, Andrea	258	CP2 (Tue, 2026/6/30 11:30-11:45; Lecture Theatre 2)
Paver, Elizabeth	192	CP17.2 (Thu, 2026/7/2 12:05-12:15; Lecture Theatre 3)
Pavithra, Prakrithi	155	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Pearson, Richard	146	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
Peck, Ashleigh	191	CP20.1 (Thu, 2026/7/2 14:00-14:30; Lecture Theatre 3)
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Peixoto, Maristela P.	152	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
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Pepey, Anaïs	235	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
Perkins, Chris	112	CP10.2 (Wed, 2026/7/1 11:45-12:15; Lecture Theatre 3)
Peters, Grace	220	CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
Phir, Kamija S.	109	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
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Piedrafita, David	171	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Pinto, Isaac L. L.	152	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Pitaksakulrat, Opal	251	CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
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Ponder, Winston	167	CP21.1 (Thu, 2026/7/2 16:00-16:20; Lecture Theatre 1)
Ponsrila, Kanda	251	CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)

Poulin, Robert	210	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)
Michelle Power		CP7 (Wed, 2026/7/1 9:45-10:30; Plenary Lecture Theatre)
Power, Cecilia	194, 130, 265	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3), CP6.2 (Tue, 2026/6/30 14:25-14:45; Lecture Theatre 3), CP6.2 (Tue, 2026/6/30 14:25-14:45; Lecture Theatre 3)
Power, Michelle	261	CP10.2 (Wed, 2026/7/1 11:45-12:15; Lecture Theatre 3)
Preativatanyou, Kanok	258	CP2 (Tue, 2026/6/30 11:30-11:45; Lecture Theatre 2)
Presburger, Rosemary	210	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)
Preston, Sarah	171, 110	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2), CP16 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 2)
Sarah Preston		P2 (Tue, 2026/6/30 16:15-17:00; Plenary Lecture Theatre)
Price, Dan	260	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Proietti, Carla	204, 211	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2), CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2)
Qamar, Abdul Ghaffar	161, 178	CP13.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 3), CP16.1 (Thu, 2026/7/2 11:50-12:05; Lecture Theatre 2)
Qian, Yunan	274	S1 (Tue, 2026/6/30 11:05-11:45; Lecture Theatre 1)
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Quang, Chau	159	CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2)

Quintana, Maria del Pilar	109	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
R. Jex, Aaron	237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Radih, Zaynab	142	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1)
Rae, Angus	223	CP15 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 1)
Rajapakshe, Jayanthe	140, 141	CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1), CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
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Ramsland, Paul A.	194	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
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Reichel, Holly	174	CP3.2 (Tue, 2026/6/30 11:55-12:15; Lecture Theatre 3)
Reis Wunderlich, Mariana	118	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
Rendall, Anthony R.	175	CP10.2 (Wed, 2026/7/1 11:45-12:15; Lecture Theatre 3)
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Rigamonti, Giulia	131	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)
Rissland, Olivia	237	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
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Rochfort Peters, Grace	146	CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2)
Rodriguez-Lapido, Marcos	248	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Roestenberg, Meta	138	CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2)
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Rogers, Madeleine	151	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
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Ryan, Jack	137, 119, 205	CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1), CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1), CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
Ryan, John H.	274	S1 (Tue, 2026/6/30 11:05-11:45; Lecture Theatre 1)
Ryan, Rachael	125	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2)
Ryan, Stephanie	221	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Ryan, Una	114, 115	CP23.1 (Thu, 2026/7/2 15:45-16:25; Lecture Theatre 3), CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3)
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Saeed, Maria	109	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Sait, Michelle	222	CP23 (Thu, 2026/7/2 15:30-15:45; Lecture Theatre 3)

Sako, Yasuhito	276	S3 (Tue, 2026/6/30 11:00-11:20; Lecture Theatre 3)
Saliba, Kevin J.	142, 158	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1), CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1)
Sandoval, Sasha	248	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Sangpeng, Jirawat	251	CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1)
Sangster, Nick	236	CP20 (Thu, 2026/7/2 13:30-14:00; Lecture Theatre 3)
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Sarkar, Eti	199	CP9.1 (Wed, 2026/7/1 11:35-12:05; Lecture Theatre 2)
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Sato, Marcello	276, 276, 276	S3 (Tue, 2026/6/30 11:00-11:20; Lecture Theatre 3), S3 (Tue, 2026/6/30 11:00-11:20; Lecture Theatre 3), S3 (Tue, 2026/6/30 11:00-11:20; Lecture Theatre 3)
Sayers, Claire	226, 220	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1), CP11 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 1)
Scharkie, Tatum	260	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2)
Scheelings, T. Franciscus	152	CP6.1 (Tue, 2026/6/30 13:45-14:25; Lecture Theatre 3)
Schrecker, Henry D.	158	CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1)
Schröder, Johann	115	CP20.2 (Thu, 2026/7/2 14:30-14:45; Lecture Theatre 3)
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Scott, Nichollas	109	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Seid, Mengistu	211	CP9 (Wed, 2026/7/1 11:20-11:35; Lecture Theatre 2)
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Natasha Sharma	, , ,	CP21 (Thu, 2026/7/2 15:30-16:00; Lecture Theatre 1), CP21.1 (Thu, 2026/7/2 16:00-16:20; Lecture Theatre 1), CP21.2 (Thu, 2026/7/2 16:20-16:30; Lecture Theatre 1), CP21Q (Thu, 2026/7/2 16:30-16:40; Lecture Theatre 1)
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Shen, Yanxiang	156	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Sherry, Norelle	222	CP23 (Thu, 2026/7/2 15:30-15:45; Lecture Theatre 3)

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Shingade, Yash	231	CP16.1 (Thu, 2026/7/2 11:50-12:05; Lecture Theatre 2)
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Hannah Siddle	, , , , , ,	CP5 (Tue, 2026/6/30 13:30-14:00; Lecture Theatre 2), CP5.1 (Tue, 2026/6/30 14:00-14:30; Lecture Theatre 2), CP5.2 (Tue, 2026/6/30 14:30-14:45; Lecture Theatre 2), CP5Q (Tue, 2026/6/30 14:45-15:00; Lecture Theatre 2), CP19 (Thu, 2026/7/2 13:30-14:15; Lecture Theatre 2), CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2), CP19Q (Thu, 2026/7/2 14:45-15:00; Lecture Theatre 2)
Siddle, Hannah	103, 250, 133	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1), CP16 (Thu, 2026/7/2 11:20-11:50; Lecture Theatre 2)
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Sikulu-Lord, Maggy	151	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)
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Simone Sleep		OP (Mon, 2026/6/29 9:00-16:00; Lecture Theatre 1)
Hannah Smith		CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1), CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1), CP8Q (Wed, 2026/7/1 12:15-12:30; Lecture Theatre 1)
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Smith, Justine R.	164	CP8 (Wed, 2026/7/1 11:00-11:50; Lecture Theatre 1)
Smith, Maxine	221	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Smith, Nick	100	CP7 (Wed, 2026/7/1 9:45-10:30; Plenary Lecture Theatre)
Smith, Rory C.	158	CP8.1 (Wed, 2026/7/1 11:50-12:15; Lecture Theatre 1)
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Song, Jiangning	156	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
Sormunen, Jani	161	CP13.1 (Wed, 2026/7/1 14:30-14:45; Lecture Theatre 3)
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Stokes, Barbara	219	CP1 (Tue, 2026/6/30 11:45-12:05; Lecture Theatre 1)

Stoter, Rianne	123	CP4.1 (Tue, 2026/6/30 13:50-14:40; Lecture Theatre 1)
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Surve, Nuzhat	242	CP13 (Wed, 2026/7/1 13:50-14:30; Lecture Theatre 3)
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Taki, Aya C.	162, 216	CP12 (Wed, 2026/7/1 13:30-14:30; Lecture Theatre 2), CP15.1 (Thu, 2026/7/2 11:50-12:15; Lecture Theatre 1)
Taleo, Fasihah	236	CP20 (Thu, 2026/7/2 13:30-14:00; Lecture Theatre 3)
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Tavul, Livingstone	235	CP17.1 (Thu, 2026/7/2 11:15-12:05; Lecture Theatre 3)

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ter Kuile, Feiko O.	109	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
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Thomas, Adam	166	CP19.1 (Thu, 2026/7/2 14:15-14:45; Lecture Theatre 2)
Andrew Thompson		P1 (Tue, 2026/6/30 15:30-16:15; Plenary Lecture Theatre)
Thompson, Andrew	170	CP18 (Thu, 2026/7/2 13:30-14:40; Lecture Theatre 1)
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Tonkin, Christopher J.	131	CP10.1 (Wed, 2026/7/1 11:15-11:45; Lecture Theatre 3)
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Trapani, Joe	165	CP9.2 (Wed, 2026/7/1 12:05-12:15; Lecture Theatre 2)
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2026 Annual Conference of the Australian Society for Parasitology Inc.

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Conference Coordinator: Lisa Jones

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- Ines Marcal Antunes, Griffith University
- Simone Sleep, AICIM
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- John Ategeka, Griffith University
- Zhetao Zhang, The Queensland Alliance for Agriculture and Food Innovation
- Chika Zumuk, QIMR Berghofer
- Sandani Athukoralage, The University of Queensland
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- Jessica Scott, James Cook University
- Rebecca Farnell, Federation University Australia
- Tanya King, Federation University Australia
- Brenna Daily, The University of Queensland
- Connor McHugh, James Cook University
- Skye Robertson, The University of Queensland

Student Social Evening Event: This event has been organised by Grace Reeves, Rebecca Farnell and Tanya King.

ECR Breakfast Event panellists: Dr Catherine Gordon, Dr Liisa Ahlstrom, Christopher Dean Goodman, Dr. habil. Kristina Lehnert, Professor Jonathn Marchant and Dr Storm Martin and hosted by Jacinta Macdonald (Griffith)

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Driving Impact in Tropical Health and Emerging Diseases

The Centre for Tropical Health and Emerging Diseases (CTHED) is a proud sponsor of the ASP Annual Conference, where researchers and practitioners dedicated to the control, elimination, and prevention of parasitic and infectious diseases meet to discuss all aspects of parasitology research. We look forward to engaging and networking with our colleagues in the 2026 ASP Annual Conference.

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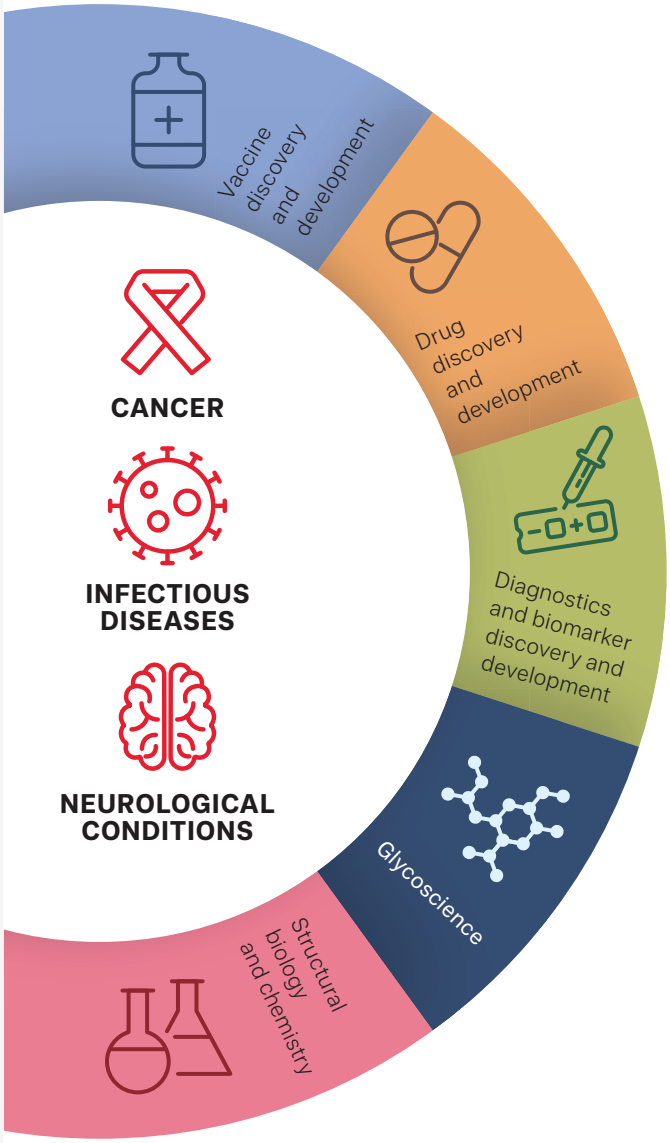

Analytical and structural biology

- Advanced mass spectrometry (including native mass spectrometry*)
- Nuclear magnetic resonance (NMR)
- Biophysical analysis (surface plasmon resonance, isothermal titration calorimetry)
- Glycomics and glycoproteomics
- Spatial transcriptomics and glycomics
- Glycobiological arrays and analytics

Drug discovery and development

- Medicinal chemistry and drug design
- Drug repurposing
- Nanobody drugs
- Compounds Australia* (over 1.5 million compounds)
- NatureBank (over 125,000 extracts and fractions)

Vaccine discovery and development

- Rapid biopolymer vaccine platform
- RNA synthesis and functionalisation
- Novel adjuvant discovery

Other therapeutic platforms

- Cell therapies for spinal injury
- Prebiotics and probiotics

Diagnostics and biomarker discovery

- Liquid biopsy biomarkers and saliva diagnostics
- Nanobody diagnostics
- Microbiomics
- MRI diagnosis (cancer and neurological disorders)
- Label-free bio- and bioaerosol sensors

Biological models and functional studies

- Zebrafish models of disease and toxicity
- Controlled human malaria infection model
- Non-animal models of respiratory infection

Biobanking and data resources

- Biobanking services
- Biobanks (saliva, Parkinson's, olfactory neuronal stem cells, Gold Coast Biobank)

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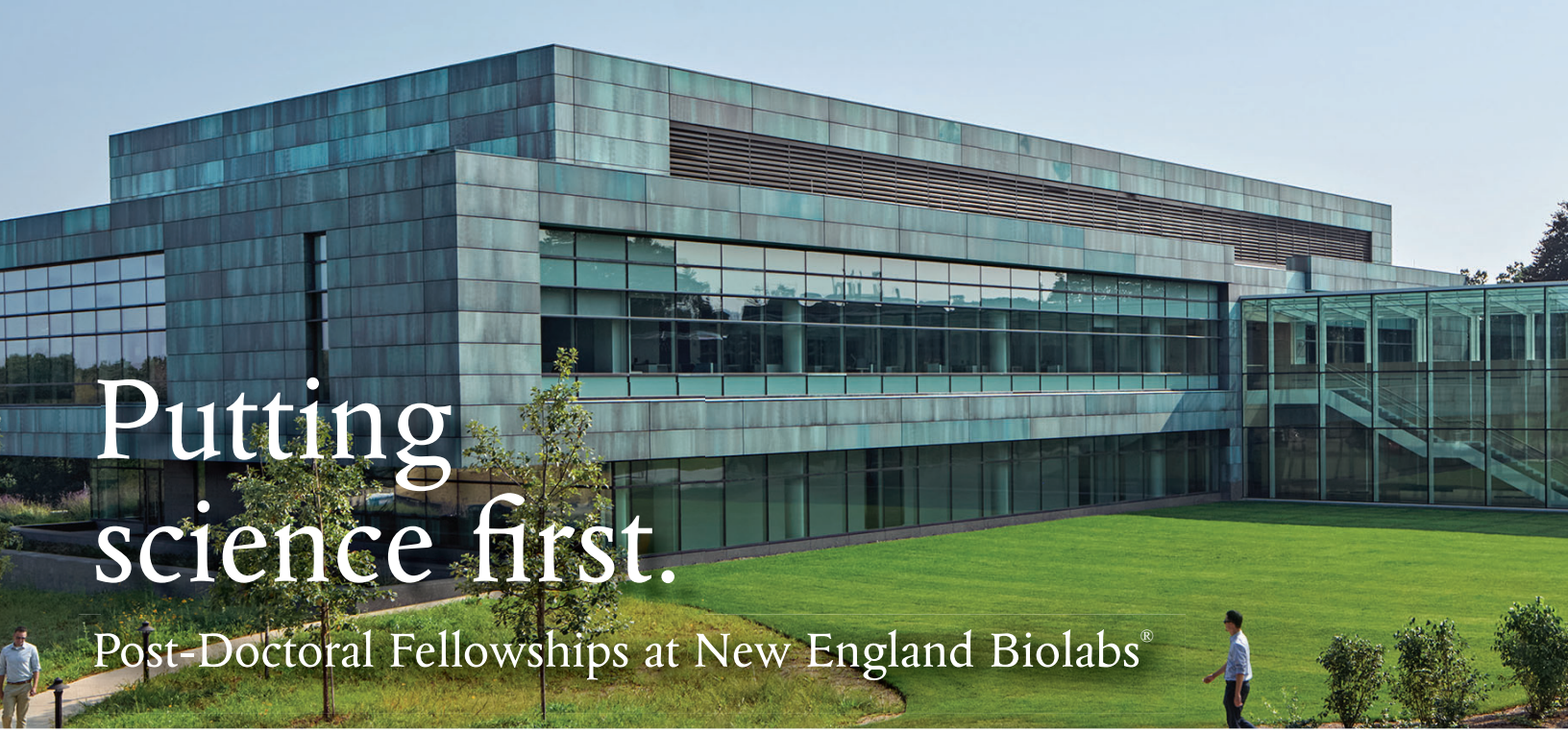

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