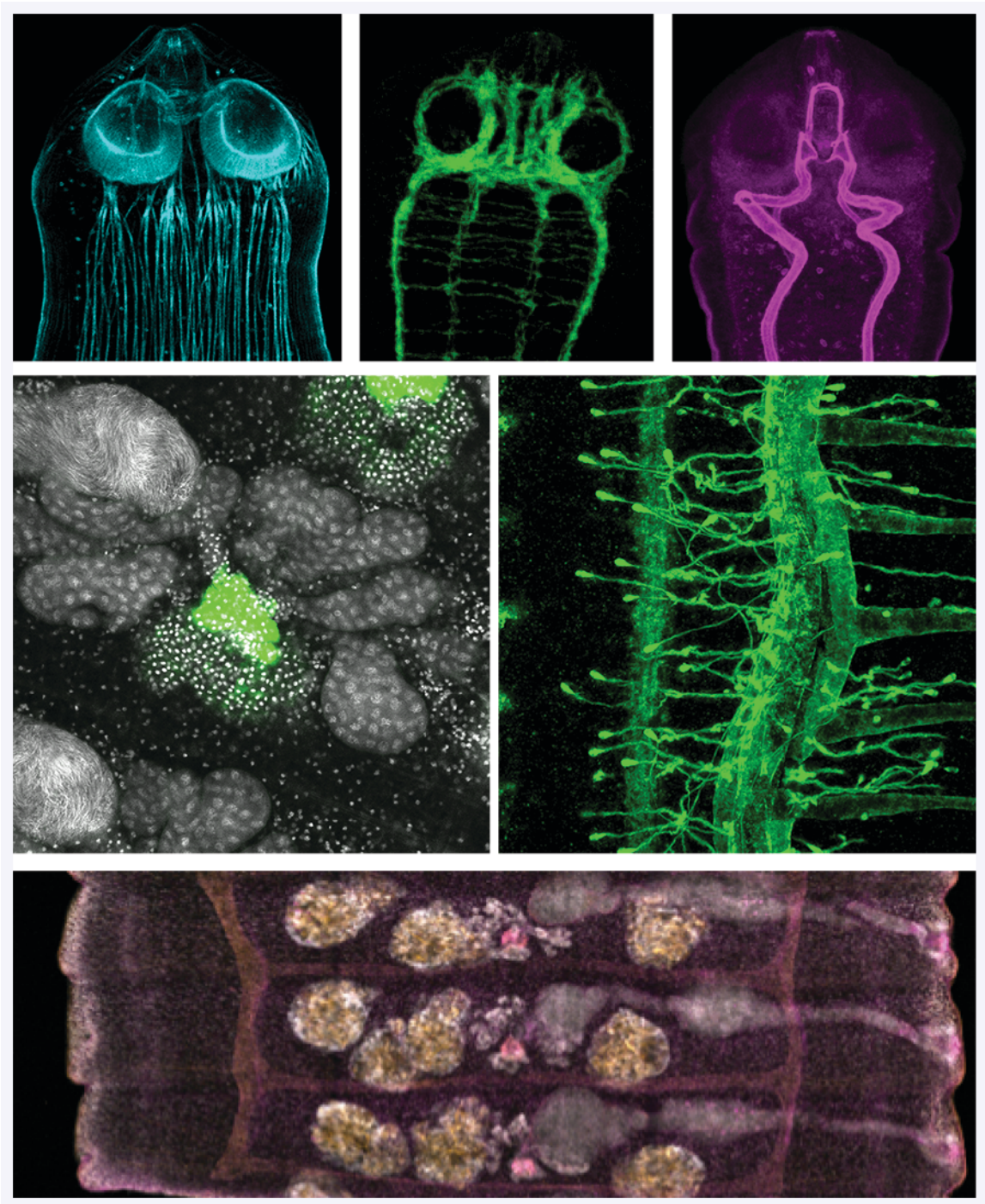
Parasitic Helminths: New Perspectives in Biology and Infection



Hotel Bratsera, Hydra, Greece

3 -8 September 2023

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Beautiful Cover Photos of *Hymenolepis dimunuta* are courtesy of Tania Rozario, University of Georgia, USA!

Parasitic Helminths - New Perspectives in Biology and Infection **3 – 8 September 2023, Hydra, Greece**

	Sunday 3 September	Monday 4 September	Tuesday 5 September	Wednesday 6 September	Thursday 7 September	Friday 8 September
	ARRIVE	**Session 1 Host-Parasite Interactions**	**Session 4 Evolution and Ecosystems**	**Session 7 Human Infection**	**Session 10 Helminth Development**	DEPART
9:00		**Maria Duque-Correa**	**Amy Pedersen**	**Moses Egesa**	**Tania Rozario**	
9:40		Omer Bay	Mark Viney	Emma Houlder	David Mangelsdorf	
10:00		Simone Haeberlein	Lewis Stevens	Bridgious Walusimbi	Christoph Grevelding	
10:20		Kerstin Fischer	Grace Ajakaye	Cornelis Hokke	Jan Dvorak	
10:40-11:10			**Coffee Break**			
		Session 2 Immune Effector Mechanisms	**Session 5 Helminth Immunology**	**Session 8 Immune Activation**	**Session 11 Immunomodulation**	
11:10		**Lida Derevnina**	Minka Breloer	**Michalis Barkoulas**	Thomas Nutman	
11:30			Shinjini Chakraborty		Shashi Singh	
11:50		Unnati Sonawala	Georgios Petrellis	Lara Linnemann	Katherine Smith	
12:10		Alexandra Ehrens	Oyebola Oyesola	Pedro Papotto	Peter Nejsum	
12:30		Nicolas Pionnier	Dionysis Grigoriadis	Kyle Cunningham	Clarissa Prazeres da Costa	
13:00-14:00			**WormBase Parasite Workshop**			
to 16:00			**Afternoon Break**			
		Session 3 Drug Development	**Session 6 Type 2 Immunity**	**Session 9 Drug Resistance**	**Session 12 Non-coding RNAs**	
16:00	**Registration Opens at Bratsera Hotel**	Andrew Fraser	**De'Broski Herbert**	Ray Kaplan	**Vicky Hunt**	
16:20		Daniel Sprague		Anne Lespine		
16:40		Hala Fahs	Pedro Gazzinelli-Guimaraes	Stephen Doyle	Murilo Amaral	
17:00		Wannaporn Ittiprasert	Xinxin Luo	Sarah Cobb	Amy Buck	
17:20		Poster Pitches 1-17	James Hewitson	Poster Pitches 18-35	*End of Session*	
18:00	**Opening Drinks (18:45)**	**Poster Session 1**	**Vlychos Taverna Dinner** (Boat leaves 19:00)	**Poster Session 2**		
19:45	**Keynote: Maria Yazdanbakhsh**				**Bratsera Farewell Dinner** (20:30)	
21:00	**Welcome Dinner Bratsera Hotel**	*20:00 End of Session*		*20:00 End of Session*		

Parasitic Helminths –
New Perspectives in Biology and Infection

2023 Programme

Sunday 3 September

16:00 Registration Opens, Bratsera Hotel

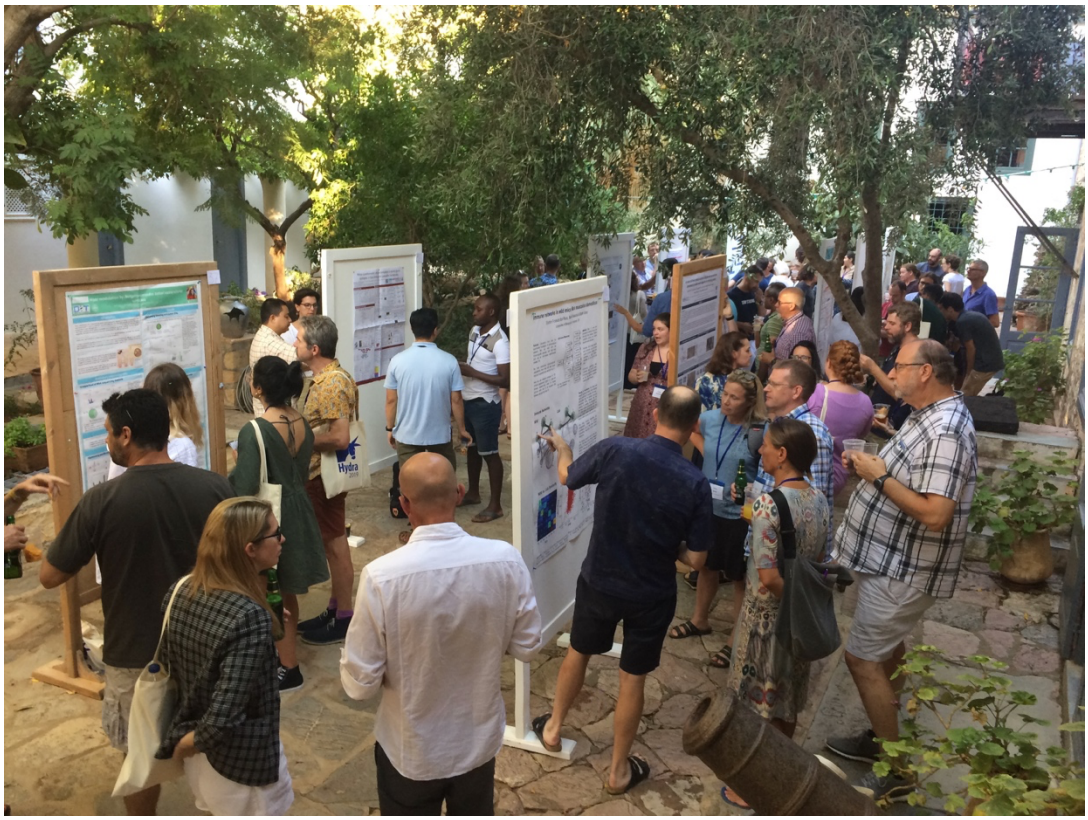
18:45 Pre-lecture Drinks

19:45 Keynote Lecture, Maria Yazdanbakhsh

Chair: Murray Selkirk

21:00 Welcome Dinner, Bratsera Hotel

Poster Session 2019



Timings for all presentations include minimum of 5 minutes for discussion.

Monday 4 September

09:00-10:40 Session 1. Host-Parasite Interactions. Chair Niki Gounaris

09:00	**Maria Duque-Correa**	**Cambridge, UK**	**Unravelling the whipworm niche at the host intestinal epithelia**
09:40	Omer Bay	Manchester, UK	The first genome-scale metabolic model of parasitic whipworm: gateway to the rapid discovery of novel host pathogen interactions and therapeutic targets
10:00	Simone Haeberlein	Giessen, DE	Combined single-cell and spatial transcriptomics provide unprecedented molecular insights valuable for basic and applied research on liver flukes
10:20	Kerstin Fischer	Washington, USA	Deep visual proteomics of the protein inventory of *Onchocerca volvulus* neoplasms

10:40 – 11:10 Coffee Break

11:10 – 12:50 Session 2. Immune Effector Mechanisms. Chair Rick Maizels

11:10	**Lida Derevnina**	**Cambridge, UK**	**Cyst nematodes counteract immunity by inhibiting activation of central nodes of a Solanaceae immune receptor network**
11:50	Unnati Sonawala	Cambridge, UK	Understanding the HYPervariability of HYP effectors in potato cyst nematodes
12:10	Alexandra Ehrens	Bonn, DE	Microfilariae-induced eosinophil ETosis is NOX- and inflammasome-dependent
12:30	Nicolas Pionnier	Manchester, UK	Natural Killer cell activation and memory-like phenotype development following helminth infection

16:00 – 18:00 Session 3. Drug Development. Chair Thomas Spangenberg

16:00	Andrew Fraser	Toronto, CA	Closing in on new anthelmintics that target rhodoquinone-dependent metabolism
16:20	Daniel Sprague	Wisconsin, USA	Developing novel flatworm ion channel ligands to treat various neglected tropical diseases
16:40	Hala Fahs	New York, USA	Multi-species nematode screening uncovers new classes of broad-spectrum anthelmintic compounds.
17:00	Wannaporn Ittiprasert	Washington, USA	All-in-one lipid nanoparticle delivery of programmed gene knock-in in *Schistosoma mansoni*
17:20	**Poster Pitches 1-17 (2-minutes each)**		

18:00 – 20:00 Poster Session, Posters 1-17

Tuesday 5 September

9:00 – 10:40 Session 4. Evolution and Ecosystems. Chair Amy Buck

09:00	**Amy Pedersen**	**Edinburgh, UK**	**The ecological drivers of helminth infection and immunity in a lab-to-wild mouse model**
09:40	Mark Viney	Liverpool, UK	Is *Strongyloides stercoralis* in people a zoonosis from dogs? – a whole-genome sequencing approach
10:00	Lewis Stevens	Wellcome Sanger, UK	Ancient diversity in host-parasite interaction genes in a model parasitic nematode
10:20	Grace Ajakaje	Adekunda, Nigeria	Proof-of-concept multilocus sequence typing scheme to investigate hybridization in *Schistosoma haematobium*

11:10 – 12:50 Session 5. Helminth Immunology. Chair Richard Grencis

11:10	Minka Breloer	BNITM, DE	CD160 regulates innate anti-helminth immune responses
11:30	Shinjini Chakraborty	York, UK	Chronic helminth infection alters bone marrow haematopoiesis via IL-4
11:50	Georgios Petrellis	Liege, BE	IL-4 receptor-α signaling regulates lung macrophages during helminth coinfection resulting in enhanced gammaherpesvirus permissiveness
12:10	Oyebola Oyesola	LPD/NIH, USA	Helminth exposure protects against murine SARS-CoV-2 infection through macrophage dependent T cell activation.
12:30	Dionysis Grigoriadis	EMBL-EBI, UK	WormBase ParaSite in 2023
13:00 – 14:00	**WormBase Parasite Workshop**		

16:00 – 17:40 Session 6. Type 2 Immunity. Chair Katie Smith

16:00	**De'Broski Herbert**	**Pennsylvania, USA**	**Skin sensory neurons repel schistosomiasis**
16:40	Pedro Gazzinelli-Guimaraes	NIAID/NIH	Decoding at single cell resolution the molecular and functional program of pathogenic Th2 cells subsets in humans
17:00	Xinxin Luo	Karolinska, SWE	Liver X receptor controls Tuft cell-ILC2 circuit impairing anti-helminth immunity
17:20	James Hewitson	York, UK	Platelet-immune cell cross-talk in the type 2 inflammatory response to *Schistosoma mansoni*

19:00 Boat Trip and Vlychos Taverna Dinner

Wednesday 6 September

09:00 – 10:40 Session 7. Human Infection. Chair Padraic Fallon

09:00	**Moses Egesa**	**MRC/UVRI/ LSHTM Uganda**	**Establishing a single sex *Schistosoma mansoni* controlled human infection model for Uganda**
09:40	Emma Houlder	Leiden, NL	Immune responses in controlled human *Schistosoma mansoni* infection, lessons from single and reinfection studies.
10:00	Bridgious Walusimbi	MRC/UVRI/ LSHTM Uganda	Are the effects of helminth infection and urbanisation on one's cardiovascular risk mediated via the gut microbiome?
10:20	Cornellis Hokke	Leiden, NL	In-depth characterisation of *Brugia malayi* glycosylation and unraveling of cross-reactive anti-glycan antibody responses in filarial nematode infections

11:10 – 12:50 Session 8. Immune Activation. Chair Oyebola Oyesola

11:10	**Michalis Barkoulas**	**Imperial College UK**	***C. elegans* as a tractable host to study natural infections by oomycetes**
11:50	Lara Linnemann	BNITM, GER	Characterization of the regulative role of the C-type lectin receptor MINCLE in the initiation of anti-helminth immune responses
12:10	Pedro Papotto	Manchester, UK	Dermal γδ17 T cells orchestrate innate and adaptive immunity in distal organs during nematode infection
12:30	Kyle Cunningham	Glasgow, UK	A family of helminth-derived TGF-β mimics provide key insights to innate and adaptive immune cell activation

16:00 – 17:20 Session 9. Drug Resistance. Chair Murray Selkirk

16:00	Ray Kaplan	St George's, West Indies	Molecular evidence of widespread benzimidazole drug resistance in *Ancylostoma caninum* from domestic dogs throughout the USA and discovery of a novel isotype-1 β-tubulin benzimidazole resistance mutation
16:20	Anne Lespine	INRAE, FR	Role of nematode ABCB transporters and their regulation in anthelmintic resistance
16:40	Stephen Doyle	Sanger, UK	Genomic landscape of drug response reveals mediators of anthelmintic resistance
17:00	Sarah Cobb	Texas, USA	Understanding the basic biology of juvenile schistosomes by studying stem cells

17:20	**Poster Pitches 18-34 (2-minutes each)**

18:00 – 20:00 Poster Session 2, Posters 18-35

Thursday 7 September

9:00 – 10:40 Session 10. Helminth Development. Chair Jim Collins

09:00	**Tania Rozario**	**Athens, Georgia, USA**	**Understanding signals that regulate stem cells and germ cells in the regeneration-competent neck of the rat tapeworm, *Hymenolepis diminuta***
09:40	David Mangelsdorf	Texas, USA	The nuclear receptor paradigm of infection in parasitic nematodes
10:00	Christoph Grevelding	Giessen, DE	Single-cell transcriptomics of *Schistosoma mansoni* oocytes identifies a retinoid acid receptor essential for meiosis entry, zygote development, and egg formation
10:20	Jan Dvorak	Czech University	Egg-cellent insights: Transcriptomic analysis of *S. mansoni* egg development – winners vs. losers

11:10 – 12:50 Session 11. Immunomodulation. Chair Minka Breloer

11:10	Thomas Nutman	NIAID/NIH	A filarial parasite-encoded IL-5 antagonist suggests a novel strategy used by helminths to modulate host responses
11:30	Shashi Singh	Glasgow, UK	Transforming growth factor beta (TGFβ) mimic 4 (TGM4) of *Heligmosomoides polygyrus* targets myeloid cells through TGFβ receptors and multiple coreceptors
11:50	Katherine Smith	Cardiff, UK	Greedy Worms: manipulating PUFA metabolism to survive and influence host immunity
12:10	Peter Nejsum	Aarhus, DEN	*Ascaris suum* extracellular vesicles target human monocytes to generate a unique phenotype affecting T-cell anergy
12:30	Clarissa Prazeres da Costa	Munich, DE	Helminthic glutamate dehydrogenase-dependent PGE2 production in monocyte and microglia potentiates Treg development with distinct transcriptional profiles

16:00 – 18:00 Session 12 Non-coding RNAs. Chair Dick Davis

16:00	**Vicky Hunt**	**Bath, UK**	**The role of secreted exosome-like vesicles in parasite-host interactions and potential as a biomarker in *Strongyloides* infection**
16:40	Murilo Amaral	Sao Paolo, BRA	Schistosomal extracellular vesicle-enclosed long non-coding RNAs are transferred to the mammalian host
17:00	Amy Buck	Edinburgh, UK	RNA communication in helminth-host interactions

20:30 Bratsera Farewell Dinner

Friday 8 September: Delegates depart.

MARIA YAZDANBAKHSH

Leiden University Center of Infectious Diseases, Leiden, The Netherlands

Maria Yazdanbakhsh studied Parasitology at London School of Hygiene and Tropical Medicine and her PhD (Amsterdam University) and her post-doc (Imperial college London) involved studies of helminth infections. She is currently heading the Parasitology department at Leiden University Medical Center in the Netherlands. Her group is interested in the interaction between the immune system and helminths. Through many collaborations with researchers in low and middle income countries, her group has been able to combine field studies with cutting edge technologies to profile the human immune system in areas where helminth infections are highly endemic. Conducting trials with anthelmintics, the contribution of current helminth infections to modulation of the immune system has been studied. More recently, her group has become interested in establishing and immune profiling of controlled human helminth infections in her department. Her lab studies the impact of a modulated immune system not only on inflammatory diseases but also on vaccine responses to be able to identify pathways that can be modulated for better control of inflammation and vaccine responses..

Helminth infections and the immune system: from field studies and controlled human infection trials

Our textbook knowledge of the immune system relies largely on studies conducted in laboratory mice or humans living in affluent regions of the world, where exposure to microorganisms and parasites, in particular to helminths, is low. In many low-income regions of the world, helminth infections are the norm and in many areas 100% of communities can be infected with helminths, which profoundly affects the immune system and its function. Studies in populations living in areas endemic for helminth infections have shown strong type 2 and regulatory immune responses that in part can be driven by helminth-derived molecules. In the same areas, lower frequencies of naïve cells along with highly differentiated innate and adaptive immune cells expressing inhibitory molecules appear to be the norm. Such immune profiles seem to be associated with lower prevalence of allergic diseases, less severe COVID-19, but also poorer responses to some vaccines. Whether helminths alone, or other environmental factors are responsible for a modulated immune system that controls responses to incoming allergens or antigens in a vaccine, might be addressed by studying the immunological perturbations during controlled human helminth infections. In such studies, the precise time of infection and the load can be controlled, and the immune response can be followed up longitudinally. Moreover, in the future, it might be possible to assess the response of these helminth infected volunteers to allergens and vaccines. Currently, the use of mass cytometry and single cell transcriptomics of samples from individuals exposed to helminths in endemic areas and those undergoing controlled helminth infection, are providing us with granular data on how the environment and helminth infections shape the immune system, with the hope to identify pathways that can be used to suppress inflammation or boost vaccine responses.

MARÍA A. DUQUE-CORREA

Wellcome-MRC Cambridge Stem Cell Institute, University of Cambridge, UK.

***Dr Maria Duque-Correa** completed her studies of Biology at the University of Antioquia in Colombia, where her undergraduate thesis focused on the role of macrophage activation in Mycobacterium tuberculosis control. She then went to the Mayo Clinic in Arizona, USA to work as a research associate in projects investigating the effect of age on macrophage and dendritic cell responses during cancer. Afterwards, Dr Duque-Correa undertook a PhD at the Max Planck Institute for Infection Biology in Berlin, Germany. Her PhD thesis studied the role of macrophage arginase in granuloma immunopathology during M. tuberculosis infection. For her postdoctoral studies, Maria joined the Wellcome Sanger Institute where she investigated host-parasite interactions that drive immune responses to whipworms (Trichuris sp).*

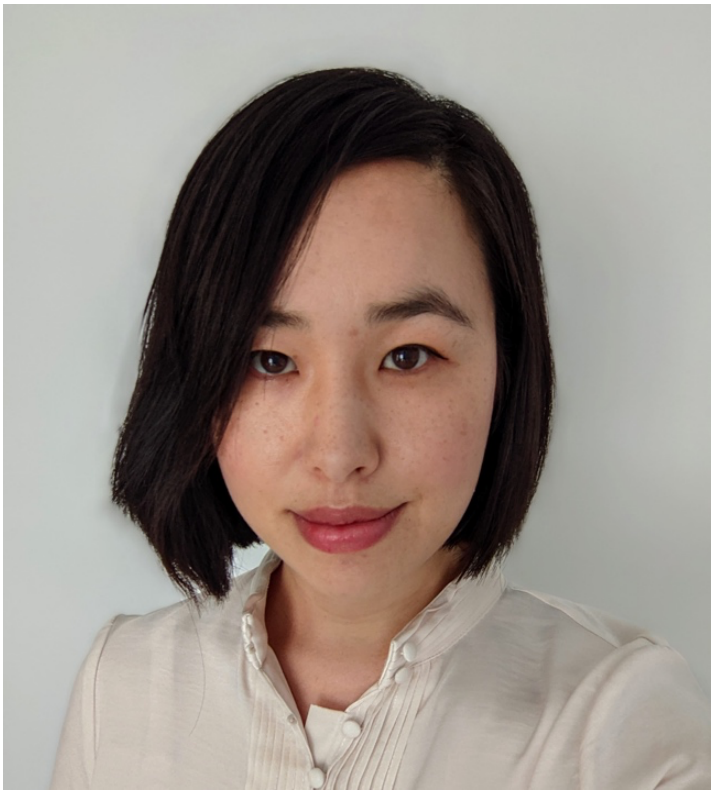
During her postdoc, Maria was funded first by a Marie Sklodowska-Curie fellowship and then, by a transition to independence David Sainsbury Fellowship from the National Centre for the Replacement, Refinement and Reduction of Animals in Research. Awarded a Wellcome Sir Henry Dale Fellowship, Maria started her own research group at the Cambridge Institute of Therapeutic Immunology and Infectious Disease at the University of Cambridge in January 2022 and joined the Wellcome-MRC Cambridge Stem Cell Institute as Principal Investigator in September 2022.

Unravelling the whipworm niche at the host intestinal epithelia

Whipworms *(Trichuris trichiura)* infect hundreds of millions of people causing trichuriasis, a major neglected disease. Whipworms are large metazoan parasites that inhabit a multi-intracellular niche within their host caecal epithelia, where they manipulate mucosal physiology and inflammation through interactions with the intestinal epithelial cells and stem cell niche. These interactions enable chronic infections where whipworms are tolerated for years; but at a mechanistic level, how they operate is not understood. My research aims to define these interactions and bring a mechanistic understanding to how they underpin whipworm invasion, colonisation, and persistence in their mucosal niche. To address this aim, my lab employs a combination of *in vivo* and *in vitro* (using caecaloids) models of *Trichuris muris* infection and imaging and transcriptomic analysis. Using these models, we have shown that *T. muris* first-stage (L1) larvae degrade mucus layers to access epithelial cells. In early syncytial tunnels, larvae are completely intracellular, woven through multiple live dividing cells. Moreover, using single-cell RNA sequencing of infected mouse caecum and caecaloids, we revealed that progression of infection results in cell damage and an expansion of enterocytes expressing of *Isg15*, potentially instigating the host immune response to the whipworm and tissue repair. Excitingly, we have now maintained whipworm-infected caecaloids for 24 days and have observed growth and moulting of whipworms up to the L4 stage at times after infection akin those *in vivo.* These results suggest whipworm-infected caecaloids can successfully support the *in vitro* life cycle *T. muris,* opening new opportunities to study host intestinal epithelial interactions with whipworms through infection, while reducing the number of animals required for these studies. Collectively, our research will unravel intestinal epithelium invasion by whipworms and reveal specific host-parasite interactions that allow the whipworm to establish and persist in its multi-intracellular niche.

LIDA DEREVNINA

Crop Science Centre, Department of Plant Sciences, University of Cambridge, UK



Lida obtained her PhD in plant breeding and plant pathology from the University of Sydney, Australia, where she identified and characterized rust resistance genes in cultivated barley. After completing her PhD, she joined the University of California, Davis, USA, as a postdoctoral researcher, working in comparative genomics of downy mildews. Following this, Lida was awarded a Marie Skłodowska-Curie independent fellowship at The Sainsbury Laboratory (TSL) in Norwich, UK, to study plant intracellular immune receptor networks. Lida is currently the head of the Crop Pathogen Immunity group at the Crop Science Centre, Department of Plant Sciences, University of Cambridge, UK. Her research primarily revolves around understanding the molecular mechanisms employed by pathogens to disrupt plant immune receptor function.

Cyst nematodes counteract immunity by inhibiting activation of central nodes of a Solanaceae immune receptor network

Mauricio P. Contreras[1], Hsuan Pai[1], Muniyandi Selvaraj[1], AmirAli Toghani[1], David M. Lawson[2], Yasin Tumtas[3], Cian Duggan[3], Enoch Lok Him Yuen[3], Clare E.M. Stevenson[2], Adeline Harant, Abbas Maqbool[2], Chih-Hang Wu[4], Tolga O. Bozkurt[3], Sophien Kamoun[1] and Lida Derevnina[1†]

[1]The Sainsbury Laboratory, University of East Anglia, Norwich, UK; [2]Department of Biochemistry and Metabolism, John Innes Centre, Norwich, UK; [3]Department of Life Sciences, Imperial College, London, UK; [4]Institute of Plant and Microbial Biology, Academia Sinica, Taipei, Taiwan[†];
[†]Present address: Crop Science Centre, Department of Plant Sciences, University of Cambridge, UK

During infection, plant pathogens deploy virulence proteins, termed effectors, that suppress plant immune responses and promote disease. Despite their important role in pathogenicity, our understanding of the mechanisms underlying effector function remain relatively limited. We previously identified SS15 – a SPRYSEC-type effector derived from the potato cyst nematode *Globodera rostochiensis* – as a potent suppressor of immunity in Solanaceous plants. SS15 targets the function of helper NLRs, namely NRC2 and NRC3, that are central nodes of a complex immune signalling network. Using biochemical and cellular approaches, we demonstrate that SS15 binds to and inhibits NRC2 oligomerization and plasma membrane association, which are critical for immune signalling. To overcome this suppression, we introduced mutations in the SS15-NRC2 binding interface and bioengineered an NRC2 variant that evades inhibition and restores NRC2 function in the presence of SS15. Our work exemplifies how a deeper mechanistic understanding of effector biology can provide valuable insights for developing novel strategies to generate disease resistant crops.

AMY B. PEDERSEN

University of Edinburgh; Edinburgh, UK

Amy Pedersen is a Professor of Disease Ecology at the University of Edinburgh. The central aim of her research is to better understand how parasites and pathogens impact the fitness and dynamics of their wild hosts, specifically by recognising and understanding the complexities that are inherent in natural systems. This novel approach is important, as while laboratory-based mouse models continue to be crucial to our understanding of infection and immunity, there are many aspects of the controlled setting that don't capture the complexity of human ecology and natural systems. Pedersen has developed a lab-to-wild mouse – parasite system to better understand what factors determine infection and immunity, and importantly how we can improve disease control strategies.

The ecological drivers of helminth infection and immunity in a lab-to-wild mouse model

Despite great concern about the current global health threat of infectious diseases in humans and domestic animals, we still don't have a clear understanding about how ecological heterogeneity determines infection burdens, disease, transmission, or how to successfully control infections in variable populations. Most drug treatments and vaccines are selected using data from laboratory animal systems such as inbred mice. Our over-reliance on highly controlled, laboratory models may underlie some of our failures to adequately manage disease burdens in real-world settings. We have established a wild-to-lab mouse model in order to investigate the causes and consequences of ecological heterogeneity for host-parasite interactions and disease control. Specifically, this talk will focus on *Heligomosoides polygyrus* and its natural host, wood mice (*Apodemus sylvaticus*) and addresses the following questions: (i) what determines susceptibility and resistance to *H. polygyrus* in the wild? (ii) how does nutritional quality and gut microbiome composition impact *H. polygyrus* infection and immunity? (iii) can we use this lab-to-wild system to test helminth vaccine efficacy and better understand the immune and non-immune pathways that drive chronic helminth infections in the wild? From a series of field and controlled laboratory infection and coinfection experiments, I will show evidence that nutritional availability significantly impacts *H. polygyrus* infection and immunity in the wild and lab. In addition, I will present recent results that demonstrate that the composition of the gut microbiome may impact *H. polygyrus* establishment and resistance. Lastly, I will present recent results suggesting that possible helminth vaccine candidates (e.g. HES and ExWAGO) show evidence of efficacy in this system, but also highlight significant variation in efficacy between individuals. Our results highlight how pairing both the lab and natural setting provides a unique and powerful opportunity to understand the causes and consequences of ecological heterogeneity on infection, immunity and disease control.

DE'BROSKI R HERBERT

University of Pennsylvania, Philadelphia, USA

Dr. De'Broski R Herbert's career has spanned 4 major research institutions across two continents: University of Cape Town, South Africa (2000-2006), University of Cincinnati/Cincinnati Children's Hospital Medical Center (2006-2012), University of California at San Francisco (2012-2016) and the University of Pennsylvania (2016-present). The over-arching goal of the Herbert research program is to use parasitic organisms as a guide to investigate basic mechanisms of host immunity, inflammation and wound healing. Most of this work has focused on the immune response to parasitic helminths, organisms that are the likely evolutionary driving force for Type 2 immunity. Helminths are a major cause of disease in impoverished populations (~2-3 billion people) and biomedical research focused on parasitic helminths has been a fertile ground for scientific discovery. In general, helminths such as hookworms pose a formidable challenge to the host immune system regarding their large size, morphological complexity and the host tissue niches they occupy. While infectious larval stages can cause tremendous damage to host tissues as they invade and migrate, the hematophagous nature of adult stages can cause persistent injury during feeding. Surprisingly, most helminth species can survive for years, even decades in their hosts due to a variety of mechanisms including those that suppress and evade host immunity. Given this complex biology, research projects based in the Herbert lab focus on the mechanisms that initiate Type 2 responses, those that drive tissue repair, and requirements for host protective immunity at the sites where worms invade and reside (e.g., skin, respiratory tract, and intestine).

Skin sensory neurons repel schistosomiasis

Schistosoma spp. cause morbidity and mortality in over 250 million people worldwide. The infectious larval stage (cercariae) enters the host through direct skin penetration. This study demonstrates that activation of itch-sensing neurons bearing the Mas-related G protein receptor A3 (A3) selectively suppressed the ability of myeloid antigen presenting cells (APC) to express the pleiotropic cytokine IL-33. Activated A3 neurons stimulated macrophage TNF production, increased IL-17-expressing γδ T cells, increased epidermal thickening, and drove resistance to the human pathogen *Schistosoma mansoni*, partially through the neuropeptide calcitonin gene related peptide (CGRP). Accordingly, cell-intrinsic deletion of IL-33 in myeloid APC basally altered chromatin accessibility at inflammatory cytokine loci, promoted the release of IL-17-inducing cytokines (e.g., IL-1β, IL-6), and induced both tissue pathology (e.g., epidermal thickening, keratinocyte hyperplasia) and host resistance to helminth infection. Our findings suggest a previously undescribed mechanism of cellular cross- talk wherein "itch" neuron activation alters myeloid cytokine expression patterns to reshape skin architecture and drive cutaneous immunity against *Schistosoma mansoni.*

MOSES EGESA

MRC/UVRI and LSHTM Uganda Research Unit

Dr Moses Egesa is a Research Fellow at the MRC/UVRI and LSHTM Uganda Unit. He received his Ph.D. (parasite immunology) from ßMakerere University, Uganda in 2020. Dr Egesa mainly works on the immunology of schistosomiasis. His overarching research goal is to contribute to the development of effective vaccines for schistosomiasis in Africa. He is an EDCTP Career Development Fellow characterizing protein and glycan epitopes recognised following controlled human infection with Schistosoma mansoni in an endemic population. The EDCTP fellowship builds on his contributions as the Project Lead for the Wellcome-funded programme "Establishing a single sex S. mansoni controlled human infection model for Uganda" (PI Prof. Alison Elliott, Co-PI Prof. Meta Roestenberg, and Co-PI Prof. Afzal Siddiqui). He is a co-investigator on various local and international collaborative schistosomiasis research grants with Infectious Diseases Institute (PI Prof. Ponsiano Ocama, NIH funding), Leiden University Medical Center (PI Prof. Meta Roestenberg, HIC-Vac Network funding), and University of York (PI Dr James Hewitson, GCRF funding).

Establishing a single sex *Schistosoma mansoni* controlled human infection model for Uganda

Schistosomiasis is widespread in Uganda affecting 86% of its districts. The control programme in the country is grounded on a single drug, praziquantel. This has had little effect on high reinfection rates and worries of drug resistance are emerging. An effective vaccine could refresh the control efforts, but no vaccine exists for schistosomiasis in the field. More recently, there is growing interest in controlled human infection studies (CHI) to accelerate vaccine development. A CHI for schistosomiasis was developed in the Netherlands. We are developing a CHI for *Schistosoma mansoni* in in endemic Uganda where responses to infection and vaccines differ in target populations. Here, we discuss the progress covering scientific, ethical, and regulatory aspects with a focus on challenges and opportunities in establishing the first single sex *Schistosoma mansoni* controlled human infection model in an endemic setting.

MICHALIS BARKOULAS

Department of Life Sciences, Imperial College, London, United Kingdom

Michalis Barkoulas is a Reader (Associate Professor) at the Department of Life Sciences at Imperial College London. He started with a PhD and postdoctoral research in plant development in the Tsiantis lab at the University of Oxford, investigating how auxin gradients sculpt the dissected leaf shape. In 2009, he became a postdoctoral fellow in the Felix lab at the Ecole Normale Superieure in Paris where he was introduced to C. elegans and addressed how quantitative perturbations of EGF and Notch signalling pathway activity influence cell fate patterns and developmental system robustness. He was then recruited in 2013 to start his own lab as a Lecturer in the Department of Life Sciences at Imperial College London. The Barkoulas lab address fundamental questions on the genotype-to-phenotype relationship and the molecular mechanisms underlying biological robustness. Recent work has focused on the resilience of epidermal development in C. elegans, an essential tissue for growth and biotic/abiotic stress protection, to various perturbations including stochastic, genetic, environmental variation and more recently to infection by natural pathogens.

C. elegans as a tractable host to study natural infections by oomycetes

Manish Grover, Ken Liu, Jonathan Saunders, Florence Drury, Mark Hintze, and Michalis Barkoulas

Oomycetes are a diverse group of eukaryotic organisms that superficially resemble fungi and can cause plant and animal disease. While plant-infecting oomycetes have been widely studied, research on animal-infecting species has been limited by the lack of genetically tractable model hosts. We have recently discovered oomycete species as common natural pathogens of the model nematode *Caenorhabditis elegans*. We have found that *C. elegans* can sense the presence of oomycetes to mount a pathogen-specific protective transcriptional response in the epidermis. This response consists of the induction of a previously uncharacterised gene family of chitinase-like (*chil*) genes, which provides partial resistance to infection by reducing pathogen attachment to the nematode cuticle. I will describe these new pathosystems and discuss recent efforts aimed at dissecting the signalling pathway underlying *chil* gene induction using comprehensive forward genetic screens. Our results illustrate how different tissues in nematodes share signals to orchestrate the host defence response against their natural pathogens.

TANIA ROZARIO

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Tania Rozario got her PhD from the University of Virginia studying embryonic development. During her postdoc she joined Phil Newmark's lab (Morgridge Institute for Research) to study planarian regeneration but pivoted toward their parasitic cousins- tapeworms. Her work (re)established the rat tapeworm, Hymenolepis diminuta, as a non-traditional model to explore the molecular mechanisms that govern how tapeworms grow, regenerate, and reproduce at prolific rates. In 2021, she established her independent lab at the University of Georgia where her work understanding extrinsic and intrinsic signals that regulate tapeworm stem cells continues.

Understanding signals that regulate stem cells and germ cells in the regeneration-competent neck of the rat tapeworm, *Hymenolepis diminuta*.

Tapeworms grow at rates that rival all metazoan tissues, including during embryonic and neoplastic growth. The rat tapeworm, *Hymenolepis diminuta*, produces up to 2,200 proglottids, increasing in length up to 3,400 fold, and weight up to 1.8 million fold within the first 15 days of infection. Tapeworms can also regenerate: they shed large parts of their body, releasing their embryos to continue their life cycle, yet are able to continuously replenish proglottids and maintain an equilibrium length. Such remarkable growth, regeneration, and reproduction are fueled by adult somatic stem cells. Using *H. diminuta* as a laboratory model, we find that regeneration is limited to the tapeworm neck. Using transcriptomic analyses and RNA interference (RNAi) we functionally validated the first molecular regulators of tapeworm stem cells. However, we find no evidence that stem cells are restricted to the regeneration-competent neck. Instead, we find that lethally irradiated tapeworms can be rescued from death when cells from both regeneration-competent and regeneration-incompetent regions are transplanted into the neck, suggesting that stem cells may be maintained throughout the whole body while the extrinsic signals that make up the neck microenvironment are crucial for regeneration. Preliminary results suggest that the head plays at least two important functions: maintaining neck identity and regulating stem cell proliferation. The microenvironment of the neck is highly patterned along the anterior-posterior axis by Wnt signaling components among other factorsthat we are now characterizing. In addition, the neck is also responsible for regulating the germ cell niche though *ebony*-dependent signaling. Together, the head and neck tissue provide a microenvironment that enables region-specific regeneration in this tapeworm.

VICKY HUNT

University of Bath, UK

Vicky Hunt is a Wellcome Trust fellow at the University of Bath. She previously held a JSPS fellowship at the University of Miyazaki, Japan, in Taisei Kikuchi's lab, and an Elizabeth Blackwell Institute early career fellowship and postdoc position at the University of Bristol in Mark Viney's lab. Her research is mainly focused on understanding the genomics and genetics of the gastrointestinal nematode parasite Strongyloides spp. Current projects include investigating the role of small RNAs, gene organisation, regulatory mechanisms and transposable elements in parasitism.

The role of secreted exosome-like vesicles in parasite-host interactions and potential as a biomarker in *Strongyloides* infection

The gastrointestinal parasites *Strongyloides ratti* and *S. venezuelensis* secrete exosome-like vesicles (ELV) during infection. The ELVs, packaged with a cocktail of small RNAs and proteins, are taken up by host epithelial cells *in vitro* where they are predicted to target and manipulate the host environment. We report that the majority of small RNA sequences in ELVs are 'extra small' between 7-15 nucleotides in length ('xsRNAs'). We propose that xsRNAs are a common ELV- associated feature and demonstrate that similar types of xsRNA are also dominant in ELVs secreted by other nematodes. We predict the genes that are targeted and repressed at the posttranscriptional level by xsRNAs. *S. ratti* and *S. venezuelensis* xsRNAs are predicted to target genes in the rat host genome. We hypothesise that xsRNAs, in combination with ELV-bound proteins such as acetylcholinesterase, work together to target the peristalsis response in the host for the benefit of the parasite. Using 11.5 day old mouse embryo intestines we have developed an *ex vivo* model for peristalsis and confirm that secretions from *S. ratti* alter the rate of peristalsis. In addition to understanding the biological role of secreted small RNAs in *Strongyloides*, we also investigate their potential as biomarkers. Small RNAs including xsRNAs can be readily detected in faecal samples and have potential as a novel method for gastrointestinal parasite diagnostics.

POSTER SESSION 1: MONDAY 4 SEPTEMBER 6 - 8 PM

1	**Kelsilandia AGUIAR MARTINS**	Royal Veterinary College - University of London	Genetic analysis of *Schistosoma mansoni* pre-and post-treatment from a high morbidity (FibroSchot) hotspot within Lake Albert, Uganda.
2	**Annia ALBA**	University of Perpignan	How the environment influences the transmission of zoonotic diseases: linking abiotic and biotic factors to the vectorial capacity of an intermediate host snail
3	**Georgia BALDWIN**	University of Manchester	Ym1 as a Regulator of IL-17
4	**Luke BECKER**	Malaghan Institute of Medical Reasearch	Methods for the evaluation of the viability and infectivity potential of reanimated cryopreserved hookworm larvae used for human therapy.
5	**Ilaria BELLINI**	Sapienza University of Rome	Exploring pathogenicity and tumorigenic potential of the nematode *Anisakis* using human intestinal organoids and extracellular vesicles
6	**Sarah BUDDENBORG**	Wellcome Sanger Institute	Optimisation of single cell, nuclei, and spatial RNA-seq in nematodes
7	**Geraldine BUITRAGO**	University of Strathclyde	The parasitic worm product ES-62 protects the osteoimmunology axis in a mouse model of obesity-accelerated ageing.
8	**Alice COSTAIN**	University of Manchester	Tissue damage and microbiota modifications provoke intestinal Type 2 immunity during *Schistosoma mansoni* infection
9	**Padraic FALLON**	Trinity College Dublin	Retinoic acid-related orphan receptor alpha (RORα) is required for generation of Th2 cells during *Nippostrongylus brasiliensis* infection
10	**Peter FISCHER**	Washington University School of Medicine	A *Paragonimus kellicotti* cysteine protease recognized by IgG4 antibodies of infected humans is found in extracellular vesicles produced by the parasites in vitro and in the lung cysts
11	**Sandra GAVA**	Oswaldo Cruz Foundation, Fiocruz	The effect of Aspartyl Proteases Cathepsin D-like knockdown in *Schistosoma mansoni* schistosomula and adult worms
12	**Richard GRENCIS**	University of Manchester	Neutrophil modulation of immunity during chronic intestinal helminth infection: Haptoglobin regulation of B cell responses
13	**Amber HADERMANN**	University of Antwerp	Onchocerciasis-associated epilepsy: the pathophysiological mechanism
14	**Kelly HAYES**	University of Manchester	Genetic manipulation of the parasitic nematode *Trichuris muris*
15	**Malcolm KENNEDY**	University of Glasgow	What mediators might the p43 immunomodulatory proteins of *Trichuris spp.* deliver to the infection site?
16	**Xeusong LI**	Justus-Liebig-University, Giessen	Identification of GPCR-neuropeptide interactions and their functional analyses in *Schistosoma mansoni*
17	**Maria Cristina LOADER**	St George's University London	Frequency of soil-transmitted infections in tuberculosis patients in the Peruvian Amazonian city of Iquitos and impact on markers of lung tissue destruction in TB-helminth co-infection

POSTER SESSION 2: WEDNESDAY 6 SEPTEMBER 6:00 – 8:00 PM

18	**Kate MACLEAN**	Malaghan Institute of Medical Research	Local vs systemic cytokine responses to human hookworm infection in healthy adults
19	**Marina de MORAES MOURÃO**	FIOCRUZ- Fundação Oswaldo Cruz	Phenotypic screening of compounds identified by molecular docking targeting *Schistosoma mansoni* protein kinases
20	**Javier MORA**	University of Costa Rica	The role of *Toxocara canis* third-stage larvae antigens in immune cell activation and induction of trained immunity
21	**Max Frederik MÖSCHEID**	Justus Liebig University Giessen	Chromosomal integration of a reporter gene by RNA-guided Cas-enzymes into a predicted genomic safe-harbor site of *Schistosoma mansoni*
22	**Pia Franziska Marie NAUJACK**	Justus Liebig University Giessen	Insights into the molecular IgE-IPSE/alpha-1 interaction responsible for basophil activation
23	**Marina PAPAIAKOVOU**	University of Cambridge	Geographic-specific variation in genomic diagnostics targets of soil-transmitted helminths
24	**Daniel PARSONS**	Kingston University, London	Molecular epidemiology and evolution of antigen-coding genes from the multi-host parasite *Schistosoma japonicum*
25	**Laudine PETRALIA**	New England Biolabs, Inc.	Identification of species-specific glycan antigens of *Schistosoma haematobium*
26	**Rajalah Pradhu PRINCE**	Centre for Biotechnology Anna University, Chennai	Structural characterization and protective response of host non-homologous epitopes of *Wuchereria bancrofti* Thioredoxin
27	**Ella REED**	Cardiff University	A possible role for helminth-derived prostaglandin in regulating intestinal permeability and colitis-associated colorectal cancer development
28	**Martina SOMBETZKI**	Rostock University Medical Center	Effects of 24-nor-ursodeoxycholic and ursodeoxycholic acid on mitochondrial dynamics in the liver of *Schistosoma mansoni* infected mice
29	**Camila SOUZA**	National Institute of Allergy and Infectious Diseases	Heterogeneity of immune response during schistosomiasis in inbred mouse strains
30	**Thomas SPANGENBERG**	Merck Global Health Institute, Eysins	Identification of small molecules interacting with a microRNA present in extracellular vesicles of Schistosomes to study the host–parasite interaction
31	**Shannan SUMMERS**	London School of Hygiene and Tropical Medicine	Investigating the genetic diversity of the *Schistosoma mansoni* Transient Receptor Potential Melastatin ($SmTRPM_{PZQ}$) channel in response to praziquantel treatment in natural Ugandan *S. mansoni* populations
32	**Franziska WINKELMANN**	Rostock University Medical Center	Utilisation of 'Omics' to unmask the interactions of adult male and female *Schistosoma mansoni* with their host
33	**Taoxun ZHOU**	Huazhong Agricultural University	Macrophages release extracellular traps against *Strongyloides stercoralis* larvae via nuclear envelope budding
34	**Isani Hubi ZULFA**	SRUC, University of Edinburgh	The impact of co-infection on host resistance and tolerance differs in mice of different genotypes
35	**Laurens ZWANENBURG**	Ghent University	Plant-based production of protective vaccine antigen against the bovine abomasal parasite *Ostertagia ostertagi*

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Sandra Gava	Oswaldo Cruz Foundation, Fiocruz
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