

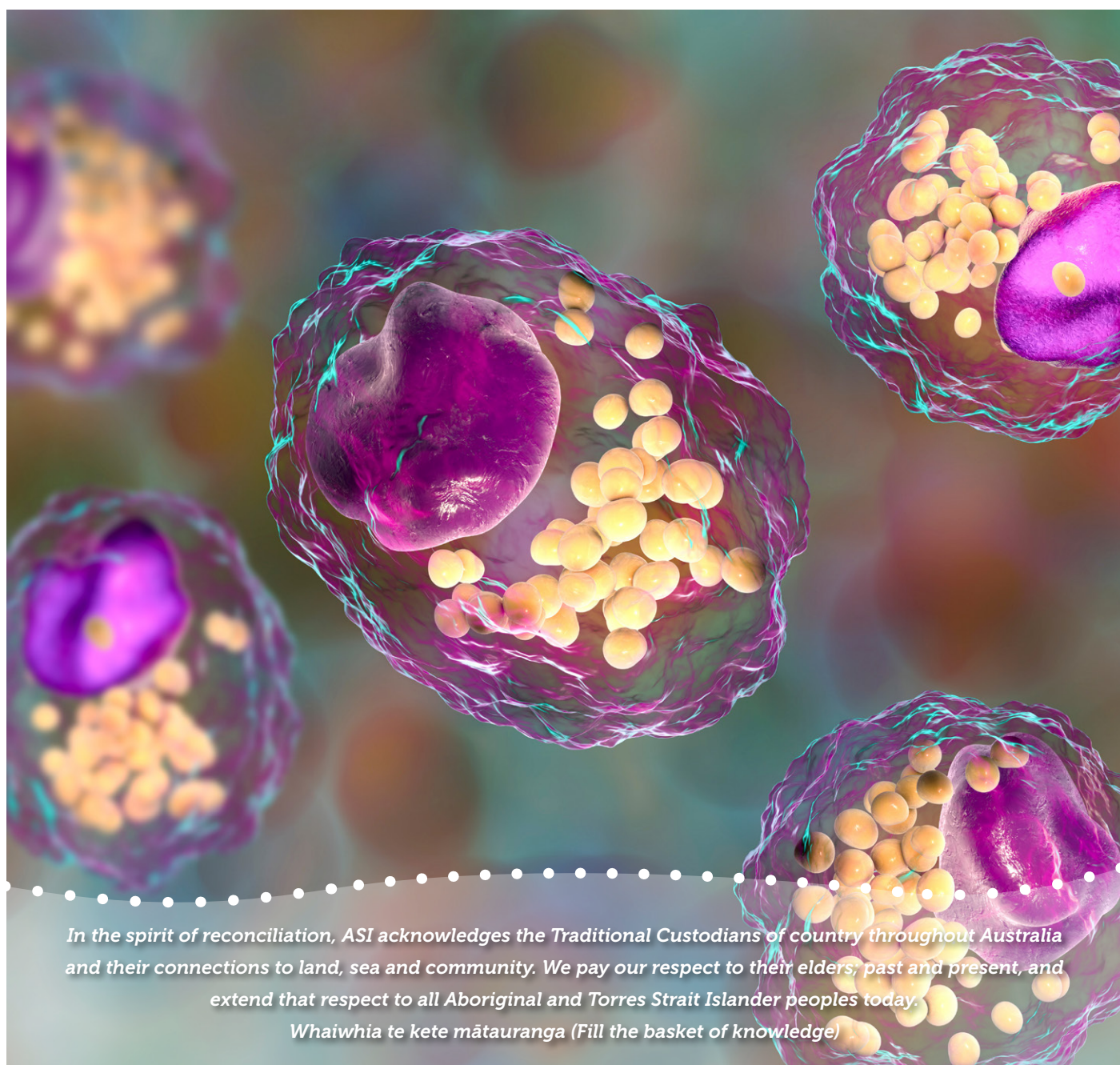
# ASINews

**JUNE  
2023**

ABBVIE  
NEW HORIZONS  
AWARD  
**PAGE 3**

CAREER  
ADVANCEMENT  
AWARDS  
**PAGE 5**

CAREER  
AWARDS  
**PAGE 9**



*In the spirit of reconciliation, ASI acknowledges the Traditional Custodians of country throughout Australia and their connections to land, sea and community. We pay our respect to their elders, past and present, and extend that respect to all Aboriginal and Torres Strait Islander peoples today.*

*Whaiwhia te kete mātauranga (Fill the basket of knowledge)*

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# AbbVie New Horizons Award

## KATE SCHRODER

Institute for Molecular Bioscience,  
The University of Queensland

Inflammation drives pathology in many devastating human diseases for which patients have no disease-modifying drugs. Inflammasomes are signalling platforms that drive pathological inflammation in many human diseases (e.g., cancers, auto-inflammatory, metabolic, neurodegenerative diseases). For 20 years, Professor Kate Schroder has studied the good,

bad and ugly of our immune system – how inflammasome signalling protects us from infection, but also causes several serious human diseases. Kate is an Institute for Molecular Biosciences Laboratory Head at the University of Queensland.

Kate's discovery research has revealed how inflammasomes allow our body to mount an

inflammatory response during infection and disease. Specifically, her lab discovered that inflammasomes recruit caspase-1 monomers to facilitate caspase-1 dimerization and self-cleavage, and thereby unleash inflammasome protease activity, so that caspase-1 can then cleave and activate substrates such as interleukin (IL)-1b and Gasdermin-D. Mature IL-1b then translocates from the cytosol to the extracellular space. Kate's lab further discovered how IL-1b traffics to the plasma membrane and uses Gasdermin-D pores to exit the cell. Gasdermin-D pores also induce membrane damage, and this was presumed to commit a cell to die. The Schroder lab made the unexpected discovery that cells can survive inflammasome signalling and that the burden of Gasdermin-D pores must pass a critical threshold to induce cell lysis. In some settings of strong Gasdermin-D activation and cell lysis, Kate's lab discovered that the nuclear envelope is compromised, triggering the release of web-like chromatin structures that trap bacteria.

Kate is passionate about ensuring her research findings are applied clinically, to ensure patients suffering from inflammation-related diseases can access disease-modifying therapies. Kate and her collaborators developed new anti-inflammatory drug candidates that formed the



basis for a UQ biotech start-up company, Inflazome. In 2020, the pharmaceutical giant Roche acquired Inflazome for AUD\$620 million plus milestones, in Australia's largest deal for academic intellectual property. Two drug candidates are now poised for Phase 2 human clinical trials as novel anti-inflammatory therapeutics.

Kate regularly works with industry to guide commercial programs, serving on the Scientific Advisory Boards for biotech and pharmaceutical companies (e.g. Inflazome, Quench Bio, Novartis). Kate also serves on the Board of Directors of the South Australian Health and Medical Research Institute.

Kate's lab seeks to understand how inflammasomes signal to provide host defence against infection. Her lab also elucidates how this pathway drives immune-driven diseases, and develops inflammasome inhibitors targeting single or multiple inflammasomes as new anti-inflammatory therapies. Kate is delighted to have won the 2022 ASI AbbVie New Horizons Research Award, and gives heartfelt thanks to both ASI and Abbvie for supporting her research. Kate's lab will use this award to perform transcriptomic studies to survey

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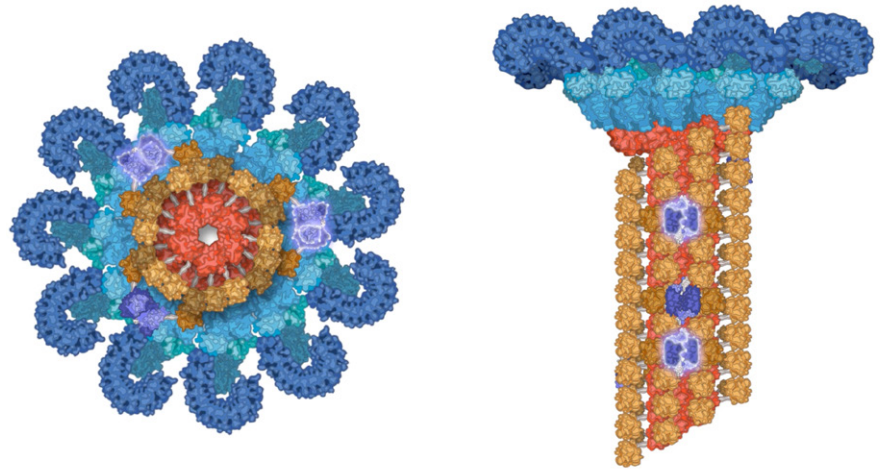
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liver inflammatory and pro-fibrotic pathways in murine models of liver disease, including those where inflammasomes are targeted by genetic ablation or pharmacological inhibition. Such analyses will leverage her lab's current *in vivo* modelling studies that assess how inflammasomes orchestrate liver inflammation and fibrosis – the Abbvie award will facilitate new experiments to elucidate the cell and transcriptomic networks underpinning liver inflammation, fibrosis, dysfunction and disease. Kate anticipates that tissue inflammatory programs revealed by such analyses will be broadly relevant to other disease areas where inflammasomes drive pathology (e.g. gastrointestinal, dermatological, rheumatological diseases), and will provide important proof-of-concept for the commercialisation of novel inflammasomes inhibitors. The significance of this research program lies in its exciting potential for delivering new therapeutics for many human diseases with urgent unmet need, such as chronic liver disease and hereditary auto-inflammatory diseases.

For more details of our discovery of mechanisms of caspase-1 activation and deactivation watch: <https://youtu.be/FNFZ9F1eB4> 🌟

*Caspase-1 is recruited to inflammasome signalling complexes for proximity-induced dimerization and autoprocessing, generating the fully active caspase-1 species (p33/p10)*



K. Schroder, © 2018 The University of Queensland

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# Career Advancement Awards

## BENTOTAGE FERNANDO

School of Biotechnology  
and Biomolecular Sciences  
(BABS) at UNSW Sydney



I am a clinically trained  
biomedical researcher  
who currently works as

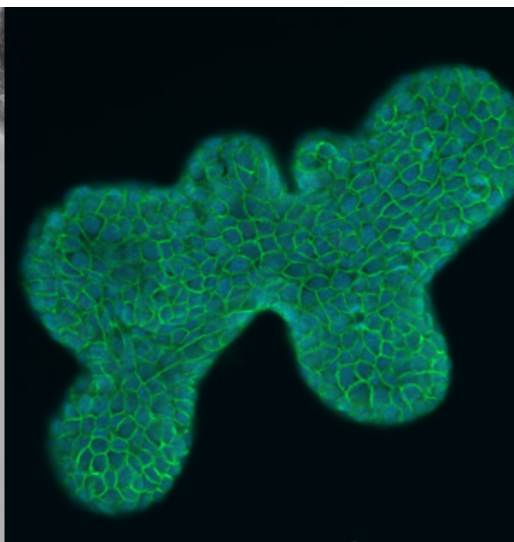
research assistant within the School of Biotechnology and Biomolecular Sciences (BABS) at UNSW Sydney. My clinical education (MBBS) and clinical internship in infectious diseases in Sri Lanka ignited a keen interest in the field of immunology research. Therefore, I pursued and completed an MPhil in immunology exploring the pathogenesis of dengue infection. I then completed my PhD at UNSW Sydney, where I studied the biochemical regulation of immune responses in primary human immune cells. More precisely, I investigated how the biochemical pathways of cellular energy metabolism and heme-iron metabolism, affect immune responses via controlling the key immunoregulatory enzyme indoleamine 2,3-dioxygenase 1 (IDO1). Understanding how biochemical changes impact on immune responses sparked my interest in exploring metabolic changes in cells leading to

gastric cancer (GC), given that there is no better example than GC to demonstrate that inflammation is a hallmark of cancer with the proven Correa's cascade (i.e., progression from gastric precancerous lesions including atrophic gastritis, intestinal metaplasia and dysplasia, to GC) initiating with inflammation due to microbial insult (e.g., *Helicobacter pylori* infection).

As a research assistant in the Castaño-Rodríguez/ Kaakoush Laboratory at UNSW, I am exploring the role of crosstalk between lactic acid metabolism and autophagy in the pathogenesis of GC. For this, I established a gastric organoid model from human gastric biopsies obtained from healthy individuals as well as atrophic gastritis and intestinal metaplasia patients. To better understand the kinetics of autophagy and lactic acid metabolism, these tissues will be examined with the help of

correlative light and electron microscopy (CLEM).

I am honoured to be a recipient of ASI Career advancement award as this provided me the opportunity to explore this high-impact area of medical research and simultaneously develop a completely novel set of cutting-edge skills in correlative light and electron microscopy and specialized cell culture techniques such as organoid culture. Therefore, I believe that this opportunity improved my skillset, confidence and potential as a scientist. Importantly, in the long-term, this study will provide a solid base to future studies on robust and cost-effective early diagnostic, preventive and therapeutic strategies that target immune and metabolic pathways to improve the clinical outcome of patients with GC. Therefore, I would like to thank ASI for supporting and paving the path to achieve these goals. 🌟





# Career Advancement Awards



## JOSH (HYUN JAE) LEE

Peter Doherty Institute  
for Infection and  
Immunity (PDI),  
University of Melbourne

I am Josh, an Early Career researcher at the Peter Doherty Institute for Infection and Immunity (PDI), University of Melbourne under the mentorship of Associate Professor Ashraf Haque. My research is focussed on understanding how CD4+ T cells and B cells respond to malaria and exploring how single-cell technologies can illuminate complex immunological questions.

I completed my PhD at the Institute for Molecular Bioscience (IMB), University of Queensland where I explored why some people get a severe form of malaria, under the guidance of Prof. Lachlan Coin. This led to ground-breaking transcriptome profiling of both the human host and Plasmodium parasites. After completing my PhD, I began

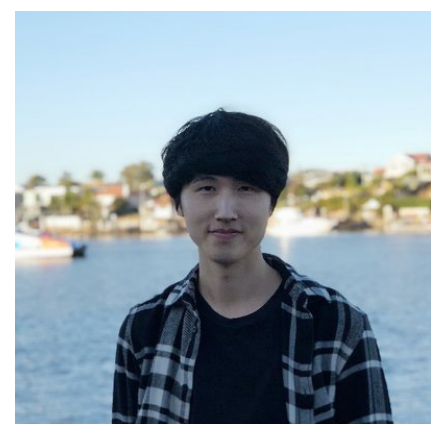
my postdoctoral research career at QIMR Berghofer Medical Research Institute, working across two research teams to understand tumour microenvironment in mesothelioma (led by Dr Nic Waddell) and T cell biology (led by Assoc. Prof. Ash Haque), using single-cell technologies.

In 2020, I joined PDI as a postdoctoral researcher, focusing on the dynamics of memory recall during re-infection with malaria parasites. Malaria is a major global health burden given that malaria mortality and morbidity is highest among children. In malaria endemic regions, children often experience multiple infections over a short period of time, with their CD4+ T cell responses becoming more regulatory with age and exposure. This suggested that repeated infections qualitatively change CD4+ T cells but to what extent remained unclear. More generally, systematic analysis of memory recall has not been done. Using scRNA-seq, VDJ-seq, and spatial transcriptomics, we have shown that CD4+ T cells exhibit a variety of recall dynamics upon re-infection. Importantly, we identified that Th1 recall

response is characterised by up-regulation of a select few genes, and GC Tfh cells is refractory to change upon re-infection. My other works involve understanding the human tissue-resident memory T cells and studying T cells during breast cancer treatment, using single-cell genomics approaches. I am also actively involved in guiding other researchers to incorporate and analyse single-cell datasets in their own projects.

I am grateful to be awarded the Career Advancement Awards to support my trip to the Cold Spring Harbour Laboratory meeting on Gene Expression & Signalling in the Immune System. This was my first international conference after almost three years of virtual meetings due to the pandemic. It was exhilarating to meet the experts in the field in person, and equally, to interact with fellow early career researchers from around the world.

During the five-day conference, we engaged in meaningful discussions about the changes and signalling involved in innate and adaptive immune responses in various contexts, such as allergies and COVID-19. These scientific discussions extended



to my work on memory recall during malaria infection, offering me fresh perspectives on hitherto unexplored angles of my project.

Importantly, this meeting was also a great opportunity to connect with fellow computational biologists who use various -omics approaches, including single-cell genomics, to answer intriguing immunological and clinical questions. Gaining insight into their research and discussing potential career paths was truly enlightening.

Again, I would like to thank ASI for supporting my research through the Career Advancement Award. 🌟

# Career Advancement Awards

## MADELEINE WEMYSS

Centre for Innate Immunity and Infectious Diseases,  
Hudson Institute of Medical Research, & Department  
of Microbiology, Monash University

My name is Madeleine (Maddie) Wemyss, and I was honoured to be awarded an ASI Career Advancement Award (Postgraduate) at the end of 2022.

I am a final year PhD student supervised by A/Prof Jaclyn Pearson, within the Centre for Innate Immunity and Infectious Diseases (Ciiid) at Hudson Institute of Medical Research, and am enrolled through the Department of Microbiology at Monash University. I completed my undergraduate studies at Monash University in 2017, graduating with a Bachelor of Science and Bachelor of Biomedical Science, before joining A/Prof Pearson's lab to complete my Honours degree in 2018. Joining the Pearson lab for my Honours year gave me my first real taste of what it was like to be a biomedical researcher, and I quickly became fascinated with the complex world of innate immunity and host-pathogen interactions.

Our research explores the key mechanisms used by pathogenic bacteria to promote virulence and evade host immune responses during infection, with a particular focus on how these bacteria interact with programmed cell death pathways within the host cell. Bacterial gut pathogens, including Non-Typhoidal serovars of *Salmonella enterica*, account for a large proportion of the global foodborne disease burden, causing symptoms ranging from mild gastroenteritis to severe systemic infection. Worryingly,

many of the *Salmonella* isolates actively circulating in the Australian community are rapidly acquiring antimicrobial resistance, making them more and more difficult to treat over time. As such, improving our understanding of bacterial pathogenesis could be crucial to our ability to combat these infections in the future.

As part of my PhD project, I've characterised a novel interaction between *S. enterica* serovar Typhimurium and human macrophage cells – whereby *S. Typhimurium* uses Type III Secretion System effector proteins to actively target cellular inhibitors of apoptosis, cIAP1 and cIAP2, thus suppressing inflammatory signalling and promoting cell death. Ordinarily, programmed cell death responses (such as apoptosis, necroptosis and pyroptosis) are important mechanisms of host immune defence against infection in the gut, interrupting replication and promoting bacterial clearance. Both cIAP1 and 2 act as key regulators of TNF-induced cell death, directing RIPK1 signalling towards pro-inflammatory NF- $\kappa$ B signalling and away from pro-apoptotic caspase-8 activation. While *Salmonella* is known to use a varied cohort of effector proteins to modify the host cell environment and mediate virulence, these effectors have not previously been shown to target cIAP1 or 2. We speculate that by activating, rather than preventing, cell death mechanisms at later stages of infection in

macrophages, these *Salmonella* effector proteins could promote re-uptake and spread of the bacteria into neighbouring cells or systemic sites, leading to more severe disease.

Although this finding provided a very exciting basis for my project, similarly to many others, my PhD research has been continually disrupted over the past 3 years – by both the ongoing COVID-19 pandemic and emerging medical issues within my immediate family. The impacts of these delays, including several months working solely from home as well as frequent periods of restricted laboratory access, unfortunately required me to

extend my course duration multiple times, and eventually exhausted my stipend funding. Thankfully, with the receipt of this award, I have been able to continue my research into 2023, performing several collaborative experiments that were key to my remaining results chapters. This will be crucially beneficial for both the quality of my final thesis, and for the readiness of this work for publication – improving my employability for future positions. I am extremely grateful to ASI for enabling me the means to complete my PhD to the best of my ability, and look forward to sharing my work with you all at future ASI events. 🌟

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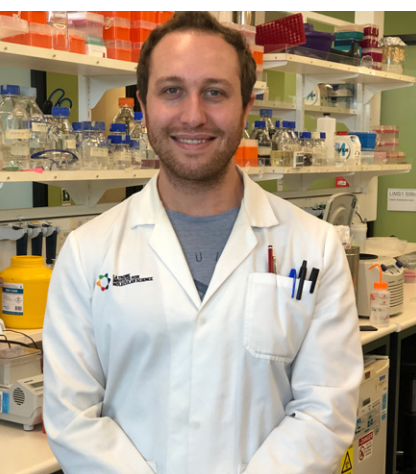
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# Career Advancement Awards

## MICHAEL HARRIS

Previously La Trobe University, now at Peter MacCallum Cancer Centre



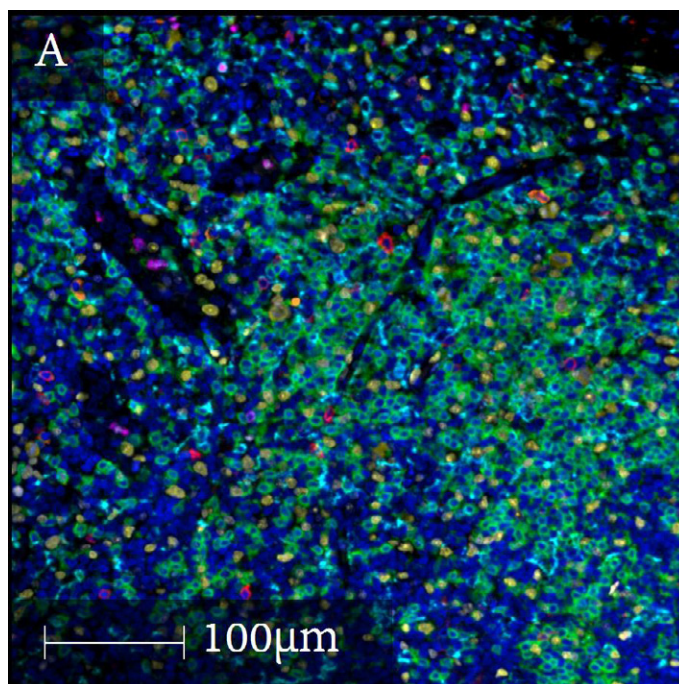
I recently completed my PhD at the La Trobe Institute for Molecular Science, under the supervision of Associate Professor Christine Hawkins. I joined the Hawkins laboratory in the second year of my undergraduate degree in 2016 through a summer research project scheme, which gives undergraduate students interested in pursuing research as a career a chance to work in a real lab setting. This was a fantastic opportunity for me to gain some invaluable insight into cancer research and really sparked my interest in having a career in science. I stayed on with the Hawkins lab as a research volunteer in the third year of my undergrad, where some extremely patient lab members supervised me while I struggled to perform very basic experiments.

The summer research project turned into an honours year which turned into doing a PhD.

The main focus of my project was looking at new treatments for metastatic osteosarcoma, which is the most common form of primary bone cancer. This type of cancer primarily afflicts children or adolescents and survival rates for osteosarcoma haven't increased in about fifty years. One of the more promising drug classes I worked with are called Smac mimetics, which cooperate with TNF produced by tumour infiltrating immune cells, to induce cell death. A lot of my experiments were centred around identifying which immune cells in my animal models were producing TNF in the tumours, under what conditions and how much.

I am extremely grateful to have been awarded a Career Advancement Award from ASI. The funding I received from ASI has allowed me to collaborate with Associate Professor Sarah Ellis at the Oliva Newton John Cancer Research Institute to optimise multi-spectral imaging on human tissue samples. This technique will help me determine what proportion of osteosarcoma patients are likely to respond to Smac mimetic treatment in the clinic and really boost the impact of my final paper from my PhD.

Since finishing my PhD I joined the Translational Breast Cancer Genomics and Therapeutics Lab at the Peter MacCallum Cancer Centre, headed by Professor Sherene Loi, as a postdoctoral researcher where I'll be exploring new strategies to treat breast cancer. 🌟



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# Carer Awards

**CARLY  
WHYTE**  
University of South  
Australia

I am an early career research fellow at the Centre for Cancer Biology in South Australia, where I work on unconventional immune responses to tumours. I am particularly interested in how eosinophils recognise and respond to tumours, and how they may influence outcomes of immunotherapy in patients. I completed my PhD in 2018 at the University of Adelaide, where I studied how migration of dendritic cells and CD8 T cells is controlled through atypical chemokine receptors. Subsequently, I did a postdoc at the Babraham Institute in Cambridge, UK, where I worked on understanding how diverse inputs of IL-2 influence immune outcomes in different ways. I am grateful that I was able to give a presentation on this work on IL-2 at ASI 2022, which was the first time I'd been able to present this study since returning from overseas and having children.

At the time of the meeting, I was on maternity leave after the birth of my second son. As he was only 4 months old, it was essential that he come with me to the conference. Realistically, I knew it would be very difficult to

fully engage with the conference while caring for a baby. Thanks to the generous support of ASI, I was able to have my mum travel with me to the conference so she could look after my son between feeds. This confidence that he was well-looked after allowed me to fully participate with the conference and all of the wonderful science being discussed. This award also supported childcare costs for my two year-old son at home.

The conference was fantastic, and was a great opportunity to discuss the latest high quality immunology research in Australia and overseas. I was able to forge new connections that will hopefully continue into fruitful collaborations, and reconnected with former colleagues in a way you only really can in person. The calibre of speakers was fantastic and the Lafferty Debate was a highlight as always.

The support that ASI offers really makes a tangible difference to its members, particularly in critical moments such as after becoming a parent. Thanks again to ASI, I'm very grateful for your generosity and support. 🌟

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# Carer Awards

**NATALIA G SAMPAIO**

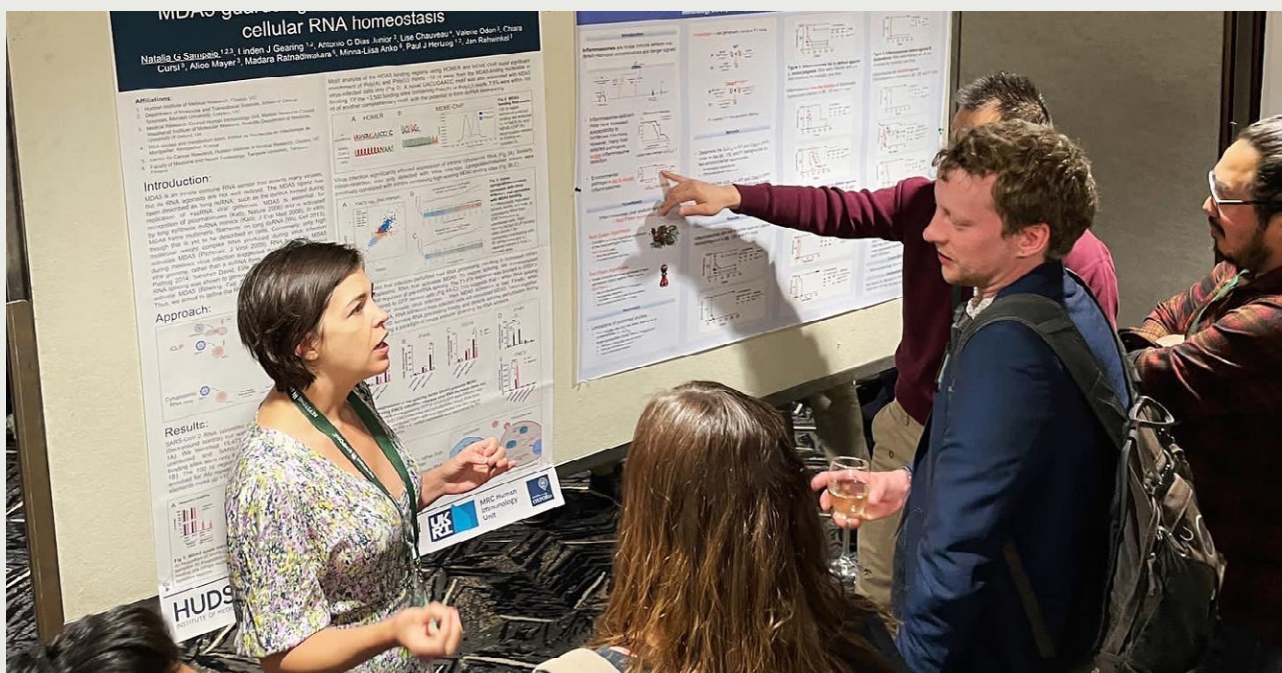
Hudson Institute in  
Melbourne

I am a postdoctoral researcher at the Centre for Innate Immunity and Infectious Diseases at the Hudson Institute in Melbourne, working in the Regulation of Interferon and Innate Signaling group led by Prof. Paul Hertzog. I have been studying innate immunity for most of my career, with a recent focus on nucleic acid receptors and viral sensing. Since joining Prof. Hertzog's group in March 2021, I have been leading new research on RNA-binding receptors of the innate immune system, studying how they are activated during virus infection and with novel RNA therapies. I was recently awarded an mRNA Victoria Activation Program Grant to study this area.

I was honoured to receive the Women's Initiative Travel Award from the Australian Society for Immunology to attend the Keystone Innate Immunity Symposium in Salt Lake City, Utah, USA. This conference coincided with my maternity leave for my second child, who was just five months old at the time and exclusively breastfeeding. To attend the conference, I had to bring my son along and have an additional carer to look after him. Luckily, my husband was able to accompany me to the conference and take care of our son while I attended the sessions.

At the Keystone Symposium, I was selected for an oral presentation on my new unpublished work on the RNA receptor MDA5, a critical sensor of virus infection. This was a significant achievement, given that this conference is one of the best in the field, and attended by many leaders of innate immunity. Presenting my work there provided me with an ideal opportunity to interact with other relevant scientists, and I received positive feedback on my presentation, including offers for future collaborations. I have already set up two new collaborations with groups in the USA as a result. Furthermore, the conference was a perfect place to discover new research areas (bacterial innate immunity, wow!) and to hear exciting new unpublished work from others.

While attending the conference came with its challenges, including traveling with a baby, dealing with time zone changes, and arranging baby feeding between sessions, the benefits of attending were worth it. I am grateful for the ASI, the Hudson Institute, and my family, who provided the additional childcare support I needed to attend the conference.e. 🌟





# Carer Awards

## STEPHANIE TREND

Perron Institute for Neurological and Translational Science and  
Telethon Kids Institute

I am the MSWA Research Fellow in the Demyelinating Disease Group at the Perron Institute for Neurological and Translational Science, and an Honorary Research Associate in the Inflammation Team at Telethon Kids Institute (where I am based), in Perth.

Our collaborative team investigates the immunology behind Multiple Sclerosis (MS) and the role of ultraviolet light in suppressing development of MS. I joined the lab of Prue Hart in 2016 when the team were investigating whether narrowband UVB phototherapy could prevent MS in people with a pre-MS condition known as clinically isolated syndrome, who are at high risk of conversion to MS. Those who attended the ASI Annual Scientific Meeting in Melbourne will recall the fantastic Burnet oration given by Prue Hart, where she



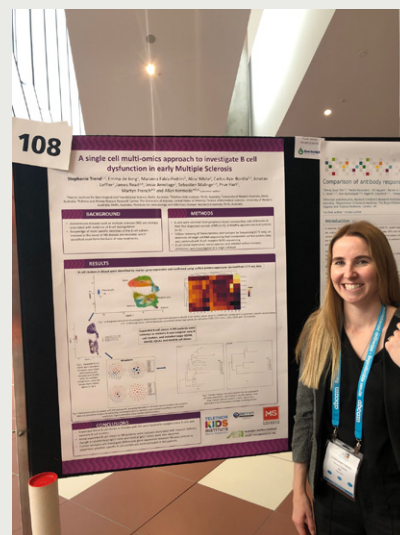
described the significant increases in naive B cell frequencies, decreased frequencies of memory B cells and decreased TNF production by B cells following stimulation with a TLR7 agonist following the phototherapy intervention.

This work developed my interest in immune regulation and B cells, as I had completed my PhD at UWA investigating host defence proteins and leukocyte content of preterm mothers' breast milk and had primarily worked on bacterial infections prior to this.

On the personal side of things, my first child was born in 2017 and I returned to work in 2018 part-time after 6 months to continue my role in the lab on this project. Continuing in my existing role within our collaborative team, I joined the Perron Institute in 2019 while continuing to work in the laboratory at Telethon Kids Institute, and received my first project grant as CIA from MS Australia to begin work in 2020. However, life had other ideas and first the pandemic, and then becoming a parent to twins, turned my life completely upside down and delayed the project until I returned to work part time in late 2021 after 10 months of leave.

Despite the enormous impact this had on my health and family, my passion for science did not fade and I was fortunate to be awarded an incubator grant from MS Australia to develop a new collaboration with Dr Luke Garratt at Telethon Kids Institute and A/Prof Anne Bruestle at the John Curtin School of Medical Research at ANU investigating the role of neutrophils in MS.

In addition, I was able to begin the major work of my project grant and this has led to some fantastic opportunities to move into research that investigates B cells at the molecular level, which has been really exciting and we have will soon be investigating B cell trajectories at the molecular level in people with



pre-MS as a result of this work. With the pandemic and WA's closed borders, and twin infants who were breastfeeding, I had not had the opportunity to leave Perth since 2019 and have found it challenging to join in networking events with my part-time workload.

Attending the ASI annual scientific meeting was a fantastic opportunity for me to focus on my work and to develop and maintain important connections. Some of my collaborators and I were able to meet in person for the first time and discuss our future plans, and it was great to see old friends and to take part in the celebration of Prue Hart's career as the Burnet Orator.

Despite all of the benefits of attending the meeting, there were personal challenges in order to get there: as I normally work part-time, attending the meeting meant that extra child care was needed for my three children, so the generous carer's grant from ASI made this a financially viable prospect for my family.

I sincerely thank the ASI and the review panel for providing me with this opportunity. 🌟

# The IUIS Corner

Joanne Reed | IUIS Coordinator



## The 18th IUIS International Congress of Immunology

ASI has awarded special travel grants to four early career members to attend the IUIS International Congress of Immunology in Cape Town, South Africa on 27th November 2023. The awardees are:

*Kerrie Foyle* (University of Adelaide)

*Raissa Fonseca* (Peter Doherty Institute)

*Christopher Jara* (Garvan Institute)

*Xiaoxia Jia* (Peter Doherty Institute)

*Congratulations to the awardees!*

ASI is providing an additional three travel awards for early career immunologists from low to middle income countries to attend the 2023 IUIS International Congress of Immunology.

## International Day of Immunology 2023 – Public Health in Immunology

IUIS celebrated the 2023 Day of Immunology on 29 April with a live panel discussion on improving vaccines for respiratory infections with Dr. Cristina Cassetti, Dr. Eric Jongert, Dr. Annateresa Palamara, Dr. Bali Pulendran, Dr. Rino Rappuoli, Dr. Gianluca Rotta and Dr. Linqi Zhang.

Joanne Reed ✨

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# Sustaining Member Publications



**1. BD FACSymphony™ A5 Cell Analyser and  
BD Rhapsody Single-Cell Analysis System**

Mair F et al. (2022) Extricating human tumor immune alterations from tissue inflammation.

*Nature.*

<https://doi.org/10.1038/s41586-022-04718-w>

**2. BD LSRFortessa™ X-20 Cell Analyser and  
BD Rhapsody Single-Cell Analysis System**

Son E et al (2023) Screening self-peptides for recognition by mouse alloreactive CD8+ T cells using direct ex vivo multimer staining

STAR Protocols

<https://doi.org/10.1016/j.xpro.2022.101943>

**3. BD FACSymphony™ A5 Cell Analyser**

Pelham S et al (2022) STAT5B restrains human B-cell differentiation to maintain humoral immune homeostasis

Journal of Allergy and Clinical Immunology

<https://doi.org/10.1016/j.jaci.2022.04.011>



**1. goGermline™ (https://www.ozgene.com/  
gogermine-knockout-and-knock-in-mice/)**

Pegoretti et al., (2023). Sequential treatment with a TNFR2 agonist and a TNFR1 antagonist improves the outcomes in a humanised mouse model for MS. *J Neuroinflammation.*

doi: 10.1186/s12974-023-02785-y.

**2. Knockout mouse model (https://www.ozgene.com/  
services/knockout-mice/)**

Kim et al., (2023). Gulp1 deficiency augments bone mass in male mice by affecting osteoclasts due to elevated 17  $\beta$ -estradiol levels. *J Cell Physiol.*

doi: 10.1002/jcp.30987.

**3. Knock-in mouse model (https://www.ozgene.com/  
services/knock-in-mice/)**

Gleneadie et al., (2023). Sequential treatment with a TNFR2 agonist and a TNFR1 antagonist improves the outcomes in a humanised mouse model for MS. *J Neuroinflammation.*

doi: 10.1186/s12974-023-02785-y.

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# Australian and New Zealand SOCIETY FOR IMMUNOLOGY INC.

## Australian and New Zealand Society for Immunology Inc.

The aim of the ASI is to encourage and support the discipline of immunology in the Australia and New Zealand region.

The Australian and New Zealand Society for Immunology Incorporated (ASI) was created by the amalgamation in 1991 of the Australian Society for Immunology, formed in 1970, and the New Zealand Society for Immunology, formed in 1975. The aim of the Society is to encourage and support the discipline of immunology in the Australasian region.

It is a broadly based Society, embracing clinical and experimental, cellular and molecular immunology in humans and animals. The ASI provides a network for the exchange of information and for collaboration within Australia, New Zealand and overseas. ASI members have been prominent in advancing biological and medical research worldwide. We seek to encourage the study of immunology in Australia and New Zealand and are active in introducing young scientists to the discipline.

The ASI membership directory, listing all current members of the Society is available at <http://www.immunology.org.au/asi-membership-directory/>

## REMEMBER: Renew your ASI membership

To renew your membership, click [here](#). Please note that if you have not held a membership within the last 2 years, you will be prompted to provide 2 Nominators willing to support your application.

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