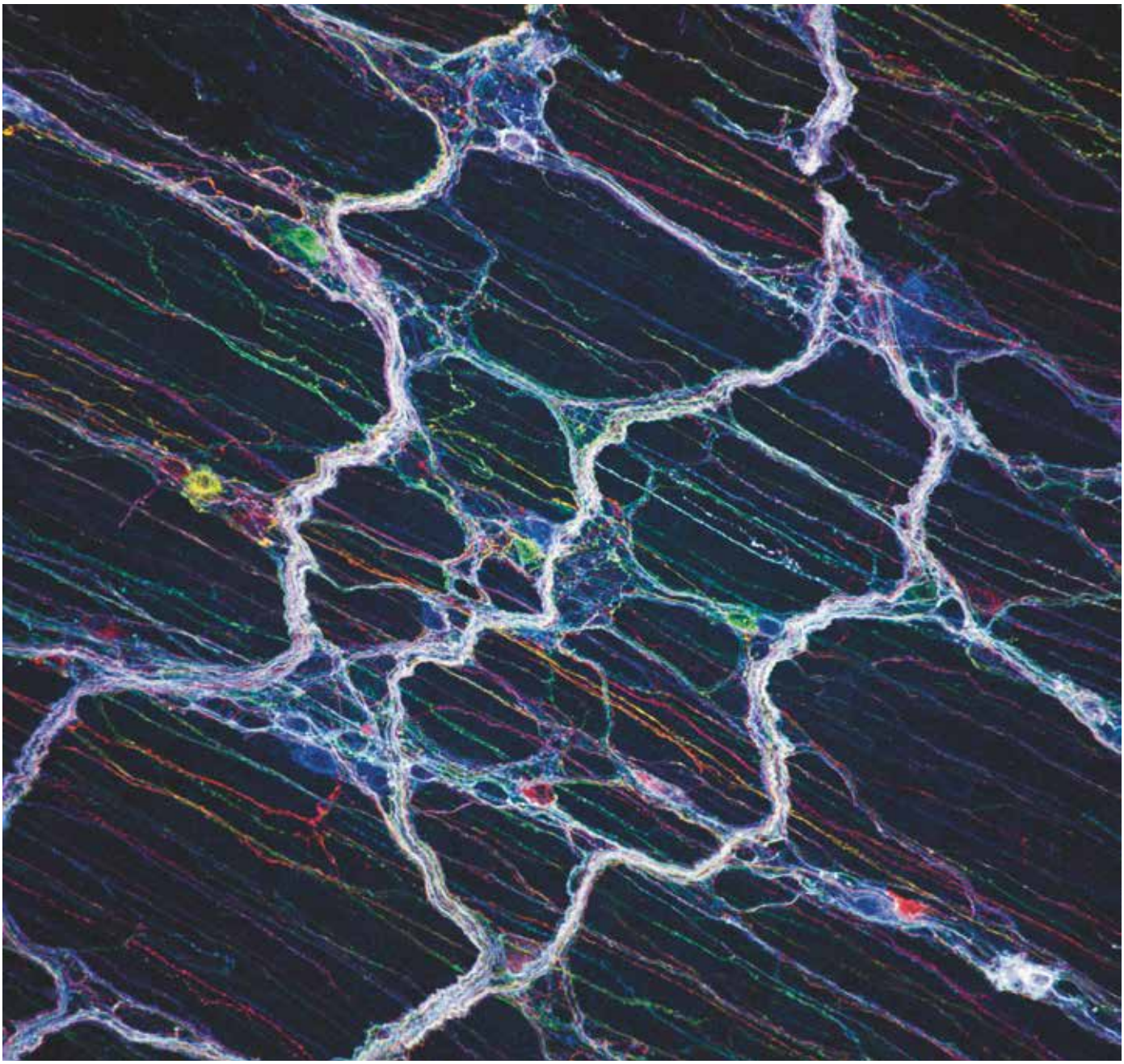
2026

BSCB Magazine

BRITISH SOCIETY FOR CELL BIOLOGY



Dynamic Cell VI

A JOINT BIOCHEMICAL SOCIETY
AND BSCB SCIENTIFIC MEETING

21-24 April 2026
Hilton, Reading, UK

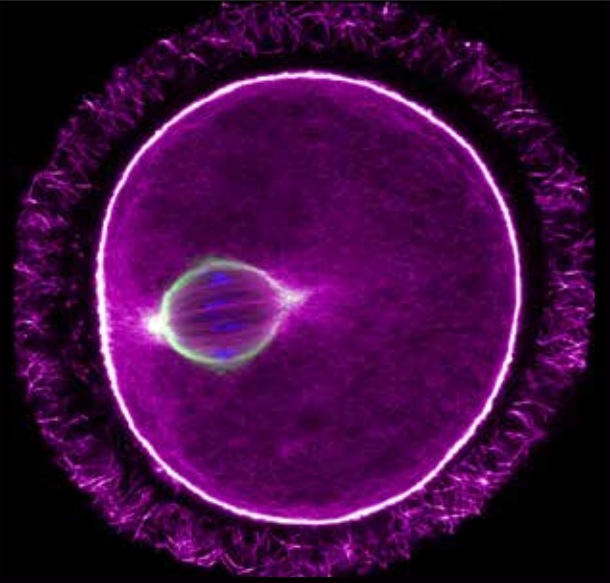


Photo credit: Sam Dunkley

Topics:

- **Cell cycle, cell division, and proliferation**
- **Cytoskeletal structure and regulation/Cytoskeletal dynamics/Cell Migration/Mechanobiology**
- **Cell biology in development and disease/Host pathogen interaction**
- **New computational and imaging technologies for analysing cell function**
- **Endocytosis and Intracellular trafficking/Organelle Dynamics and Interactions/membranes and lipids**

Super early booking deadline: **21 January 2026**
Abstract & earlybird booking deadlines: **21 February 2026**
Register online: **bit.ly/Dynamic-Cell-VI**

Invited Speakers:

David Barford (UK)
Tobias Baumgart (USA)
Marianne Bronner (USA)
Roberto Dominguez (USA)
Anna Duncan (Denmark)
Jingyan Fu (Beijing)
Chuanhai Fu (China)
Anna Huttenlocher (USA)
Xing Liu (China)
Helen Matthews (UK)
Teresa Thurston (UK)
Paul Timpson (Australia)

Scientific Programme Committee:

Aymen al-Rawi (UK)
Kurt Anderson (UK)
Viji Draviam (UK)
Antreas Kalli (UK)
Matthias Krause (UK)
Laura Machesky (UK)
David Tumbarello (UK)
Melissa Webby (UK)
XueBiao Yao (China)

BIOCHEMICAL SOCIETY

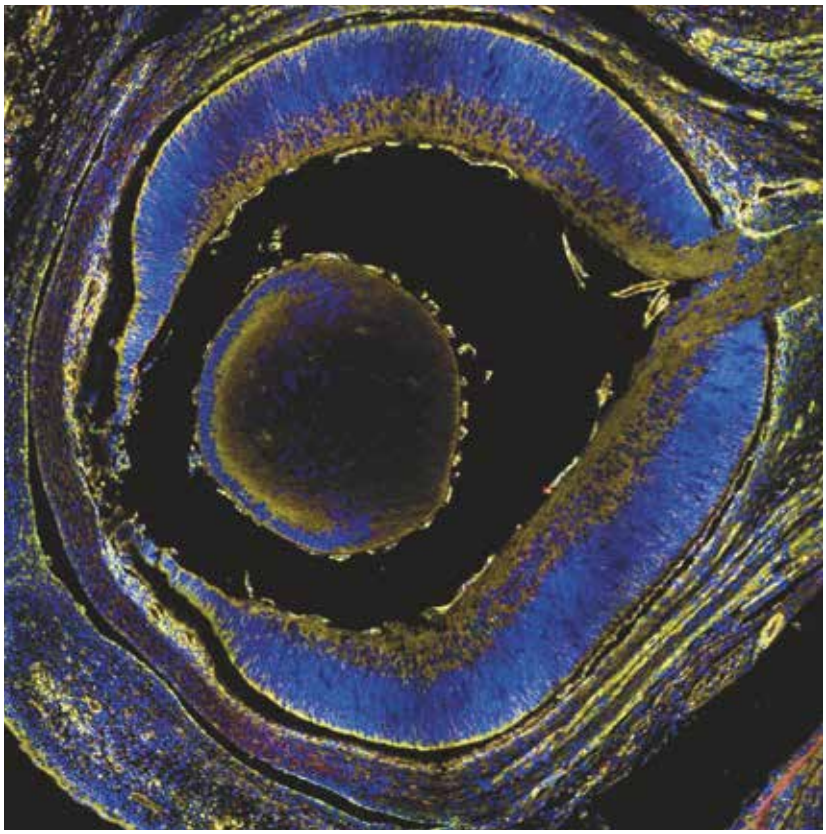
BSCB

In association with:

CSCB 中国细胞生物学学会 CHINESE SOCIETY FOR CELL BIOLOGY

RMS

BSCB Magazine 2026



Editorial

Welcome to the 2026 BSCB Magazine!

Last year we celebrated the 100th anniversary of the Company of Biologists, the not-for-profit publishing organisation dedicated to supporting the Biology community and biological societies, including the British Society for Cell Biology. The BSCB celebrated its own 60-year anniversary last year, and with this latest issue we want to highlight new insights from our members and future developments of BSCB and Biology at large. Our articles feature the new design of our Hooke Medal for Emerging Leader in Cell Biology, and the growing-role of artificial intelligence that is transforming how we study biology. We put a spotlight on new generations of cell biologists, with features from our early career researchers and students funded by the BSCB to attend conferences with its travel awards, the BSCB Postdoctoral Researcher Medal and the Raff Medal for PhD students.

We also want to flag up the brand-new and fantastic BSCB website (https://bscb.org/) where you will find the list of competitions, awards and support grants you can apply to, the past and future events relevant to our BSCB members, and how new members can join the BSCB.

We thank all contributors for this year's Magazine. You will learn about the many BSCB activities over the last year, the reports on the amazing work from our summer students, and all our award winners. You will also find a list of some of the cell biology events to look forward to in the coming year.

We encourage all current BSCB members to reach out, make suggestions for conferences and activities that would benefit the BSCB community, and to encourage your colleagues to join; it is your engagement that makes the BSCB a success so it can continue to thrive and support more Cell Biologists in the future.

James Brown and Mattéa Finelli

Front cover: The brainbow technique allows the visualisation of the neurons of the adult mouse gut.The image, by Alvaro Castano-Medina, won 1st place in the BSCB Image Awards 2025.

Above: This image represents a set of tiled images taken from a frozen section of an eye from the mitoQC mouse. The image, by Alan Prescott, won 2nd place in the BSCB Image Awards 2025.

Magazine Editors: James Brown and Mattéa Finelli **Production:** Giles Newton, Deadlift Media **Printer:** Hobbs
BSCB website: www.bscb.org

The Company of Biologists

Special Issue
Imaging Cell Architecture and Dynamics

Guest Editors: Francesca Bottanelli and Giulia Zanetti

Deadline: 1 April 2026

Journal of Cell Science

Call for papers

Find out more at:
journals.biologists.com/jcs/pages/imaging

Image: Antara Chakraborty, Indian Institute of Science Education and Research (IISER), Pune

Society News

BSCB President's Report 2025

As 2025 draws to a close, it is once again an appropriate moment to reflect on a highly successful year for the BSCB. I remain deeply grateful to our outstanding committee, whose dedication and voluntary service continue to support the British cell biology community and to nurture the next generation of researchers, ensuring that cell biology remains vibrant and resilient. I am also very thankful for the generous grant support that we receive each year from the Company of Biologists, allowing us to support the community. I would encourage all BSCB members to engage with the Company, who provide five excellent international journals, online communities and grants for UK-connected biologists. Finally, I am grateful to all of you- the members of the BSCB, who are the reason for the BSCB and whose great science we love to support.

Our annual meeting this year was a very special joint meeting with the Company of Biologists, who celebrated their centenary – 100 years of community journals- such as the Journal of Cell Science, Disease Models and Mechanisms, Development, Biology Open and the Journal of Experimental Research. The meeting was held in Liverpool on March 24-27, 2025. It was a great success, with representation from all of the COB journals and parallel sessions on diverse topics ranging from ecology to cell biology, developmental biology and biomedicine. The meeting was very well-attended and provided a great chance for different disciplines to come together and explore the latest scientific discoveries. The meeting opened with an early career discussion and workshop led by Richard Sever of Biorxiv. The gala dinner was held in the beautiful St George's Hall, a striking neoclassical building in the centre of Liverpool.

We held the BSCB award talks during the meeting, with Helen Weavers from the University of Bristol receiving the Women in Cell Biology Medal, Pei Yee Tay of the University of Liverpool receiving the Raff PhD student medal, Ian Ganley of the University of Dundee receiving the Hooke Medal and Pamela Swiatlowska of Imperial College London receiving the BSCB postdoc medal. Nominations for 2026 are now closed, so we look forward to announcing the winners at the next meeting. The meeting closed with a vibrant panel discussion about the future of science with Jennifer Lippincott-Schwartz, Jim Smith, Nick Hopwood, Maria Leptin and Tamina Lebek leading the discussion.

Please join us for the next BSCB Dynamic Cell VI meeting in Reading April 21-24, 2026. This meeting will be joint with the Biochemical Society and the Royal Microscopical Society and will be broadly focussed on the latest breakthroughs and application of new technologies to solving problems in cell biology.

We are particularly proud to announce that we have officially launched our new BSCB website, which can be found at the same location as the previous one: https://bscb.org/ Please try it out and let us know what you think. Helen Matthews (Sheffield University), our incoming social media and website member, will be helping to oversee the transition to the new site. Please be patient with us as we iron out the last few bugs, but do let us know if we can do anything to improve your experience and we will do our best.

Stay tuned for some updates from our early career representatives Harriet Smith (Francis Crick Institute) and Emily Lucas (Southampton University), who are launching a new online cell biology lecture series "BSCB NextGen". There will also be some exciting initiatives from Yee Dee Tay, our education committee member, to be announced later in 2026.

BSCB offers you many opportunities to participate in your cell biology community and we are always looking for new ideas and initiatives. If you would like to get involved in science policy, Darius Koester (Warwick University) would love to hear from you. If you are a cell biologist working in Ireland who would like to be more involved in the community, contact James Brown (University of Limerick), our Irish Area representative and co-editor of the BSCB magazine. If you have items for the BSCB magazine or ideas about how the BSCB can better serve the community, get in touch with James Brown or Mattéa Finelli (Nottingham University) and we will do all we can to make this your BSCB.

Laura Machesky

Meetings Calendar 2026–27

ScotFly26 – the Scottish Fly Meeting. 01 April 2026
https://www.eventbrite.com/e/scotfly26-the-scottish-fly-meeting-tickets-1710954303379

Dynamic Cell VI. 21–24 April 2026
https://bscb.org/meetings/dynamic-cell-vi/
To register: https://www.eventsforce.net/biochemsoc/frontend/reg/thome.csp?pageID=135392&eventID=249&traceRedir=2

2nd Scottish Cell Biology Meeting
Glasgow, Friday, 4 September 2026
Contact: Stephen Tait, stephen.tait@glasgow.ac.uk
Look for updates on the BSCB website

BSCB Hooke Medal Design

To mark the awarding of the Hooke medal we spoke with Pui Sze Ling, the artist chosen to design this prestigious medal, to understand her inspiration and motivation for the images used.

The Medal Design

"I am profoundly privileged to design the Hooke Medal, drawing inspiration from Robert Hooke's exquisite hand-drawn illustration of cork cells from his seminal 1665 publication, Micrographia. This groundbreaking work introduced the term "cell" into the realm of biology, marking a pivotal moment in our understanding of cellular structure.

The medal's intricate cellular pattern faithfully recreates Hooke's manuscript, symbolising his foundational contributions to cell theory and the inspiration he provided to subsequent generations of biologists. Through this design, I seek to honour Hooke's legacy, celebrating the unyielding curiosity of scientists who continue to unravel the mysteries of nature. The medal design stands as a tribute to their enduring contributions to humanity's quest for truth."

Knowing the artist

Ling Pui Sze is an artist who works at the intersection of science and art. With a focus on experimental ink and collage, she incorporates images of nature captured by a range of instruments, including microscopes and satellites. Over the past decade, Ling's work has been inspired by biological cells, including her own body cells and x-rays.

Her creative expression takes the form of printing, tearing, and shading microscopic images of cells, then piecing together the treated fragments using her intuitive understanding of nature. By collaging different types of paper and adjusting digital images through computer and inkjet printers, Ling creates textured artworks that evoke incidental organic forms, resembling the evolution of life and the creation of an alternate world on canvas. Ling also engages with other mediums such as sculpture, video, and installation. Through her artistic practice, Ling examines the relationship between biological traits and the intricacies of human experience.

Ling's work is included in the collections of the M+ Museum for Visual Culture, the Ashmolean Museum at the University of Oxford, and Robinson College, University of Cambridge. In 2012, Ling received the Wucius Wong Creative Ink Painting Award, and in 2023, she received an Honourable Mention for the Liu Kuo-sung Ink Art Award. Ling was an artist-in-residence at Robinson College and Jesus College, University of Cambridge, in 2023 and 2024. In 2025, she became a senior research associate at the Intellectual Forum, Jesus College, University of Cambridge.

The cells through art

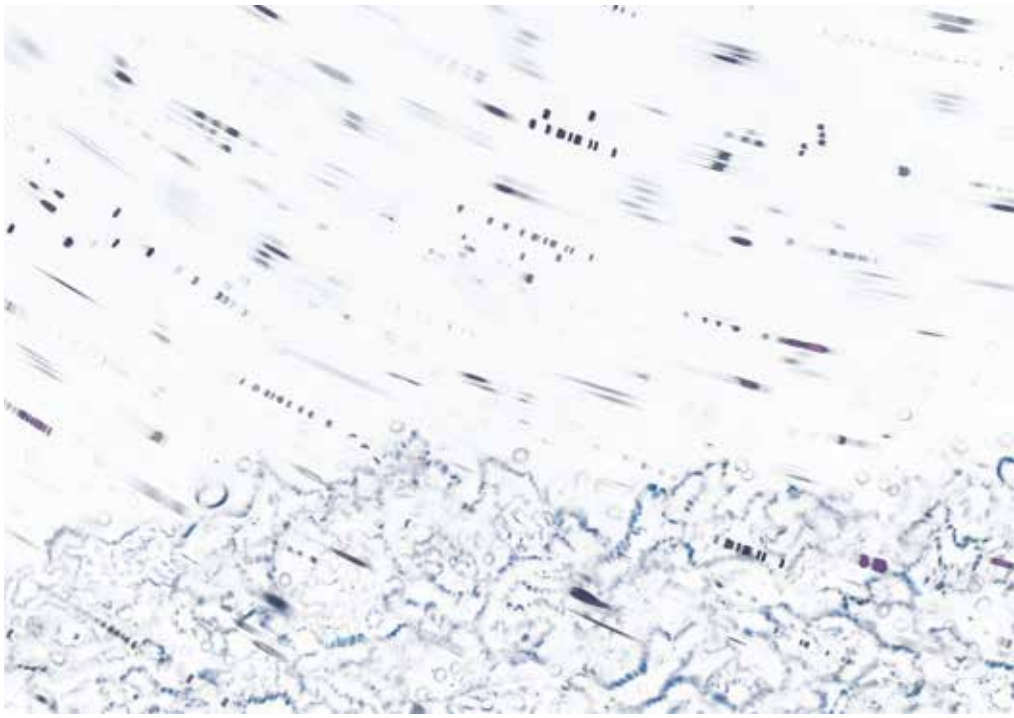
The White Mirror series (shown in the panel above) is composed of microscopic images of Ling Pui Sze's body cells and tears. By ink-jet printing these images on Washi paper, spraying them with water, and manually tearing them into small fragments, she then crafted a collaged image akin to galaxies. The body, as a vessel, is also a mirror of the mind, reflecting the connection between our inner essence and outer existence. This series of works embodies her introspective self-reflection. The presence of a single, minute human cell is akin to a minuscule particle within a vast universe.

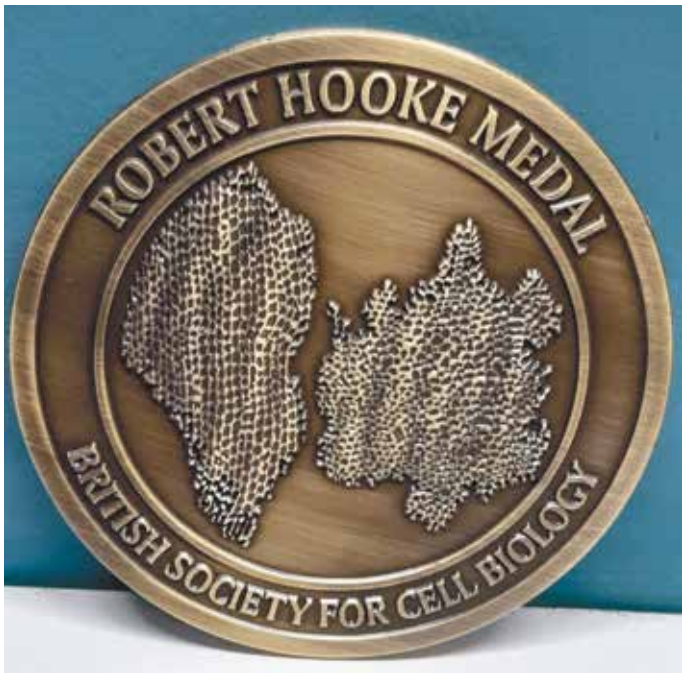
Tunneling 2 and Tunneling 3 (facing page, top) are based on DNA gel electrophoresis images combined with microscopic images of Ling Pui Sze's blood cells. These images are printed on washi paper using inkjet technology, then hand-torn into small pieces and collaged to evoke the imagery of a meteor shower.

Left, top: White Mirror 2, 57 x 133 cm, mixed media on paper, 2023.
Special thanks to Professors Laura Machesky and Robert Insall from Robinson College, University of Cambridge, for their invaluable support.

Above: Tunneling 3, 106.5 x 131 cm, mixed media on canvas, 2025
Right: Tunneling 2, 106.5 x 131 cm, mixed media on canvas, 2025. Excerpt.
Special thanks to Thieme T. Schmidt from the Cavendish Laboratory, University of Cambridge, and Professors Laura Machesky and Robert Insall from Robinson College, University of Cambridge, for their invaluable support.

Below: The Hooke Medal





Hooke medal winner 2025: Ian Ganley

Ian Ganley is the Professor of Cellular Homeostasis in the MRC Protein Phosphorylation and Ubiquitylation Unit at the University of Dundee. Ian obtained his undergraduate and Master's degree in Biochemistry from the University of Oxford, followed by a Ph.D. at the Babraham Institute in Cambridge where he studied the lipid hydrolase phospholipase D1 under the supervision of Nick Ktistakis. For his postdoctoral studies, Ian moved to Stanford University in California), joining the team of Suzanne Pfeffer to investigate the role of Rab proteins in intracellular transport. He then continued his research at the Memorial Sloan-Kettering Cancer Center in New York, working with Xuejun Jiang to study autophagy. In 2010, Ian started his own research group at the University of Dundee, focusing on identifying physiological signalling pathways that induce autophagy. In recognition of his work, he was awarded the 2025 Hooke Medal by the British Society for Cell Biology (BSCB).

Congratulations on winning the 2025 Hooke Medal; what does winning this award mean to you?
Thank you! It really means a lot. Pursuing a career in scientific research is a long and often challenging journey, full of bumps and obstacles. For me, it has involved a lot of doubt and stress along the way. That's why receiving recognition from peers with an award like this is incredibly meaningful. It's a sign that I am on the right path and gives me a real boost in confidence and motivation to keep going.

What inspired you to become a scientist?
Honestly, I never thought I'd become a scientist. Growing up, I wanted to be an astronaut and go into space. I probably still would if the opportunity came along! But like many scientists, I've always had a deep curiosity about how things work. I remember being in high school, looking through biology textbooks and being amazed by scanning electron micrographs. They let you peer inside the cell and see a whole other world, with these weird and wonderful membrane compartments. I thought it was so cool. I kept asking myself: how do these structures get there? What do they do? How do they form? That was probably the moment I realised I might end up exploring inner space instead of outer space.

How did you choose what to do and where to go for your PhD?
I didn't follow the most traditional path into a PhD. I studied biochemistry as an undergraduate and enjoyed the science, but I wasn't sure if I wanted to pursue it as a career. In my final summer of university, I spotted a small note on the department noticeboard advertising a summer job at a nearby biotech company, Oxford Glycosciences. I applied, got the position, and ended up working there full-time as a cell biologist. I tested compounds in cell death assays alongside chemists developing drugs. This work was hands-on and engaging. Because it was a small company, I was also involved in meetings with chemists and managers to discuss next steps, which gave me real insight into the drug discovery process. Eventually, I realised that to develop a long-term career in science, I would need a PhD. I was drawn to a project advertised by Nick Ktistakis at the Babraham Institute in Cambridge (UK), focused on kinase regulation of phospholipase D. It combined membrane biology and signalling, which was exactly the kind of science I was curious about. When I visited Babraham, I was impressed by the open-plan labs shared by multiple groups: Nick's group, as well as those lead by Len Stephens and Phil Hawkins, Simon Cook, and others. It was a collaborative and dynamic environment. The Babraham Institute was a fun and exciting place to do science, and it was where I truly fell in love with research.

Following your PhD with Dr Nick Ktistakis at the Babraham Institute (Cambridge, UK), you moved to the lab of Professor Suzanne Pfeffer at Stanford University for your postdoctoral research – what did you work on there?
For my postdoc, I wanted to broaden my scope and study membrane trafficking pathways. I started emailing scientists whose work I found exciting, hoping they might have a position available. Suzanne Pfeffer (University of Stanford (USA)) was one of them. She had developed an elegant in vitro system to study endosome-to-Golgi trafficking by reconstituting the transport pathway in a test tube. I saw this as a powerful way to dissect the underlying mechanisms, so I was thrilled when she invited me to visit and then

offered me a position in her lab. In Suzanne's lab, I used that assay to study retrograde transport from endosomes to the Golgi. I helped identify SNARE proteins involved in the pathway and explored how GTPases, particularly Rab proteins, regulate the process. It was a productive and rewarding time. I spent five years in Suzanne's lab, and the experience shaped my thinking about mechanistic cell biology.

Afterwards you did a second postdoc with Dr. Xuejun Jiang at the Memorial Sloan Kettering Cancer Centre. Why?
Doing a second postdoc was never part of the plan. I had intended to apply for group leader positions directly from Suzanne Pfeffer's lab. But life took an unexpected turn. My wife, Emma (we met during our PhDs in Cambridge, UK), was offered an incredible opportunity to become Executive Editor of The Journal of Cell Biology, based in New York. It was one of those offers that was simply too good to turn down. So, we moved to New York, and I started a postdoc in the lab of Xuejun Jiang (Memorial Sloan Kettering, USA). His group focused on cell death mechanisms and was beginning to explore autophagy. Together, we worked on autophagy initiation and identified an important protein complex in triggering the process. That project helped me gain a foothold in the autophagy field, which has since become central to my research.

Undertaking mobility is part of the career of many scientists. You have worked in different cities in the UK and the USA. Tell us more about your experience as a global nomad.
Academia is one of the few careers where moving abroad is relatively straightforward, especially compared to many other professions. I've always believed that variety is the spice of life, and living in different places exposes you to different perspectives. In the lab, you meet people from all over the world, often working right beside you. Your colleagues might come from five or ten different countries, each bringing a unique approach to science. That diversity of thought and experience is incredibly enriching, both professionally and personally. Of course, moving abroad can be daunting. It pushes you out of your comfort zone. But for me, relocating made me more resilient and confident, in the lab and in life. There's real value in challenging yourself, especially by doing things that feel uncomfortable at first. Those are often the experiences that help you grow the most.

After two postdocs in the USA, you moved back to the University of Dundee in 2010 to establish your research group. What motivated that decision?
I think it reflected the stage of life we were in. Both of our children were born in New York, and we had started to really miss being close to family. The idea of moving back to the UK, or somewhere in Europe, started to take root. Around that time, Sir Philip Cohen, then director of the MRC Protein Phosphorylation Unit in Dundee, gave a lecture at Memorial Sloan Kettering. He gave a fantastic talk and, almost as an aside at the end, mentioned they were looking for new PIs to join the institute. I remember thinking, 'interesting timing'. I knew Dundee had a strong reputation, and Philip made it sound like a fantastic place to do science. I figured I had nothing to lose by applying. To my surprise, Philip called and invited me for an interview. When I arrived in Dundee, I was genuinely impressed. The environment was welcoming and collaborative, and I immediately felt it was a place where I could do the kind of science I cared for. So, when they offered me the position, it was an easy decision. I accepted right away. As a bonus, my family was happy to relocate to Scotland. Dundee is a brilliant place to raise a family. The move felt right for the science, for the family, and for the balance we were looking for at that point in our lives.

What are the main research questions that your lab addresses?
One of the central questions we're trying to answer is: what signals lead to the formation of an autophagosome? This ties back to the questions that first drew me into science: how do membrane compartments form? How do organelles form from scratch? Autophagy is a powerful process to explore these questions, because you can introduce defined signals and observe a new organelle, the autophagosome, form de novo. It's a dynamic example of membrane biogenesis. In the lab, we're especially interested in mitophagy, the selective autophagy of mitochondria. We've learned a lot about the molecular tags that mark mitochondria for degradation, and we are now focusing on the physiological signals that trigger mitophagy under normal and disease conditions. This is particularly relevant to diseases like Parkinson's, where mitophagy appears to be disrupted. So we're not just studying the molecular machinery, but we're also investigating how it's regulated in a physiological context. We've developed several tools, including assays and reporters, that have attracted interest from pharmaceutical companies. These collaborations allow us to test compounds in more physiologically relevant models. In many ways, it brings me back to my early experience in industry and the translational science that first got me interested in research.

Are you still doing experiments yourself?
Not anymore, no. I still give a lot of advice, but I'm no longer at the bench. The main reason is simply time. Experiments often take several days, and it's hard to carve out the uninterrupted time needed to see one through from start to finish. I could start something, but someone else would inevitably have to finish it, which isn't fair. Honestly, the longer I've been away from the bench, the more disruptive I've became. I think my lab quietly dreads those moments when I show up asking, "Where's this? What's that?" They've made everything far more efficient than how I used to do it, and at this point, I tend to get in the way more than I help. It was a tough decision to officially give up my bench space, I had a real emotional attachment to it, but it was the right call. That said, I do miss it, and I still get the itch to be hands-on.

Did you have any mentors that helped you in your career?
I have been incredibly lucky to have great mentors at every

stage of my life and career. It started with my parents, and in high school I had fantastic biology and chemistry teachers. Honestly, I probably wouldn't be here without them: they made science engaging, fun, and something I could see myself doing. As I progressed academically, I sought out environments with people I could talk to, who offered honest and thoughtful advice. That's something I've valued and actively looked for in every position. I believe mentorship matters at every career stage, not just early on. Everyone needs someone to turn to for guidance. I've been fortunate to have brilliant mentors: my PhD supervisor Nick Ktistakis, and my postdoc mentors Suzanne Pfeffer and Xuejun Jiang. They gave me invaluable support and direction. That support has continued here in Dundee, where colleagues like Philip Cohen and Dario Alessi have generously shared their time and advice. Mentorship has played a big role throughout my journey, and I always encourage others to seek it out and take full advantage of it.

What is your approach to mentorship with your lab members?

I think one of the most important things I realised early on in mentoring is that every person is different. We all have unique needs, goals and ways of working. A key part of my role as a mentor is to understand what each individual wants from their research experience. Once I understand that, my job is to support them in doing their best science while making the process as enjoyable and manageable as possible. I strongly believe in keeping an open-door policy. Being approachable and available is essential. Making time to really listen when someone comes to me with questions or concerns helps build trust and creates a supportive environment. Ultimately, mentorship for me is about helping people thrive both scientifically and personally by tailoring guidance to their individual strengths and challenges.

If you could change one thing in academia, what would it be?

Academia is far from perfect, and like any system, it has its pros and cons. If I could change one thing, it would be to improve stability. So much depends on grant success, which is often unpredictable and stressful. This uncertainty makes it difficult to plan for the future. Even now, I'm not entirely sure where I'll be in five years. That insecurity is especially challenging for excellent postdocs and scientists who may not want to become group leaders but still have much to contribute. In the UK, it's very hard to find permanent or stable roles for senior postdocs. We risk losing highly trained, competent scientists simply because there aren't enough long-term positions available. Unfortunately, much of this stems from a broader issue: lack of funding, which is a widespread issue beyond academia itself.

Finally, is there anything our readers would be surprised to find out about you?

Actually, yes! I have a pet snowflake moray eel named Eel Armstrong. I've had him for about three or four years. Keeping saltwater aquariums has been a hobby of mine for over a decade. I got Eel Armstrong from a local pet shop. He usually stays tucked away, just like in nature documentaries, with his head poking out from a crevice. But he definitely knows when it's feeding time! He'll come out, swim around the tank, and happily eat his food.

Ian Ganley contact details: MRC PPU, Sir James Black Centre, School of Life Sciences, University of Dundee, Dow St., Dundee, DD1 5EH, UK E-mail: i.ganley@dundee.ac.uk

Ian Ganley was interviewed by Sara Morais da Silva, Reviews Editor at Journal of Cell Science.

This piece has been edited and condensed with approval from the interviewee.
Reproduced with permission from JCS, original article published 1/8/2025: https://doi.org/10.1242/jcs.264278

BSCB Women in Cell Biology Early Career Medal Winner: Helen Weavers

Helen Weavers is a Wellcome Trust Career Development Fellow in the Faculty of Health and Life Sciences at the University of Bristol, UK. She completed her PhD studying renal morphogenesis with Prof Helen Skaer at the University of Cambridge, before moving to Bristol for her postdoc, where she investigated tissue repair and inflammation. She established her own group in Bristol in 2018 studying the cell biology of tissue resilience, funded by a Wellcome Trust and Royal Society Sir Henry Dale Fellowship. Helen was awarded the 2025 BSCB Women in Cell Biology Early Career Medal.

What does winning this award mean to you?
I've attended the BSCB Spring Meeting regularly since my postgraduate days, and have watched many inspirational women receive this award, so it is an honour to be a recipient myself. It was particularly special to receive the medal at the Biologists@100 Conference celebrating The Company of Biologists' 100th anniversary - and at the same Meeting where my PhD Supervisor, Helen Skaer, very deservedly received the BSDB Waddington Medal. As a young female PI, receiving this recognition whilst on maternity leave was also especially poignant.

What first inspired you to become a scientist and what was your career path?
Ever since childhood, I've been very curious about the way things work – always taking objects apart and figuring out how to put them back together again! So the problem-solving aspect of research is something that I'm naturally drawn to. I also love being artistic and creative. The research I do now, which relies on cutting-edge microscopy, really allows me to combine these interests. I'm constantly inspired by the beauty of the cells and tissues we study.

My scientific journey has been quite serendipitous. I didn't set out to establish my own research group; I've followed my interests and fortunately this has led to my current role. As an undergraduate at the University of Cambridge, I spent a Summer in Helen Skaer's lab studying renal biology. This was my first taste of scientific research and I loved it - so I stayed on in Helen's lab for my PhD. During this time, I became fascinated by how our tissues develop – how different cell types get to the right place, at the right time – so they can ultimately work together as a functional, integrated tissue.

For my postdoc, I moved to Bristol to study not how tissues develop, but how they return to their functional state if damaged, with Paul Martin and Will Wood. Tissue repair is now widely considered a recapitulation of earlier development, reusing many of the same dynamic cellular behaviours. During this time, I harnessed the fruit fly *Drosophila* as a powerful in vivo model to study the dynamic cell biology that underpinned tissue repair following injury. We made important discoveries about the wound-induced signals that recruit inflammatory cells to the injury site, and how these immune cells develop a 'molecular memory', squeezing out of vessels en route to the wound. My postdoc was highly collaborative, working with mathematicians and initiating links with genetic epidemiologists and clinicians, many of whom I still collaborate with today.

Your research focuses on uncovering what drives tissue and cellular resilience, what drew you to work on this research area?
In my own group, we now investigate the dynamic cell biology that enables diverse tissues to thrive under challenging conditions. In particular, we explore the fundamental molecular adaptations that enable cells to resist and recover from everyday stressors, including metabolic byproducts, toxic inflammatory mediators or physical damage – and how dysregulation of these resilience mechanisms underlies premature ageing or disease. Our ultimate goal is to identify ways to improve life-long tissue health and recovery.

When searching for a research niche for my own group, my prior PhD and postdoc work aligned in delightfully unexpected ways. After identifying a number of key genes upregulated in wounded skin that were essential for efficient tissue repair, I serendipitously discovered these genes were also highly expressed within the renal system, even under healthy uninjured conditions. Since kidneys must routinely function

under challenging conditions, I realised diverse tissues (such as the skin and renal system) must employ surprisingly similar molecular 'resilience' strategies to stay healthy. We now successfully employ this cross-tissue approach to identify fundamental mechanisms of resilience – and continue to add more tissues to our (growing) collection.

What are the main research questions that your lab addresses?

Harnessing powerful in vivo models, we study cellular resilience from a whole-tissue and whole-organism perspective. First, we probe how individual cell types program their molecular cell biology within living, intact tissues. Second, we explore how these unique biochemical adaptations more widely shape the health and function of whole organs and organisms. In this way, our work spans biological scales.

While it is not surprising that barrier tissues (e.g. the skin and airways) are vulnerable to damage encountered in their environment, we find similar protective mechanisms are utilised on a daily basis by internal body tissues (e.g. kidneys). Many internal organs must be well-adapted to resist challenge, but the kidneys are particularly vulnerable as they possess very long-lived cells that are highly metabolically active. My group have identified numerous self-defence strategies, including redox control and metabolic reprogramming, that collectively limit cellular damage, senescence and cell death within the tissue. Supported by my recent Wellcome Trust Career Development Award, we're now asking how this cell biology is patterned in space and time across the tissue – and whether loss of resilience is the root cause of many (age-related) diseases.

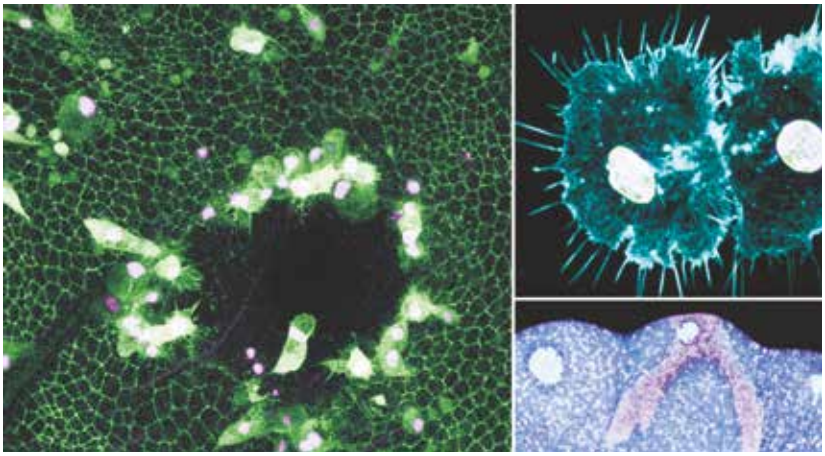
We take an integrated, interdisciplinary approach, that combines in vivo studies in the fruit fly *Drosophila* (with state-of-the-art live imaging, genetics, molecular cell biology and 'omics) with computational modelling and genetic epidemiology. We're now exploring ways to therapeutically boost cellular resilience over the life course and thus collaborate with clinicians, including those studying regenerative medicine and organ transplantation. You can read more about our work here (www.tissueresilience.com) and discover more about our open postdoc positions!

Did you face any challenges when you started your lab that you didn't expect?

A key challenge when first starting a lab, is building a cohesive and supportive team. When recruiting new members, it's important to consider not only scientific skills but how an individual might integrate into the wider group. As well as bringing together complementary research expertise from different individuals, it is important to recruit personalities that will foster a supportive lab culture. Recruiting my first wave of postdocs and PhD students was daunting – it's quite nerve wracking to ask if anyone wants to come and work with you! Fortunately, I was lucky to recruit many enthusiastic and creative researchers with diverse skillsets, who were excited to help build the lab infrastructure from scratch. The group quickly felt like an integrated, supportive team. I'm currently recruiting new post-docs and will strive to continue this approach as my lab grows and matures.

Is there any other advice you would give to someone starting their lab?

It's a cliché, but resilience is important when building an academic scientific career. There will inevitably be lots of bumps in the road, which are often no fault of your own. Many people find it challenging to identify their research niche – you may not stumble on this straight away - but if you can find an area that you genuinely find fascinating, you'll be far more resilient in coping with the inevitable challenges that your group will face. When starting out as a group leader, your role transitions dramatically overnight – and often without any formal project or people management training. New PIs often feel under considerable pressure to quickly build a team and publish data. Looking back, I probably got too caught up worrying about trivial issues – now I try to focus on the bigger picture and just enjoy the process of scientific discovery, bumps and all.



Above: Confocal images showing wound inflammation (left), immune cells (right, top) and renal tissue (right, bottom) in *Drosophila*.

Did you have any mentors that helped you in your career?

My PhD supervisor, Helen Skaer, has always been an inspirational role model for me. She is a creative, thoughtful scientist who explains her work in a beautifully clear way. Helen's also very supportive of everyone she interacts with. This year, she was awarded the prestigious Waddington Medal by the British Society for Developmental Biology at the Biologists@100 conference, so it was particularly special that we were both awarded medals at the same meeting.

What is your approach to mentorship with your lab members, and what advice would you give to other mentors?

I've been fortunate to receive great mentorship from my PhD and postdoctoral supervisors, so I know first-hand how important this guidance can be when navigating academia and considering different career paths. It's important to remember that every group member is an individual, so you must adapt your leadership and mentorship approach to best fit each unique personality. Many of the PhD students and postdocs in my group are supervised by, or collaborate closely with, other group leaders across the Faculty, which enables them to receive mentorship from different perspectives.

You received a NC3Rs Public Engagement Award, can you describe how important it is for you to incorporate PPI in your research?

Patient and public engagement really helps guide and inform our research. We regularly present to patient groups (e.g. individuals living with kidney disease), as well as at public events (including Science Festivals and 'Pint of Science') and Schools/Colleges. We recently took hands-on microscope activities to a local Summer Science Festival - often at these events, the kids ask the best questions! Our Public Engagement Award from the National Centre for the Replacement, Refinement and Reduction of Animals in Research (NC3Rs) is now helping us generate more interactive resources for these outreach activities.

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BSCB Postdoctoral Researcher Medal Winner: Pamela Swiatlowska

Dr Pamela Swiatlowska is a Postdoctoral Researcher at the National Heart and Lung Institute in the Faculty of Medicine at Imperial College London and a visiting Postdoctoral Researcher at Queen Mary University London. Her independent research explores how cells of the heart sense and respond to their mechanical environment using cross-disciplinary approaches.

What does winning this award mean to you?
I'm really honoured to receive this award. It represents many years of my hard work, dedication, persistence, and personal sacrifice. At the same time, it champions curiosity, the main driver of my research, while highlighting the importance of basic research in Cell Biology and further motivating me to continue my work.

The career path of a scientist is well captured by the Iceberg Theory of Success – what is visible to everyone is just the tip, while beneath the surface lies an immense amount of unseen effort, setbacks, and resilience.

I always approached every unsuccessful grant or application not as a failure, but as another lesson to learn from. That's why the BSCB Medal means a lot to me. Dream it, believe it!

What inspired you to work in Cell Biology?
My interest in Cell Biology started during my Undergraduate studies in Biotechnology, where I was particularly fascinated by its medical applications. From as early as my first year, I was involved in public engagement activities to promote Cell Biology, a passion that has remained constant throughout my career. During my PhD, I developed a deeper interest in the interface between Cell Biology and Mechanobiology. This interdisciplinary focus has provided me with a new landscape for exploring how cells interact with their physical environment, and it continues to shape my research trajectory till this day.

What's your favourite cell and why?
I am fascinated by two cell types: cardiomyocytes and smooth muscle cells. The structural complexity and the ability to generate rhythmic contractions make cardiomyocytes a really interesting cell model to study. Watching them contracting 'in a dish' is really mesmerising. On the other hand, smooth muscle cells looking maybe less complex, but hold a huge potential. These somatic cells contain incredible degree of plasticity that allows them to shift the phenotype and the function.

What first inspired you to become a scientist and what was your career path?
I used to watch *The Magic School Bus* animated series very often as a child, which made science feel like an adventure. It sparked my imagination and inspired me to one day explore and solve scientific mysteries of my own. Along the way, Maria Skłodowska-Curie served as a major role model for me.

My scientific path began with Undergraduate studies in Biotechnology at the Intercollegiate Faculty of Biotechnology, shared between the Medical University of Gdańsk and the University of Gdańsk. A game-changing moment in my career was receiving a Fulbright Scholarship, which enabled me to conduct my MSc research on smooth muscle cell plasticity in Atherosclerosis at the University of Virginia. This led to two co-authored publications in *Nature Medicine* and a PhD Studentship at Imperial College London.

During my PhD at Imperial College London, I specialized in Cardiac Mechanobiology, employing mechano-Scanning Ion Conductance Microscopy to probe the transverse Young's modulus of cardiomyocytes and other cell types. I then pursued Postdoctoral Research in Cardiovascular Mechanobiology at both, New York University and Queen Mary University of London. Currently, I hold a British Heart Foundation Centre of Research Excellence Fellowship at imperial College London, which supports my ongoing work in Cardiovascular Nuclear Mechanobiology.

How would you describe your research in a nutshell?
Now, as a Fellow, my independent research explores how mechanical stimuli regulate the nuclear structure, signalling, epigenetic modifications (mechano-epigenetics), chromatin 3D architecture, nuclear lipids (mechano-metabolism) and nuclear mechano-properties in Heart Failure, Atherosclerosis, and Aging; while emphasizing the sex differences.

J Cell Sci (2025) 138 (15): jcs264278.
https://doi.org/10.1242/jcs.264278

The Algorithmic Lens: How AI is Reshaping Cell Biology

With the advent of high-throughput and high-content microscopy, the primary bottleneck in the life sciences is no longer image acquisition, but interpretation. As "Generalist" AI agents and self-driving microscopes enter the lab, cell biology is undergoing one of its most significant transformations since the invention of the lens.

For centuries, the trajectory of biological discovery was defined by glass. From van Leeuwenhoek's single lens to the super-resolution revolution, seeing better meant better optics. But recently, the paradigm has shifted. We have entered an era where human interpretation is increasingly replaced by automated quantification, so the hardware is secondary to the software that interprets it. The Deep Learning era, characterised by massive Foundation Models and Generative AI, is not just analysing our images; it is effectively creating them, denoising them, and even deciding which ones to take next.

For the cell biologist, this shift could lead to an exciting future. The days of manually counting cells or squinting to see if cells have changed shape are ending, replaced by tools that automate much of the scientific workflow. Self-supervised learning automatically finds interesting patterns in massive imaging datasets, virtual staining bypasses chemistry to generate synthetic spatial proteomics, and intelligent microscopes can run their own experiments while the researcher sleeps.

Seeing

The Rise of the Generalist

Until recently, using AI in microscopy meant training a specialist. If you wanted to segment mitochondria in cultured cells, you trained a U-Net on hundreds of annotated mitochondria. If you switched to nuclei in zebrafish, you started over. This brittleness has been the major barrier to widespread adoption.

The defining recent breakthrough is the arrival of the foundation model. Borrowing from the success of models such as ChatGPT and Gemini, these models are trained on such vast, heterogeneous datasets that they learn a generalised representation of biological morphology. AI models typically perform poorly on data outside their training domain, and foundation models overcome this by effectively making the entire universe in-domain.

Take CellSAM1 or cyto32 as prime examples. Trained on millions of cellular structures, these models possess a "zero-shot" capability: they can segment a dividing yeast cell, a neuron, or a kidney glomerulus with human-level accuracy, without necessarily having seen that specific experiment before.

This shift is profound. It means that high-end image analysis is no longer the preserve of labs with massive training datasets and computing resources. A newcomer can now take a raw image, feed it into a model, and receive annotations that previously would have taken weeks of manual curation to produce.

Label Free Virtual Staining

Traditionally, visualising specific structures required chemical staining - a process that is often toxic, time-consuming, and destructive to the sample. New generative models allow researchers to take a standard, label-free image (like phase contrast or autofluorescence) and computationally "paint" it to resemble a chemically stained specimen.

The implications for tissue preservation are enormous. A biopsy can be virtually stained with H&E for diagnosis, then "wiped clean" and virtually stained again for an unlimited number of specific protein markers, all while leaving the physical tissue pristine for other downstream uses such as genomic sequencing.

This could also enable the staining of live cells to generate new insights into previously intractable cell processes. Live cell staining is strictly limited by the need to preserve the health and functionality of the cells. Virtual staining circumvents this, avoiding the physiological perturbations caused by toxicity, photobleaching, stain variability, and fixation, effectively allowing us to watch "invisible" biology unfold.

Interpreting

Mining the Latent Space: Self-Supervised Hypothesis Generation

The most exciting frontier in 2025 is not just automating what humans can do, but doing what humans cannot. Traditionally, we taught AI to look for things we already knew—nuclei, edges, or specific manual labels. The new wave of Self-Supervised Learning (SSL) flips this script. Models like HPL3, UNI4, Virchow5, and HyPERSTAC6 are designed to learn a deep understanding of cellular morphology without any human labels.

The result is a "phenotypic fingerprint" for every cell or tissue patch: a mathematical representation that captures subtle changes invisible to the human eye. This allows for unbiased hypothesis generation. Instead of asking, "does this drug change the nucleus size?", researchers can ask, "what did this drug do?". The model might reveal that a candidate compound creates a unique phenotypic cluster distinct from known toxicity profiles, suggesting a novel mechanism of action. It transforms each image into a data

point; if a set of points lands in uncharted territory, you've found a new phenotype.

This does not discount the value of human knowledge. In many cases, we know that a specific protein or image feature is particularly relevant to what we want the model to learn. Architectures such as TriDeNT7, TANGLE8, and SpliCER9 have been designed to integrate paired data, such as spatial proteomics, transcriptomics, or manual annotations, to distil knowledge into models, leading to better downstream performance and more specific biological discovery. In scenarios where data is scarce, we can even utilise synthetically generated data to train these models, with methods such as SyMPP10 distilling complex biological information into the model without the need for endless manual experimentation.

This capability is evolving into active scientific reasoning. New "Agentic AI" systems, such as Google's AI Co-scientist11, can analyse these morphological patterns to formulate structured, testable hypotheses. By identifying statistical anomalies in the latent space, these agents effectively act as virtual collaborators, suggesting new experimental directions that a human researcher might overlook.

Collaborating

The Self-Driving Lab

While software analyses data, who is collecting it? Researchers are pioneering the concept of the self-driving microscope, which integrates AI directly into the hardware control loop. Instead of passively snapping images every 5 minutes, the microscope uses a lightweight AI model to watch the sample in real-time. It waits for a specific biological event - a cell entering mitosis, or a mitochondrial fission event - and only then triggers high-speed super-resolution imaging.

This event-driven acquisition solves two of the biggest headaches in live-cell imaging: phototoxicity and data overload. By keeping the light off until something interesting happens, the system protects the sample and ensures the outputs are primarily signal, not noise.

Similarly, the new Cell-TRACTR12 model can use AI to keep tabs on individual cells as they move, divide, and interact over multiple days. By treating tracking as a language-like sequence problem, it maintains lineages with a robustness that heuristic algorithms do not match.

Democratisation: "Chat-to-Analyse"

The most culturally significant shift, however, is accessibility. The barrier to entry for these powerful tools is collapsing. Python is no longer a pre-requisite for accessing state-of-the-art models.

Many code-free platforms have been integrated into web-based ecosystems, allowing researchers to build complex deep-learning pipelines using a simple graphical interface. Even more intuitive are the new "Conversational AI" tools. Omega13, a plugin for the multi-dimensional image viewer napari, acts as an intelligent assistant. A user can simply type, "Segment the nuclei in the red channel and plot their size distribution," and the assistant writes the code, executes the analysis, and displays the graph.

Risk: When AI Hallucinates

The rapid progress in AI is not without its drawbacks. Generative models are prone to hallucination, where models fabricate plausible but non-existent biological features. A virtual stain might insert a nucleus where there is only debris, or "clean up" a pathological lesion, rendering a pathological tissue healthy.

The community is responding with "Watchdog AI", such as AQuA14, which runs alongside the generative model, autonomously flagging pixels where the AI's "imagination" has drifted too far from the statistical reality of the raw data. Other approaches render models able to quantify their uncertainty, enabling them to tell the user what they don't know, alongside what they do.

Outlook

As we move through 2025, the definition of a "microscopist" is expanding. It now includes the ability to curate data, validate algorithms, and navigate the "latent space" of high-dimensional biology. Tools like EfficientBioAI15 are even making these models "greener" and faster, ensuring they can run on standard lab computers rather than massive clusters.

The microscope of the future isn't just a tube with lenses; it's an intelligent agent. The challenge for us, as biologists, is to learn how to collaborate with it.

Lucas Farndale and Ke Yuan
School of Cancer Sciences and Cancer Research UK Scotland Institute

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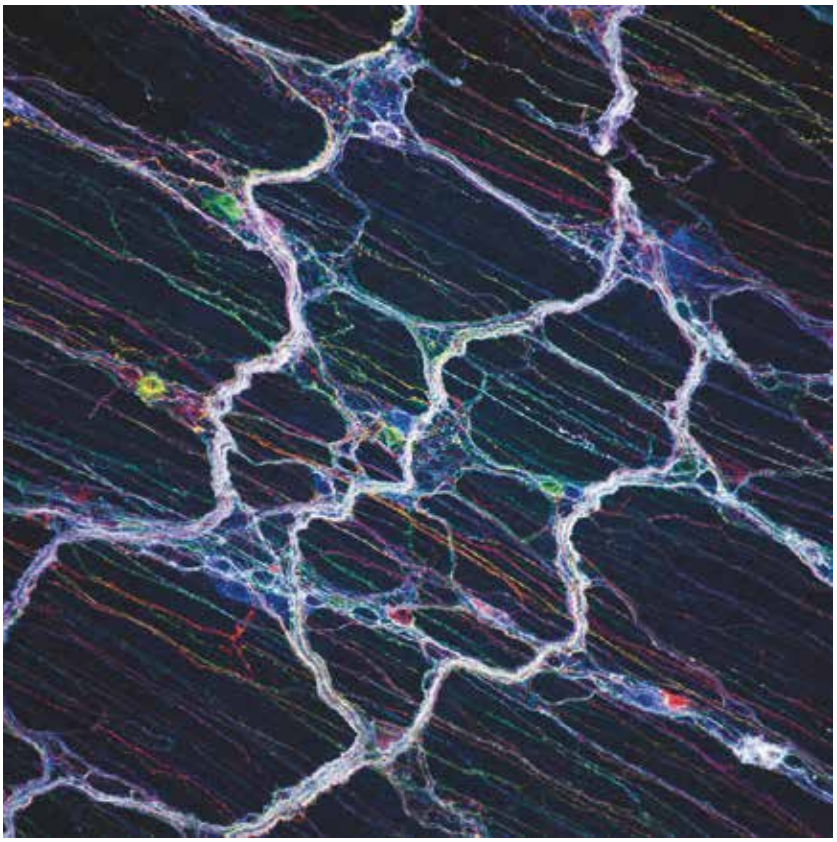
Image Competition 2025

1st place: Alvaro Castano-Medina



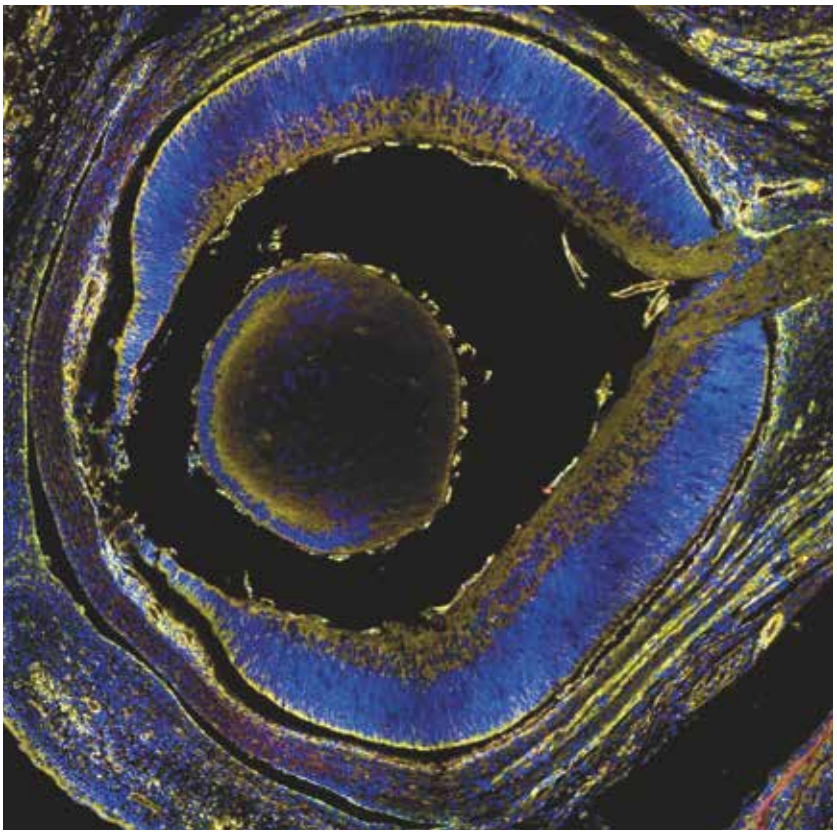
"Did you know there is a 'second brain' in the gut, with around 100 million nerve cells, more than in the spinal cord? In this image, we used fluorescent labelling to capture the beauty of our gut's own brain, known as the enteric nervous system (ENS).

To create this image we used the 'brainbow' technique, where viral vectors (adeno-associated brainbow viruses) deliver genes tagged with different colours into the neurons of the adult mouse gut. Different neurons will express three or four fluorescent proteins in different ratios, resulting in colour combinations that are unique to specific types of neurons and its projections. This high-resolution image was acquired using a Zeiss LSM Airyscan confocal microscope.

Clusters of enteric neurons, called ganglia, can be found at the junction of these tracts. Collectively this network of cells form the ENS, which independently orchestrates various gastrointestinal functions, such as sensing changes in the environment and coordinating muscle movements for digestion and gland secretions.

My research focuses on understanding how environmental factors such as diet, microbes and hormones impact ENS circuits to regulate intestinal physiology. During my PhD in the labs of Irene Miguel-Aliaga and Vassilis Pachnis at Imperial College and The Francis Crick Institute in London, I investigated how and why the maternal ENS adapts to the dramatic growth of the gut during pregnancy and lactation in mice."

2nd place: Alan Prescott



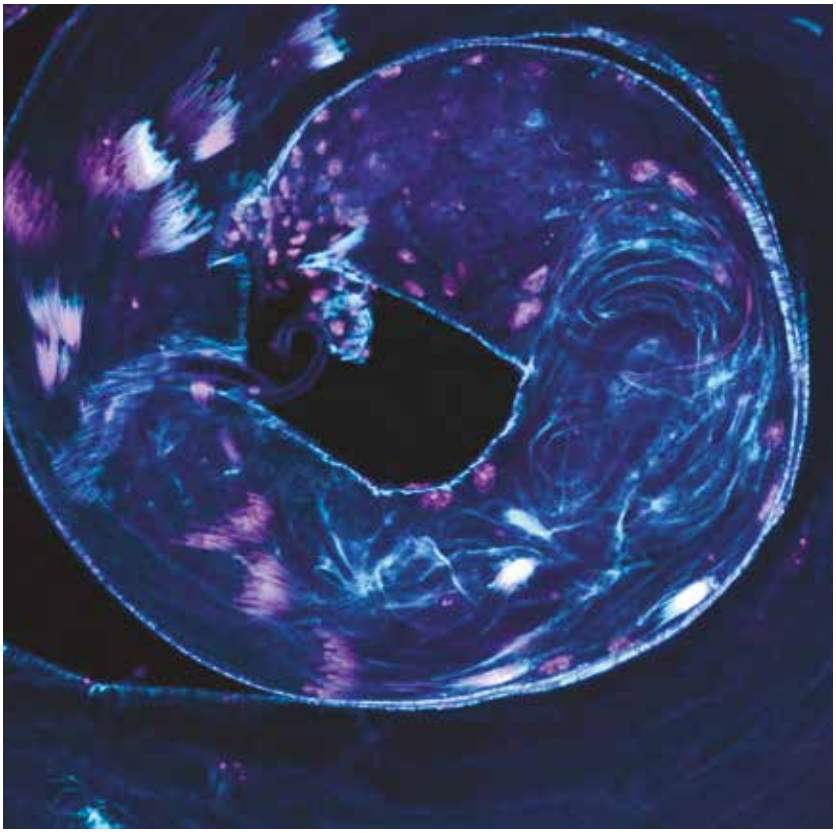
"This image represents a set of tiled images taken from a frozen section of an eye from the mitoQC mouse (McWilliams et al. J Cell Biol. 2016 Aug1;214(3):333-45) at developmental stage E16.5. Mitochondria are labelled with GFP(Green) and mCherry(Red). DAPI-stained nuclei are blue. Large red dots are mitochondria in mito-lysosomes demonstrating turn-over of damaged or worn-out mitochondria in active tissues-in this case the developing eye. The acidic environment of the lysosomes quenches the GFP fluorescence.

The mitoQC mouse model has revealed the distribution of Mitophagy in diverse active tissues such as the heart and retina. In addition, it unveils the tissue architecture as delineated by the distribution of mitochondria.

At this developmental stage the eyelids are closed, and the posterior chamber still contains blood vessels to support the developing lens and retina. We have characterised Mitophagy and Autophagy across several tissues of the eye with interesting findings in the selectivity of these processes in vivo (McWilliams et al. Autophagy. 2019 Jul;15(7):1296-1308).

My main research role is as Electron Microscopist for the Dundee Imaging Facility but for a long time I have also used laser scanning confocal microscopy as a second tool. My career has run alongside the life of the commercial confocal microscope beginning on the original LMB prototype through the first commercial Bio-Rad instruments

to the current crop of super-resolution 'scopes. I also help to run courses in the Duncan of Jordanstone College of Art and Design, in Dundee, working with art students using microscopy to generate imagery which they take forward into their work. These art-science interactions not only extend my horizons beyond the laboratory but allows me to enthuse about my interests in science with a large cohort of non-scientists. The BSCB Image Competition is another great opportunity to share the hidden beauty of the natural world with everyone."



3nd place Katarina Grobicki, Bristol

"This image shows the late stages of *Drosophila* spermatogenesis and was taken during my PhD. Whole testes were dissected, fixed for immunofluorescence, and imaged on a Leica SP8 confocal. DAPI (magenta) stains DNA while phalloidin (cyan) shows F-actin. In the top left of the image, bundles of 64 interconnected spermatids are individualised by actin-rich investment cones.

I recently finished my PhD in Felipe Karam Teixeira's lab at the University of Cambridge, where I used Drosophila genetics to investigate the roles of ribosomal protein paralogues in the germline. Whilst I did not find any evidence to support the much-anticipated "specialised ribosomes", I uncovered interesting multi-tissue responses to ribosome insufficiency. This sparked my interest in stress, which I am now pursuing as a postdoc in Helen Weavers' lab at the University of Bristol, studying cellular stress responses in ageing, development and disease."

Science Writing Prize Winner 2025 – Katie Birditt

A Drop of Blood – Holding the Secret to Dementia Diagnosis

When I envisioned becoming a scientist, I never imagined my work would resemble that of the suave, moustached detective immortalized in Agatha Christie's famous novels. There is something undeniably compelling about detective stories - the thrill of uncovering hidden clues, piecing together fragmented evidence, and solving puzzling mysteries. In many ways, my work as a PhD student mirrors this process. Instead of searching for a criminal, I scour blood samples for clues in individuals with Alzheimer's disease, hoping to solve the mystery of what could be causing the devastating condition. Just as detectives follow leads to crack a case, neuroscientists are learning to interpret the cryptic messages hidden in blood. These biological fingerprints could hold the key to unraveling the mystery of neurodegenerative diseases and may one day transform how we diagnose, monitor, and treat these conditions.

Decoding a Complex, Multifaceted Crime

Dementia is not a single disease but rather an umbrella term that encompasses an array of neurodegenerative conditions which include Alzheimer's, Parkinson's, frontotemporal, and vascular dementias.(1) Imagine dementia as a master criminal employing different but interconnected methods to disrupt the brain's neural networks. Proteins have typically been the prime suspects in these neurodegenerative conditions. The burden of guilt has largely fallen on amyloid-beta and tau proteins in Alzheimer's disease. Under normal conditions, both proteins support essential brain functions. However, when they misfold and subsequently accumulate, they trigger a cascade of cellular dysfunction that culminates in the death of brain cells. (2) Unfortunately, by the time the characteristic symptoms of

Alzheimer's appear, significant and often irreversible damage has already occurred.(3)

Blood: The Silent Witness and Forensic Archive

Alzheimer's is a relentless and insidious disease, and whilst protein aggregation has long been scrutinized as a primary suspect, it may not be acting alone. (4) Like a skilled detective looking beyond the obvious leads, researchers are broadening their investigative scope. One promising avenue of exploration lies in the blood. Traditionally, brain scans have been the primary tool for studying neurodegenerative changes. However, these imaging techniques are costly, and time-consuming. (5) Analysis of cerebrospinal fluid (the fluid protecting and nourishing the brain and spinal cord) is another valuable diagnostic approach, but its extraction requires an invasive procedure performed by a specialist, limiting its widespread application. In contrast, blood sampling offers a minimally invasive, cost-effective, and scalable alternative for clinical use. (6)

The bloodstream can be thought of as a forensic archive, preserving molecular evidence of neurodegeneration. It holds crucial information - from clues about misfolded proteins that accumulate in the brain to indicators of neuroinflammation and metabolic dysfunction. (7) By analyzing these subtle biochemical changes, scientists can gain deeper insights into the underlying mechanisms of dementia, paving the way for earlier and more accessible diagnoses.

Expanding the Suspect List

Modern dementia research acknowledges that neurodegeneration is an intricate and multifaceted process. Therefore, by looking for novel indicators of inflammation, immune dysregulation and metabolic shifts, scientists are hoping to construct a more comprehensive understanding of how the disease develops and progresses. (7) These additional molecular signals, akin to forensic traces at a crime scene, may provide early insights into subtle biological changes that precede cognitive symptoms. (8) Just as a detective meticulously examines a crime scene for overlooked details -footprints in the dust, a misplaced object, or a hidden fingerprint, scientists are scrutinizing blood for elusive biomarkers that could signal neurodegeneration in its earliest stages. Tracking these changes over time may one day allow us to not only predict an individual's risk of cognitive decline but also tailor interventions that could alter disease trajectory before irreversible damage occurs. (9)

However, as with any complex case, numerous challenges remain. Distinguishing meaningful biological signals from irrelevant background noise is no easy feat. The aging process itself complicates the picture, as elderly individuals often exhibit some degree of neurodegeneration and inflammation independent of disease. (10) Determining which molecular changes are truly indicative of pathology rather than normal aging remains an ongoing challenge. Additionally, underlying health conditions can further obscure biomarker patterns. (10) To address this, scientists are continuously refining analytical techniques, working to minimize variability between individuals and standardize laboratory methods for assessing blood-based biomarkers. As this research advances, the hope is that we will move closer to a future where neurodegenerative diseases can be identified and consequently treated at their earliest stages.

A Paradigm Shift in the Courtrooms of Clinical Neuroscience

With the techniques used to study markers of brain dysfunction becoming increasingly sophisticated, we find ourselves shifting away from reactive diagnostics, treating the aftermath of neurodegeneration to proactive, predictive strategies focused on early detection and prevention. In this new era, blood is not merely a conduit of life-sustaining nutrients but functions as a detective's notebook, chronicling the subtle biological changes that characterise dementia's presence and precede the onset of the disease.

Just as forensic science has transformed criminal investigations, blood biomarker research is poised to revolutionize dementia diagnosis. History is filled with moments when a single discovery reshaped the course of an entire field - DNA fingerprinting dramatically altered forensic investigations, and blood-based diagnostics may similarly redefine our approach to dementia. The answers we seek are not buried in unsolved case files but are flowing through us, carried by the very substance that sustains life. Like Poirot, we approach our work with curiosity and determination, knowing that the next crucial discovery may already be hidden in plain sight -perhaps, within a single drop of blood.

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UK Bioimaging Launches User Access Fund for Researchers

A new five-year initiative provides up to £5,000 per application for access to cutting-edge bioimaging facilities

UK researchers now have increased access to state-of-the-art bioimaging technologies through a new fund announced by the UK Node of Euro-BioImaging. The UK Bioimaging User Access Fund, supported by £1.8 million in strategic funding from UKRI-BBSRC and UKRI-MRC over five years, aims to democratise access to advanced imaging facilities across the country and beyond.

The fund offers up to £5,000 at 100% Full Economic Cost (FEC) for bioimaging hardware access, or up to £2,000 for image analysis services only. This investment covers all aspects of facility access, including consumables, travel and accommodation expenses, and comprehensive technical support and training.

"This funding represents a significant opportunity for UK researchers across all biological disciplines," explains Georgina Fletcher, UK Node Manager. "We're removing financial barriers that might prevent researchers from accessing the cutting-edge imaging technologies they need to advance their science."

The UK Node comprises 14 facilities across seven sites, offering access to an impressive range of advanced biological imaging techniques. Researchers can access correlative, multi-modal, high-content, and super-resolution microscopy, along with expert image analysis services. Six of these sites are eligible for this funding opportunity: ES-RIC (Edinburgh Super-resolution Imaging Consortium), the University of York's Imaging and Cytometry Facility, multiple centres at the University of Liverpool, Oxford Brookes University's Centre for Bioimaging, several imaging centres at King's College London, and The Francis Crick Institute's CALM facility.

If the required technology is not available within the UK, the fund also supports access to more than 200 Euro-BioImaging facilities across Europe, ensuring UK researchers can access exactly the expertise and equipment their projects demand.

Beyond equipment access, successful applicants receive comprehensive support throughout their projects, including initial consultation and experimental planning, hands-on training, sample preparation assistance, and high-quality data acquisition services.

Applications are reviewed on a rolling basis every four months, with evaluation dates on 31 March, 31 July, and 30 November. For the inaugural round, the deadline has been extended to 9 December 2025, with decisions expected within four weeks of each review cycle. Projects will be assessed based on technical feasibility and scientific merit.

There is one mandatory requirement: researchers must discuss and agree their experimental plan—including feasibility, cost, and timeframes—with their chosen host facility before submitting an application. This ensures projects are well-planned and technically viable from the outset.

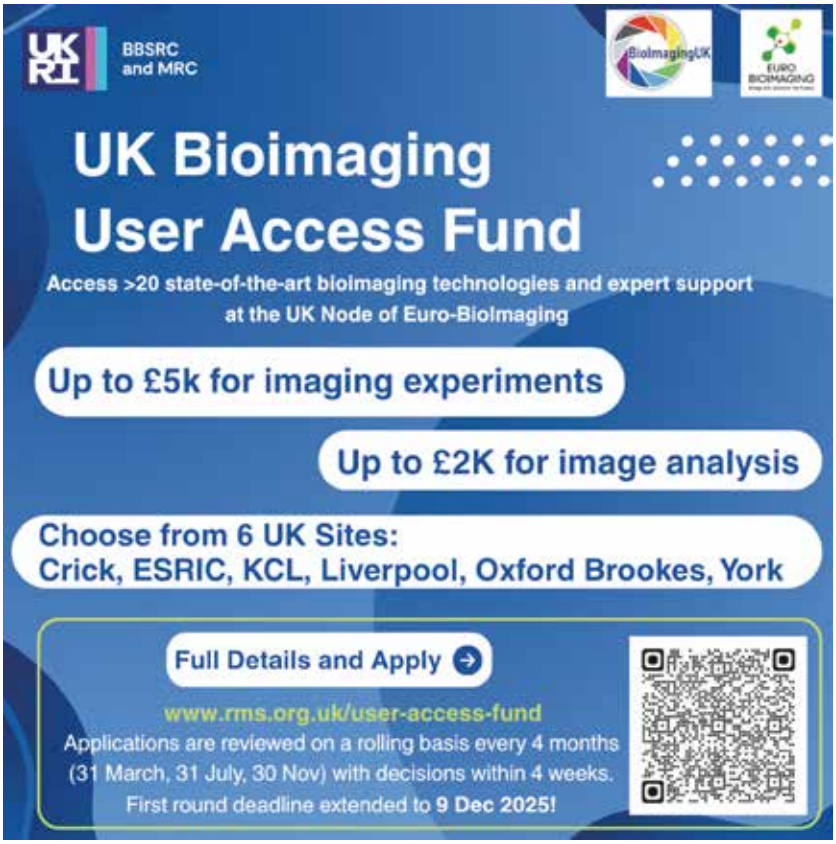
"We encourage researchers to reach out early to discuss their imaging needs," adds Fletcher. "Our facilities are ready to help develop robust experimental plans that will make the strongest applications."

For informal enquiries or guidance on which facility might be most suitable for specific research needs, researchers can contact info@eurobioimaging.eu or georgina@rms.org.uk.

Full details about the funding opportunity, eligibility criteria, and the application process are available at www.rms.org.uk/user-access-fund.

Contact: georgina@rms.org.uk

The CLF OCTOPUS facility at Harwell is excluded from this scheme as it already benefits from dedicated UKRI-funded user access arrangements through the Central Laser Facility.



Meet the BSCB Committee

Mattéa Finelli

Dr Mattéa Finelli is an Assistant Professor at the University of Nottingham. The Finelli group (mattea-finelli-lab.com) studies the molecular mechanisms of neurodegenerative diseases including amyotrophic lateral sclerosis and Alzheimer's disease. It focuses on how changes in structure, interaction, and activity of proteins of the brain can impact cellular function in health and in neurological conditions. To address these questions, the Finelli group uses a combination of cellular models (cell lines, primary, stem cell-derived models), gene editing, functional and recombinant protein-based assays, as well as in silico approaches (drug screening, protein modelisation).

Mattéa received a MSc in bioengineering from SupAgro (France), and a MRes in Biomedical Research from Imperial College London, before being awarded her DPhil from the University of Oxford. She completed her postdoctoral work at Mount Sinai School of Medicine (New York) and the University of Oxford. She started her independent research at the University of Nottingham in 2020 with an Anne McLaren Fellowship.

Mattéa joined the BSCB Committee in 2024, and is currently co-Editor of the BSCB Magazine.

Ye Dee Tay

"I am a lecturer in Biomedical Sciences at Cardiff University, with a particular focus on the scholarship of teaching and learning. My teaching focuses on genetics, cell biology, and molecular biology. I am committed to delivering research-informed, inclusive education that supports students in developing a strong conceptual understanding of biological principles.

I grew up on Borneo Island in Malaysia and came to the UK to study Biomedical Sciences at the University of Hull. Inspired by my final-year research project, I decided to pursue a career in research and was awarded a Cancer Research UK PhD Studentship to study Medical Oncology at the University of Oxford. This transformative experience introduced me to the molecular and cellular basis of disease, while also providing a broader exposure to basic science research. I subsequently undertook postdoctoral research at the Paterson Institute (now the CRUK Manchester Institute) with Prof. Iain Hagan, where I studied cell cycle regulation in fission yeast. I later moved to the Wellcome Trust Centre for Cell Biology at the University of Edinburgh, where I worked with Prof. Ken Sawin and Prof. Andrew Goryachev on Cdc42 GTPase–mediated cell polarity by integrating fission yeast cell biology, genetics, and advanced microscopy.

Alongside my research career, I have always been enthusiastic about teaching. During the COVID-19 pandemic, I took the opportunity to formally develop my teaching practice by completing a Postgraduate Certificate in Academic Practice and was subsequently awarded Fellowship of the Higher Education Academy. As the first person in my family to attend university, I am particularly committed to widening participation and supporting students for whom higher education or academia may not feel like a natural or accessible path.

I have been a member of the BSCB since my early career and have long valued the Society's role in supporting the UK cell biology community. I am delighted to be able to give back by contributing to the BSCB, particularly through promoting excellence in cell biology education, supporting early-career scientists, and strengthening links between teaching and research within the discipline."

Nathalie Signoret

"I am a molecular cell biologist and academic leading research on a set of molecular sensors called chemokine receptors. These are essential components of our immune system, coordinating host cell responses and implicated in numerous pathologies, including cancer.

I completed my undergraduate, master's, and PhD training at the University of Aix–Marseille II (France). During my PhD in Immunology at the Centre d'Immunologie de Marseille-Luminy (CIML), in the laboratory of Prof. Quentin Sattentau, I investigated the role of the glycoprotein CD4 in HIV infection. I subsequently undertook postdoctoral training at the MRC Laboratory for Molecular Cell Biology (MRC-LMCB) in London with Prof. Mark Marsh, where I developed a strong interest in chemokine receptor biology. My work focused on the molecular mechanisms regulating the surface expression and intracellular trafficking of the HIV co-receptors CXCR4 and CCR5. in 2006 I joined the Hull York Medical School and established my independent research group at the University of York, spreading my expertise to immunological and cancer aspects of chemokine receptors molecular regulation.

My research is highly interdisciplinary, combining molecular cell biology with approaches from physics and chemistry to develop innovative tools to address biological questions. I also work closely with bioinformaticians and clinical teams on translational studies exploring chemokines and their receptors as potential biomarkers in disease."

Meeting reports

Cell Death UK meeting

6 October 2025. CRUK, Scotland Institute, Glasgow

CJ Anderson and Andrew Davidson are two junior group leaders who have recently established labs in Glasgow and Edinburgh, respectively. They recognised that there was no meeting to support the vibrant UK cell death community, despite the wealth of European and transatlantic conferences on the topic. They felt this omission was especially pressing for Early Career Researchers, depriving them of a platform to interact with the UK community and of opportunities to share their research. Inspired by the long-running, BSCB sponsored Actin UK meeting held in Bristol, they decided to host a conference dedicated to fundamental research in cell death.

"We thought that Scotland in particular was a particularly apt place to hold the first meeting as it has played a historical role in cell death research, including the original recognition of apoptosis at University of Aberdeen by Kerr et al., as well as the seminal discoveries that came out of the Centre for Inflammation Research, University of Edinburgh. Therefore, having secured the support of the CRUK Scotland Institute to host the meeting, we welcomed 73 attendees to the glorious autumnal Garscube estate, in the less-than-glorious Glaswegian weather.

After welcomes and introductions, we kicked off the meeting in the Robertson Trust lecture theatre with our ThermoFisher sponsored keynote lecture (40 min + 15 min Q&A) by Prof. Kodi Ravichandran, WashU. Prof Ravichandran is a world-leading expert in the clearance of cell death ("efferocytosis") and delivered a talk titled "The beauty & complexity of cell death & efferocytosis". This was a brilliant overview of the field in general, as well as highlighting the many significant contributions his lab has made. This included studies that have recently come out of the Ravichandran lab, such as the role of Death Induced Nutrient Release fuelling microbe dysbiosis in the gut as well as the importance of transcriptional

unpausing in supporting macrophage efferocytic capacity, as well as exciting unpublished work. Following an engaging Q&A session, we moved swiftly on to our 3 min ECR flashtalks, where 10 selected students and post-docs from across the UK were given the opportunity to advertise their posters. After the close of this session, we braved the Glaswegian weather (which kindly took pity on us) and took a 5 min walk through the estate to the Mary Stewart building for a 2 hour lunch and poster session. This offered far more space to accommodate lunch, table seating and the poster session itself. Following the conclusion of the poster session and a further reprieve from the rain during our walk back to the CRUK Scotland Institute, we resumed our programme of scientific talks in the Robertson Trust lecture theatre. This second session was sponsored by Assay Genie and was entirely dedicated to ECR presentations. Our first ECR short talk (15 min + 5 min Q&A) was presented by Nathalia Moraes de Vasconcelos from ICR, London, on her work on SUMOylation inhibition as a potential anti-cancer strategy to trigger immunogenic cell death, via mobilisation or transposable elements. Next, we had Lyndsey Flanagan from the CRUK Scotland Institute presenting how sublethal mitochondrial outer membrane permeabilisation promotes caspase-mediated DNA damage, contributing to oncogenic mutation during cancer. Third up we had Julien Mambu from King's College London who has been studying the complicated role of type III interferons in the intestine. The final talk of this session was given by Michael Rimmer from the University of Edinburgh, a clinical academic investigating how extracellular vesicles released in the pre-pubertal testis during chemotherapy might contribute to infertility via intercellular transfer of components of the apoptotic pathway.

After a short coffee break we returned with David Salvador-Garcia, King's College London, who presented his live-imaging of necrotic opsonins during wounding of the Drosophila embryo and how this contributes to efficient wound clearance and repair. Next, Benjamin Raymond from University of Newcastle, shared his work on the proteomic analysis of efferocytic macrophages and his identification of novel player required for efficient uptake of apoptotic debris and wound healing. Finally, Sam Benson, University of Edinburgh, brought the short talks to a close with a presentation on how bacteria hijack purines released during death-induced nutrient release within the gut, which can fuel dysbiosis. To finish a great day of presentations, we ended with our ECR plenary talk (40 min + 15 min Q&A), awarded to Rhona Christie, from University of Glasgow, who presented her work on how Limited availability of selenium within the spinal fluid may offer a vulnerability to ferroptosis as a way to stop leukaemia establishing itself in the CNS. It was incredibly difficult for the selection committee to decide our ECR plenary talk from amongst all the submitted abstracts. However, Rhona's abstract was selected as it represented a great example of fundamental research in cell death being translationally applied for potential clinical benefit. After closing the meeting, we enjoyed a drinks reception sponsored by Atlantic Imaging Ltd, before those that could make it joined us for a meal in the Bothy Restaurant in the West End of Glasgow.

In total, we were thrilled to be able to offer 18 speaking slots to ECRs- an opportunity that is exceptionally rare across science (not just the cell death field). Each submitted abstract was independently reviewed by 4 judges, and total scores (assessing for clarity on impact, novelty, and methodology) combined to select applicants. We were very happy to have

achieved career stage (PhD student, PDRA, clinical fellow), geographic, and sex/gender diversity amongst our selected speakers in an independent and unbiased way. We have solicited feedback from all participants, and have received particular enthusiasm for 1) giving ECRs an opportunity to present their work, 2) having a diverse speaking panel, and 3) giving preference to multiple talks (15 min + 5 min Q&A) over fewer longer talks. There is resounding enthusiasm amongst the ECRs for this event to be held annually. We have now included Dr. Rebecca Coll (Queen's University Belfast) as a new co-organiser and have begun preparations for 2026.

Overall, as co-organisers of this inaugural meeting, we were most pleased with the buy-in from the cell death community, with researchers joining us from across the whole of the UK. We'd like to thank the generosity of our commercial sponsors (ThermoFisher, Assay Genie and Atlantic Imaging Ltd) as well as the BSCB for their support to make this meeting possible. Onward to next year and Cell Death UK 2026!"

CJ Anderson and Andrew Davidson

Biologists @100

24–27 March 2025. Liverpool

The Company of Biologists celebrated its 100-year anniversary in 2025. The Biologists @ 100 conference, hosted in Liverpool on 24–27 March 2025, was a major event in their plans.

As the scopes of our journals span across diverse fields of biology, it was important for us to host a unique conference that brought together all our communities. The conference was organised with three societies, including the British Society for Cell Biology (BSCB) along with the British Society for Developmental Biology (BSDB) and the Society for Experimental Biology (SEB), all which we have a long historical association with. Given that two of the societies are UK-focused, it's no surprise that most attendees were from the UK, but altogether we hosted 585 attendees from 27 countries, representing 6 continents.

Over four days, we had 76 speakers. Add to the mix 286 posters, an image competition, a sustainability zone, five Editor meetings, slido polls, delicious vegan and vegetarian food, author focus groups, 'Have your say' tables, 10 sponsors and 14 exhibitors, framed selfies and an artist creating a mural live for us to bring back to our office in Cambridge.

On Monday, we kicked off the conference with a great first day featuring an SEB satellite meeting focused on 'Experimental biology: solutions to climate change and biodiversity loss', a career session for 130 early-career researchers, the conference launch and a welcome reception at The Museum of Liverpool, UK.

On Tuesday, our focus on climate change continued with plenaries on biodiversity and climate change as well as a 'silent theatre' session on climate change. Furthermore, the second day of the conference also launched the cell and developmental biology sessions and the first poster session.

Wednesday featured plenaries on health and disease, a one-day Disease Models & Mechanisms (DMM) strand on combatting antimicrobial resistance, a 'silent theatre' session on the future of scientific publishing and our second poster session. A science-filled day was followed by our gala dinner at the beautiful St George's Hall, UK, and the compulsory disco feature at the annual BSCB and BSDB spring meetings.

The last day of the conference featured plenaries on emerging technologies, the final scientific sessions which included the BSCB Hooke, BSCB Postdoc and BSDB Beddington medal talks, and a panel discussion pulling lots of subject themes together, before the delegates headed home (almost all by train, in line with our sustainability aspirations for the conference which you can read more about).

This is the first time we get to celebrate with all our communities simultaneously so thank you to all of our attendees and sponsors and thank you to our wider community members who were not able to be present – our readers, authors, peer reviewers, community site members – who have made it possible to inspire biology for the past 100 years. To revisit – or view for the first time - you can find videos from the conference on The Company of Biologists' YouTube channel.

1st Scottish Cell Biology Meeting

6 October 2025. CRUK, Scotland Institute, Glasgow

The first-ever Scottish Cell Biology Meeting (SCBM), sponsored by the British Society for Cell Biology (BSCB), was held at the University of Dundee on 18 September 2025 and proved to be a tremendous success. Designed to bring together early career researchers from across Scotland, the meeting created an exciting and inclusive space for collaboration, knowledge-sharing, and the celebration of outstanding cell biology research.

The programme showcased the breadth and quality of cell biology being carried out across Scotland, with 120 attendees (50% Dundee, 26% Glasgow (including Strathclyde and cancer institute), 14% Edinburgh, 5% Saint Andrews, 4% Aberdeen, 1% other). Masters, PhD, post-docs represented 59% of attendees and 100% of our speakers (aside of the guest speaker). From inspiring research talks to lively flash presentations and dynamic poster sessions (50 posters in total), participants highlighted world-class science and engaged in stimulating discussions that fostered new connections and ideas. The strong representation of early career researchers was a particular strength of the meeting, reinforcing its role as a platform for the next generation of scientists.

The meeting was officially opened by Sir Mike Ferguson, whose introduction of Professor Ian Ganley, recipient of the 2025 BSCB Hooke Medal, set the tone for an engaging and high-calibre scientific event. Congratulations are due to the winners of the best presentations, best flash talks, and best posters, whose work stood out among an exceptionally strong field. We also gratefully acknowledge all sponsors, whose generous support was instrumental in making this meeting possible.

Sincere thanks are extended to the local volunteers and co-organisers — Dr. Marta Murray, Dr. Xiaohan Li, and Dr. Matthew Watt—for their dedication and excellent organisation, which ensured the smooth running of the day.

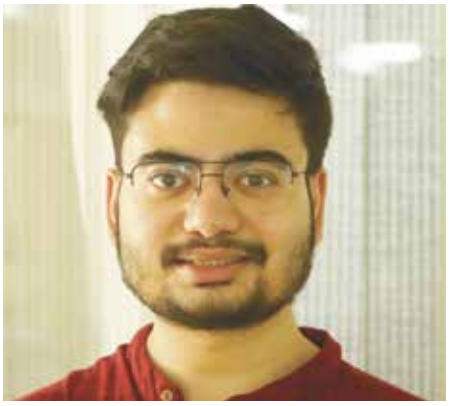
With enthusiasm already building, we look forward to the next Scottish Cell Biology Meeting, to be held in Glasgow on Friday, Sep 4th. Further details to follow in due course, with registration opening Spring 2026.

The future of Scottish cell biology is undoubtedly bright.

The organisers: Carine de Marcos (UoD), David Murray (UoD), Noor Gammoh (UoE), Riko Hatakeyama (UoA), Judith Sleeman (UoSA) and Stephen Tait (UoG).

BSCB PhD Best Poster Prize: Jashaswi Basu

Jashaswi Basu is a final-year PhD student at the Indian Institute of Science Education and Research Pune where he studies the collective behaviour of microtubule dynamics and transport and how biophysical and biochemical factors shape them. Jashaswi won the BSCB PhD Best Poster Prize at the Biologists@100 conference.



"As a doctoral student, I was incredibly fortunate to attend Biologists@100, where I had the opportunity to connect with biologists working across diverse fields and to join in the global celebration of life sciences. It was my first international conference where I presented my research to a global audience.

At the conference, I presented my PhD work on how the crowded cell cytoplasm influences microtubule polymerization dynamics *in vitro*. While microtubule (MT) growth at a single filament has been reported to be crowdant size dependent, its influence on collective MT polymerization in vitro which comprises both nucleation and growth remains unclear. Using bulk turbidimetry, we observed polymerization rates of brain tubulin in presence of stabilised microtubule 'seed's decreased for small crowdants but increased for large crowdants. However, in the absence of 'seed's (de novo), polymerization rates increased irrespective of crowdant size, with a corresponding decrease in critical concentration. Using label free microscopy we report that while microtubule density increased independent of crowdant size, microtubule lengths decreased only for small crowdants. This confirmed the differential effect of crowdant size on MT nucleation and elongation.

In a parallel project, I have been also working on the evolutionary divergence of tubulin polymerization kinetics. Tubulin sequence, despite being highly conserved shows significant kinetic divergence across species. In a previous study from the lab, we reported activity purified mung tubulin to show transient polymerization kinetics, lower critical concentration, and shorter microtubules than brain tubulin, with simulations attributing this divergence to a higher GTP hydrolysis. However due to the low yield of mung tubulin, we could not test this experimentally.

Recently, using TOG-based affinity chromatography, I purified higher yields of mung bean tubulin and demonstrated experimentally a ~11x higher GTP hydrolysis rates compared to goat brain. Currently we are trying to explain the kinetic divergence using a combination of structural and theoretical predictions.

In my doctoral thesis, I have been able to elucidate how biophysical factors like crowding and evolutionary divergence in tubulin shape microtubule polymerization. These findings contribute to a deeper understanding of the complex cytoskeletal dynamics in cells. Once again, I am really grateful to the British Society for Cell Biology for honouring my work at Biologists@100, and to the scientific community for such a rewarding first international experience. Looking ahead, I am excited to further continue exploring microtubule dynamics and their role in cellular function."

BSCB Postdoc Best Poster Prize: Asifa Islam

Asifa Islam is a postdoc at the University of Nottingham who studies intracellular transport of organelles. She won the BSCB Postdoc Best Poster Prize at Biologists@100 conference.

"Winning the Best Postdoc Poster Prize at the Biologists@100 conference was both an honour and a moment of personal reflection. The award, which included a £1000 travel grant, enabled me to attend the Biochemical Society's meeting Small G Proteins in Cellular Signalling and Disease, where I had the privilege of delivering a 15-minute talk in a session alongside senior and established researchers, including professors whose work I have long admired.

My journey to this point has been shaped by movement across countries, disciplines, and career paths. I was born in Pakistan and raised in Saudi Arabia, before returning to Pakistan for my medical degree. While I

enjoyed clinical medicine, I found myself increasingly drawn to the molecular intricacies of cell biology. That curiosity brought me to the UK, where I completed an MRes in Oncology at the University of Manchester, a PhD in Cell Biology at Queen Mary University of London, and now work as a postdoctoral researcher at the University of Nottingham.

The research that earned me the BSCB award focuses on how cells transport organelles within their internal space – a deceptively simple process that depends on finely tuned interactions between cytoskeletal tracks, motor proteins, and small GTPases. In pigment-producing melanocytes, for instance, Rab27a plays a crucial role in dispersing melanosomes by recruiting both motor proteins, such as myosin Va, through Rab effectors like melanophilin (MLPH) and actin assembly factors, including the SPIRE family of nucleators. What continues to intrigue researchers is how small GTPases like Rab27a coordinate these molecular networks. While the Rab27a–MLPH-myosin Va interaction is well characterised, its interaction with SPIRE-type actin nucleators remains an emerging area of interest. My work investigates how Rab27a may regulate actin assembly at organelle membranes, using a combination of live-cell imaging, cellbased assays, and quantitative analysis. Understanding these mechanisms can deepen our knowledge of intracellular trafficking and potentially inform research into pigmentation disorders and other diseases involving organelle mislocalisation.

Attending the Biochemical Society's focused meeting on small G proteins was a fantastic opportunity. The conference brought together researchers from across Europe, the US, and Asia to explore how GTPases influence processes ranging from development to disease.

Speaking in a session on G protein functions in cell migration, adhesion, and morphology, alongside senior academics, was a highlight of my postdoctoral career. The discussions, feedback, and fresh perspectives I encountered were truly enriching.

This award not only supported my travel but also boosted my confidence in sharing my work on an international stage. I'm deeply grateful to the BSCB for recognising the efforts of earlycareer researchers and for fostering such a vibrant and supportive scientific community. For me, this experience reinforced the idea that scientific journeys – like cellular ones – depend on dynamic connections, unexpected directions, and steady forward movement."

BSCB Travel Awards

The recipients of the BSCB Travel Award share how this Award benefited their research and their development as scientists.

Imogen Ramsdenheld received a BSCB Travel Award to attend the Biologists @100 Conference

"At the Biologists @100 Conference (24–27 March), I had the privilege of presenting and discussing my PhD research on novel mechanisms of Arf GTPase regulation. My work focuses on identifying phosphorylation-dependent protein-protein interactions of Arf1 and Arf6, which could expand the canonical view of their regulation beyond GTP-binding and hydrolysis controlled solely by GAPs and GEFs. Using phosphomimetics and proximity labelling, I've uncovered potential phosphorylation-dependent interactors that may represent these unexplored additional states. Sharing this work during the poster sessions led to insightful conversations with scientists from diverse backgrounds, which gave me new ideas for refining my experimental plans and helped me to better see the broader biological context.

I also gave a flash talk during the conference, which was an intense but rewarding exercise in communicating complex science in under two minutes. It pushed me to prioritise clarity, impact and storytelling—skills I will value in all my future presentations whatever their length. I greatly appreciated the questions, interactions, and new connections that arose from both formats, and it was a pleasure to receive so much interest at my first large conference.

Beyond presenting, I was energised by the many varied talks. While I primarily followed the Cell and Developmental Biology track—attending excellent sessions on cell division and proliferation, organelle dynamics, cell migration and communication, and conceptual frontiers—I also took the opportunity to branch out into the one-day interdisciplinary Antimicrobial Resistance programme hosted by Disease Model & Mechanisms. These sessions brought a fresh perspective and cutting-edge methods for translating fundamental cell biology into urgent challenges like drug discovery and environmental monitoring.

I noted the strong sustainability theme throughout the conference, highlighted in plenary lectures and the additional lunchtime 'silent theatre' session. The latter suggested practical actions for reducing plastic waste, optimising energy use (e.g. -80°C freezers) and exploring animal-product alternatives, while also openly discussing the financial and reputational challenges of pioneering new methods and maintaining reproducibility.

A real highlight for me was the Early Career Researcher (ECR) session that kicked off the conference. We were able to interact with professionals working in publishing, biotech, and science communication. Hearing about their varied career paths was both encouraging and eye-opening, reminding me that impactful scientific careers can take many forms beyond the academic lab.

Attending the conference gave me valuable feedback on my work and a platform to build new connections both within the UK and internationally. The scale and inclusivity of the event made it easy to have meaningful conversations across career stages and specialisms, exchanging ideas that will directly improve my research. I left feeling more confident in presenting and discussing my science.

I am grateful to the BSCB for supporting my attendance with the Honor Fell Travel Award; this conference was a fantastic opportunity to grow as a scientist and forge new connections."

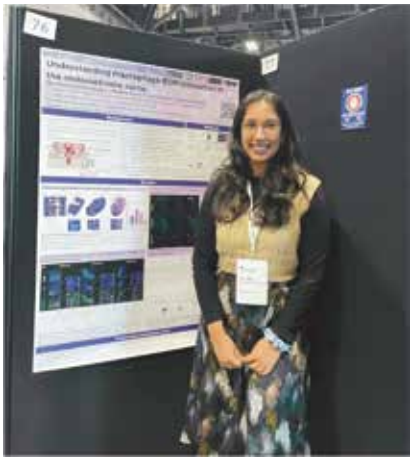
Niky Moolchandani Adwani received a BSCB Travel Award to attend the Biologists @100 Conference

"I'm a PhD student at the University of Warwick, funded by the Medical Research Council (MRC), and my research focuses on understanding the interactions between macrophages and the extracellular matrix (ECM) in endometriosis lesions. I recently presented my work at the Biologists 100 Conference in Liverpool!

My project investigates how macrophages adapt to their environment, altering their phenotype and behaviour in ways that contribute to endometriosis progression. I'm particularly interested in how the physical properties of tissues, such as stiffness and collagen content influence macrophage behaviour. I'm also exploring how macrophages migrate on different substrates and respond to chemokines to better understand their activity within the ECM. This research could uncover key mechanisms behind endometriosis and help identify potential targets for future therapies.

Attending the Biologists 100 Conference was a fantastic opportunity to share my findings and connect with researchers in the field. I particularly enjoyed the wide range of talk topics and learning about exciting developments across biology. The feedback I received was incredibly helpful, and I left feeling more confident and inspired to pursue new ideas in my work.

I'm very grateful to the BSCB for awarding me a travel grant to attend the conference. It's been an excellent opportunity and great experience to attend such a vibrant event and learn so much from the community! "



Martin Grundy received a BSCB Travel Award to attend the Biologists @100 Conference

"It was a pleasure to present my research at the Biologists@100 conference in Liverpool earlier this year, an opportunity afforded to me by generous travel funding from the BSCB. Coming from a background in leukaemia research and previously restricting myself to mainly haematology-based meetings I was excited to attend a conference with a much broader biological research theme. Biologists@100 more than lived up to my expectations with the initial plenary lectures both fascinating and inspiring, setting the tone for the rest of the conference. The evening poster sessions were very well attended and interactive providing valuable feedback on my own research and the opportunity to explore, and gain ideas from other attendee's novel work. The quality and diversity of the research presented throughout the conference was incredible.

The background to the research I presented is that mutations in the Nucleophosmin (NPM1) gene represent the most common genetic alteration in Acute Myeloid Leukaemia (AML) and result in mis-localisation of the mutated protein from a predominantly nucleolar localisation to a predominantly cytoplasmic distribution. Historically, most studies of NPM1 mutated AML have focussed on this aberrant cytoplasmic localisation of the mutated protein although efforts to reverse this mis-localisation therapeutically have so far resulted in limited clinical benefit. We used high resolution imaging to demonstrate that NPM1 is crucial for maintaining normal nucleoli architecture and specifically the integrity of the nucleoli rim, the least understood nucleolar compartment. We demonstrated that cell lines and primary cells with NPM1 mutations from individuals with AML have aberrant nucleoli architecture and that intriguingly, this abnormal nucleolar phenotype is reversible. Using a surrogate for rRNA synthesis, we showed that the aberrant phenotype is associated with differences in nucleolar function, specifically that activity of RNA polymerase I is increased in NPM1 mutated cells. Using EM, we demonstrated that perinucleolar chromatin organisation is also markedly different in NPM1 mutant cells. Finally, we reported the novel finding that the NPM1 mutated protein forms distinct aggregates and characterised these for the very first time. More recently, others have described these aggregates as condensates that drive aberrent gene transcription and maintain NPM1 mutated cells in an undifferentiated, leukaemic state. Our work revealed how nucleolar organisation contributes to the molecular mechanisms underpinning NPM1 driven AML revealing novel therapeutic vulnerabilities.

A fulfilling and well organised conference in a great location, I look forward to returning in the future."

Apolline Delahaye received a BSCB Travel Award to attend Biologists@100

"Morphogenetic processes are shaped by tissue-extrinsic boundary conditions (mechanical, chemical, geometrical), but how these constraints influence tissue organization and cell behavior remain unclear. Studying these interactions in vivo is particularly challenging due to the embryo's inherent complexity and limited accessibility. To overcome these limitations, we use a simplified and highly reproducible stem cell-based model known as gastruloids. Gastruloids mimic key aspects of early embryonic development, including germ layer differentiation and body axis formation. This model not only circumvents the technical and ethical challenges associated with using embryos but also offers a tightly controlled experimental system, allowing us to systematically manipulate individual parameters and dissect their impact on morphogenesis.

In the work I presented at BSCB, we investigated how cell-substrate interaction affects cell differentiation and morphogenesis by comparing traditional 3D gastruloids with gastruloids plated on defined extracellular matrix (ECM) substrates. Laminin coating promotes strong attachment and flattening of gastruloids. Upon mesoderm activation, 3D gastruloids form a single T/Bra-expressing pole and elongate through cell–cell interactions. Instead, flattened gastruloids develop multiple T/Bra-expressing poles forming tightly connected cell streams elongating from the main body, likely through substrate-based migration. Gene expression analysis shows both exhibit comparable differentiation markers. However, while 3D elongation presumably relies on cell-cell traction, laminin gastruloids elongate through substrate based collective cell migration involving dynamic cellular protrusions (filopodias, lamelipodias). Inhibiting those protrusions show that lamellipodia restrict elongation on laminin, likely by generating unproductive lateral forces, while filopodias are essential, potentially by transmitting mechanical /signalling cues to the follower cells. Notably, 3D gastruloids are unaffected. Together, this suggests that cell behaviours associated with their respective elongation mechanisms differ. This work provides insights into how physicochemical constraints impact morphogenesis and reveals how gastruloids can achieve similar developmental outcomes through distinct strategies depending on tissue-extrinsic cues.

Attending the BSCB meeting was a valuable experience. It allowed

me to present my work, receive constructive feedback, and discuss ideas with others from the field. I also truly appreciated the chance to hear about other projects from more distant research areas! As an early career researcher, I attended the ECR session on the first day, which was a great opportunity to learn about the range of career paths available. Overall, it was an enriching and inspiring experience, and I came back with many new ideas and connections."

Theano Kyriakou received a BSCB Travel Award to attend the Gordon Conference on Adhesion and Growth Factor Receptors in health and disease

"I was very grateful to receive travel funding from the BSCB to attend both the Gordon Research Seminar (GRS) and Gordon Research Conference (GRC) on Adhesion and Growth Factor Receptors in Health and Disease, which took place at the Southern New Hampshire University in July 2024. At these meetings, I presented my PhD research titled "Crosstalk mechanisms in lung fibrosis: Galectin-3 regulates integrin αVβ6 proteostasis and mechanotransduction in fibrotic cells."

My work focuses on understanding how Galectin-3 modulates the trafficking and proteostasis of integrin αVβ6 in lung epithelial cells - a critical axis in the progression of lung fibrosis. We are particularly interested in how this crosstalk influences TGF-β activation which is a key step in the initiation and progression of the disease. Our data reveal a functional relationship between Galectin-3, EGFR, and integrin αVβ6. Most importantly, we demonstrate that Galectin-3 acts as a novel regulator of integrin αVβ6, directly binding to αVβ6 to control its trafficking and proteostasis. A feed-forward model is proposed, whereby galectin-3 promotes TGF-β- dependent αVβ6 expression, which further promotes αVβ6-mediated TGF-β activation, to nucleate and propagate fibrotic foci formation. The mechanism by which galectin-3 amplifies αVβ6-mediated TGF-β activation may ultimately serve as a novel therapeutic target or prognostic indicator in IPF.

The GRS provided a supportive setting to present my work among early-career scientists, fostering open discussions and helpful feedback. The subsequent GRC allowed me to connect with leaders in the field and engage in deeper conversations about shared mechanisms of adhesion signalling across tissues and disease contexts. A particular highlight was the mentorship panel at the GRS, led by early career researchers at various stages. Their reflections on careers in academia, publishing, and beyond helped clarify potential paths after my PhD and gave me confidence to explore diverse opportunities.

Thanks to the BSCB's generous support, I was able to fully participate in both of these inspiring meetings. I returned with fresh ideas for my project, valuable professional connections, and renewed motivation for the next stages of my research career."

Summer studentships

The BSCB Summer Vacation Studentships offer financial support for high calibre undergraduate students, who wish to gain research experience in cell biology during their summer vacation. Our aim is to encourage students to consider a postgraduate research career in cell biology after their undergraduate studies. The application form and complete information about the scheme are available on the BSCB website.

The latest recipients of the BSCB Summer Vacation studentships explain what they have been up to and learned during their time in the lab.

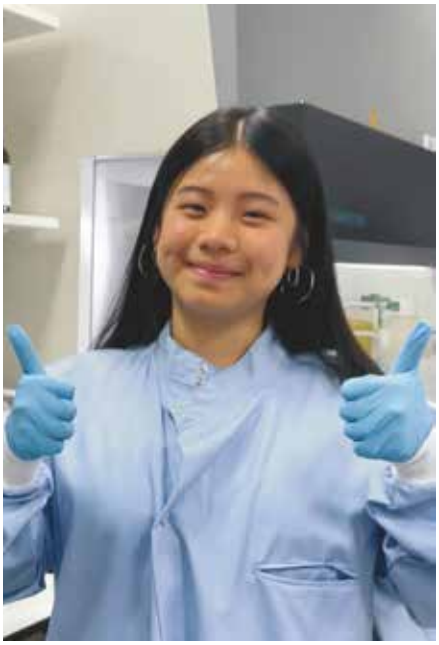
Characterizing the PuffHalo neighbour labelling system in human embryonic stem cells (hESCs)

Natalie Chan held a studentship with Prof Sally Lowell at the University of Edinburgh

"As I wrapped up my final-year project which, at just three months, felt far too brief. I was eager to gain more hands-on research experience before deciding on my next steps. Thanks to the BSCB and the Lowell Lab, I was able to extend my time at the bench and immerse myself in a full-time research environment over the summer.

After previous experiences in both clinical and basic research related to cancer and early embryo development, these sparked my interest in regenerative medicine and cancer therapeutics. I chose the Lowell Lab at the Centre for Regenerative Medicine because of its focus on cell communication. That fascinated me as cell communication is a fundamental process that governs development, homeostasis, and disease progression, thus lies at the heart of many of my scientific questions. I was particularly intrigued by the lab's innovative neighbour-labelling technologies. These cutting-edge tools offer new ways for researchers to explore how cells interact within their environments. They work by marking or labelling cells based on their proximity to a target or "sender" cell, then allowing us to track molecular changes in both sender and neighbouring cells easily.

PUFFFIN is a versatile plasmid-based construct that can be easily transfected into different cell types, offering a key advantage over other systems, as it does not require complex or extensive genome engineering. Furthermore, PUFFFIN's modular design enhances its flexibility, allowing us to modify key factors, such as fluorescent colour, to suit specific experimental needs. During my project, I worked with lab members to characterise the PUFFFIN neighbour-labelling system in human embryonic stem cells using flow cytometry and live imaging. This is a novel extension of a technique previously validated only in mouse embryonic stem cells. We were also able to explore the labelling dynamics when using different ratios of senders to receivers. Our preliminary results were promising; the PUFFFIN system successfully labelled neighbouring cells and is compatible with human ESCs. While more work is needed to confirm if this system performs comparably to its use in mouse ESCs, these findings are an exciting step forward.



Throughout the project, I learned key techniques like human embryonic stem cell culture and flow cytometry. I also had the chance to shadow other lab members and gain exposure to core molecular biology and microscopy methods, skills often discussed in lectures but rarely seen up close at the undergraduate level. One of the biggest lessons I took away was how much time and careful planning go into designing and troubleshooting experiments. Not everything works on the first try, and problem-solving often meant returning to the literature and adapting our approaches.

This experience has solidified my desire to pursue a career in research. I plan to apply for research assistant roles to broaden my skills further, with the long-term goal of continuing on to postgraduate study and I believe this studentship has made me more marketable. The studentship not only provided valuable lab experience but also helped me grow as a researcher through weekly seminars and lab journal clubs, which improved my presentation and critical thinking skills.

I am incredibly grateful to the BSCB and Professor Sally Lowell for this opportunity, and to lab members Sophie and Alicia for their support, guidance, and mentorship throughout the summer."

Investigating the role of glycosylation in SERPIN function in Inflammatory Bowel Disease

Margot Field held a studentship with Dr Rachael Barry at Imperial College London

"This summer, I carried out my research project in the Barry Lab at Imperial College London as part of the Undergraduate Research Opportunities Program. My project was entitled "Are SERPINS in IBD glycosylated, thereby contributing to excessive protease activity?". This experience was incredibly enriching as I learnt and applied a range of new research techniques that I will take with me as I continue my studies in biological research.

I have always been interested in pursuing a career in research as I have really enjoyed the time spent in the lab during my Biochemistry degree at Bristol as well as in previous placements. I was therefore inspired to spend this summer continuing to further my passion by finding a research opportunity. I reached out to different labs in London, especially at Imperial, and had many conversations with Dr Rachael Barry about the fascinating work her group does on serine protease inhibitors (SERPINS) in IBD and other diseases. I was particularly interested by the Barry Lab as I have always been drawn to understanding the mechanisms of disease and I found it exciting to learn more about how proteases are implicated not only in IBD but also in cancers and other conditions. Dr Barry kindly offered to host me in her lab and encouraged me to apply for BSCB funding to support my placement.

I am so grateful to my supervisors, Clara Finnigan and Dr Sara Fontalva Ostio, for their teaching and support throughout the summer. I started off my project by preparing faecal water samples for western blotting; carrying out BCA assays and further sample processing. I then went on to perform a series of western blots using a range of anti-SERPIN antibodies to identify potential glycosylation patterns. These experiments involved samples from patients with Crohn's Disease, Ulcerative Colitis and Irritable Bowel Syndrome, alongside healthy controls. From these results, I was able to select a subset of samples to treat with a protein deglycosylation mix before running further western blots. Comparing treated and untreated samples, I observed pattern differences that may have been due to the loss of glycosylation, though more experiments would be needed to confirm this.

What I enjoyed most was applying myself to learning and refining these techniques, particularly western blotting, and having the chance to discuss my project with other members of the Barry Lab. Troubleshooting experiments together was not only interesting but also really helped me to develop confidence in my own abilities. Over the course of this project, I feel that I have grown a lot as a scientist. I am excited to carry forward everything I have learnt, not just the techniques but also the experience of working collaboratively in a lab group, into my final-year research project at Bristol.

Thanks to the Barry Lab and the BSCB, this summer has been a hugely valuable experience for me. It has made me more confident about pursuing a career in research, and I am really looking forward to taking the next step on that journey by applying for PhD positions after I graduate."

Metabolic profiling of microglia using immune stimuli for the development of small-molecule treatments for Alzheimer's disease

Katie Rogers held a studentship with Dr Fiona Ducotterd at UCL

"The BSCB's longstanding commitment to supporting early career researchers in cell biology inspired me to apply for funding to support me through my summer studentship. As an undergraduate approaching my final year, I was eager to gain practical lab experience to enhance my future career development in the field of neuroscience. I visited the ARUK Drug Discovery Institute (DDI) at University College London last summer for a 6-week project which taught me cell culture, assay development and general lab etiquette. I thoroughly enjoyed my time there and learned a lot about what is involved in the process of drug discovery and development, so I knew there was a lot to gain from another summer placement to further develop my skills in the lab.

My project involved metabolic profiling of microglia, the brain's resident macrophages, using immune stimuli for the development of small molecule treatments for Alzheimer's disease. The project focused on these main research questions: 1. What is the effect of increasing concentrations of IFN- gamma on the metabolic function of primary microglia and HMC3 cells? 2. What genes are expressed during different timepoints of IFN-gamma treatment in HMC3 cells? 3. What is the optimal timepoint for IFN-gamma treatment in HMC-3 cells?

During the project I utilised the Agilent Seahorse Mito Stress Test on primary rat microglia that had been treated with IFN-gamma, an inflammatory stimulus. In addition, I established the Seahorse assay for HMC-3 cells by performing cell titrations to determine the optimal seeding density. Furthermore, HMC3 cells were treated with IFN-at different timepoints, I extracted the RNA and performed an RT-qPCR to determine the levels of expression of inflammatory genes. Throughout the placement I developed many transferable skills, for example improved my time management skills through planning my work around treatment times and assay run times. I expanded my critical thinking and problem-solving skills by working independently to overcome technical problems when things went wrong. I further enhanced my data analysis skills by using Excel and GraphPad Prism to analyse the experimental data. Finally-I gained technical skills in key molecular biology techniques, learned to work with different cell lines and microglial models and experienced cell imaging using the

Opera Phenix. I enjoyed the independence this project gave me, especially once I was able to work alone in the lab without the help of my supervisor, planning and driving experimental design independently. It was frustrating when things didn't go as planned, but I found the best way to learn was from my mistakes.

The assay results indicated that the primary microglia cells had a slight, dose dependant increase in Oxygen Coupled Respiration (OCR) in response to IFN-gamma, whereas the HMC-3 cells did not exhibit a comparable response. RT-qPCR analysis was used to identify the optimal timepoint for IFN-gamma treatment, showing the highest upregulation of the target genes at 24 hours compared to 6-hour and 2-hour treatments. This suggests that the HMC3 cells may require a longer treatment exposure than the primary microglia cells to elicit a measurable response. This information will improve efficiency and reliability of future experiments using this assay to evaluate the effect of candidate compounds.

I am hoping to do a wet lab research project in the final year of my undergraduate degree and am planning to apply for a Master's program involving a practical project. This grant allowed me to travel and stay in London to gain invaluable experience which has reinforced my interest in a neuroscience research career and will give me the skills needed to succeed in the future."

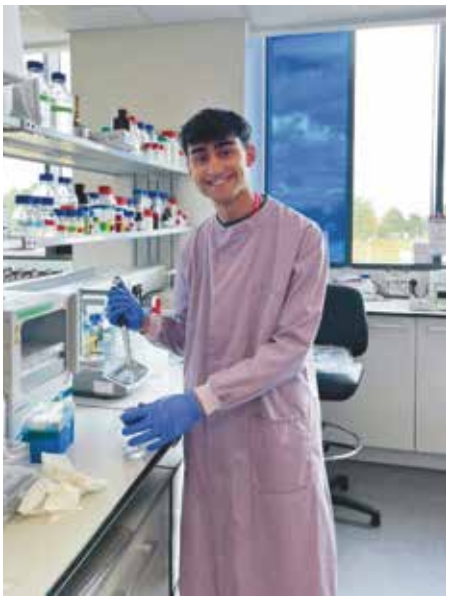
Disruption of phase-separated structures by pathological RNA foci

Ben Zakers held a studentship with Dr Judith Sleeman at the University of St Andrews

"During my research project I investigated how toxic RNA foci disrupt phase-separated structures in myotonic dystrophy type 1 (DM1). DM1 is caused by expanded CTG repeats in the DMPK gene, which form toxic RNA foci when transcribed. These foci sequester key splicing regulators, leading to widespread gene expression defects. Previous research by the Sleeman lab has shown that these pathological RNA foci also disrupt phase-separated structures such as stress granules and p-bodies.

I used a HeLa cell model with inducible CUGexp foci and applied chemical treatments followed by fluorescence microscopy to investigate the biophysical nature of the disease-associated foci and their effect on phase-separated structures. To observe these compartments, I used RNA-FISH, a method I was particularly excited to learn, having studied the theory in my undergraduate degree, but never having had the opportunity to use it in practice. My results supported the lab's current hypotheses, showing altered dynamics and numbers of nuclear compartments in disease models. I also captured high-resolution Airyscan microscopy images to build on previous findings from the lab.

Interestingly, shortly before the start of my project, new research suggested that foci in this type of model might simply represent sites of transcription rather than disease-relevant structures. Therefore, I also decided to investigate this as part of my project, to assess the relevance of the model. I carried out live cell time-lapse microscopy over extended induction periods, and after removing induction. My results showed that the foci persisted in the nucleus beyond transcriptional bursts, dispersing into smaller foci over time, supporting their relevance as a disease model. I found it particularly exciting that my experiments could directly challenge interpretations in the recent literature. It showed me how science is not fixed, but constantly evolving as new data emerges, and being a part of that process was especially rewarding.



I was motivated to apply for a BSCB studentship to work in this lab because of my interest in the molecular cell biology of disease, and my desire to gain hands-on research experience discovering more about this condition. This specific area of research also has a personal connection for me, as DM1 affects members of my family, so it was especially meaningful to contribute to the current understanding of the condition.

This project has been an important step in my development as a scientist, giving me valuable training in methods such as cell culture, fluorescence microscopy and RNA-FISH. Equally important, it has developed my transferrable skills such as patience and resilience when experiments didn't go to plan, qualities that are essential in research. The experience has strengthened both my technical abilities and my confidence, and confirmed my ambition to pursue postgraduate and further research in cell biology.

I am grateful to the Sleeman Lab for their guidance, and to the BSCB for supporting this opportunity."

Why dicentric chromosomes may predispose cancer

Evie Mattison held a studentship with Prof Jonathan Higgins at Newcastle University

"I applied for the BSCB funding because the project aligned well with the society's interests, and it allowed me to gain experience in a research lab without the financial burden of working unpaid. The project was advertised internally and was of interest to me because I have both a personal connection and real passion for this field and aspire to a career in cancer research. It was a great opportunity to explore research in more depth and put some of my knowledge into practice whilst also learning lots of new techniques and concepts.

The aim of my project was to characterise the properties of constitutional dicentric Robertsonian chromosomes using FISH and IF. A Robertsonian chromosome is a type of translocation that occurs when two acrocentric chromosomes fuse together. We were specifically interested in rob(15;21)c where chromosomes 15 and 21 fuse. If this results in a dicentric chromosome, this can predispose a subtype of acute lymphoblastic leukaemia (ALL) known as iAMP21-ALL. Through the project I was

introduced to immunofluorescence, FISH, super-resolution microscopy, high-throughput microscopy and many fluorescence microscopy techniques. These were all very new to me as they aren't included in university taught labs, which gave me an insight into the sort of experiments that are carried out in this area of research.

A highlight of this project was the independence I gained from carrying out repeats of experiments such as chromosome spreads. When successful, it felt very rewarding as I could analyse the results, using software such as ImageJ and QuPath, to look for monocentric and dicentric rob(15;21)c. However, this was not always the case as some experiments weren't as successful. For instance, when imaging some chromosome spreads, there was no visible FISH staining which meant we couldn't identify rob(15;21)c. We suspected that this was an issue with the FISH probes as they had appeared very dull on images in previous experiments. After slightly altering the protocol and using some additional probes, we could not pinpoint the specific issue as these all worked. This was quite frustrating as we had to delay other planned experiments to troubleshoot the issue. However, it was good to experience these kinds of issues as they are very common in research. In addition to this, I was able to do some extra experiments towards the end of my project, one of which was using a PCR detection kit to see whether our cell lines had been contaminated with mycoplasma (they weren't).

Overall, our experiments mostly worked well to give us results that showed rob (15;21)c chromosome can be actively dicentric in multiple cell lines, although with different intra-sister chromatid kinetochore distance in different cell lines. The results also suggested that a shorter intra-kinetochore distance correlates with less mis-segregation.

I am going into my third year of studying pharmacology at Newcastle University which will include another research project. After completing both my summer project and final year project, I hope to have a better understanding of whether I want to pursue a future in research/academia.

Funding from the BSCB allowed me to gain real-life research lab experience without worrying about trying to gain this experience in an unpaid/voluntary role, meaning I could put my full focus on the project. This whole experience has boosted my confidence in both my lab and analytical skills, providing a strong foundation for my final year project. Therefore, I am very grateful for my supervisor, Connor Gilkes-Imeson, and the Higgins lab for their continued support throughout the project, and to the BSCB for providing the studentship that made this all possible."

Deep Learning Analysis of Chemotaxis and Cell Behaviour, and Building a Database of High-Resolution Microscope Images for Training

Mateo Martin held a studentship with Prof Yanlan Mao at UCL

"BSCB funding provided the perfect opportunity to gain hands-on laboratory experience and explore whether a career in research is the right path for me. Thanks to this studentship I was able to pursue a project that aligned with my interests, combining biology with computer science and mathematics.

I chose this lab after attending a talk by Professor Robert Insall at Imperial, where I was fascinated by his research on chemotaxis. After the talk, I contacted Professor Yanlan Mao to discuss a potential collaboration with him. They explained that the mechanisms of chemotaxis lack quantification, since all existing studies use cells that alter their imposed gradient by either secreting or degrading chemoattractant. To address this, we decided to build a high-resolution database of chemotaxing cells under fully controlled gradients, allowing precise quantitative analysis of how cells respond to chemoattractant. We achieved this by using genetically engineered cells incapable of secreting chemoattractant, along with a chemically modified chemoattractant that cannot be degraded by the cells.

My research experience was highly diverse, spanning both wet and dry lab procedures. I learned to culture cells, optimize experimental conditions, and collect high-quality DIC data. At the same time, I trained our own AI model, extracted cell outlines, and tracked cell movements to generate the dataset. Beyond the technical skills, I valued being part of a collaborative and interdisciplinary team, where everyone brought different expertise and made the lab an engaging and welcoming environment.

In the last few weeks of my internship, we began to draw some preliminary inferences from our data. We found that persistence in cell motility, driven by the periodic extension and retraction of pseudopods, is tightly governed by the background concentration of the chemoattractant. This was surprising, as existing literature describes persistence as an intrinsic property of the cells, independent of the external environment. We hypothesized that this discrepancy may be due to the fact that cells in previous studies were capable of secreting chemoattractant, thereby modifying the gradient around them. If correct, this finding is particularly exciting, as it could provide a potential physiological indicator of whether cells are under the influence of a chemoattractant.

The project was not without its challenges. Fine-tuning the exact cell concentration was difficult, and several repeats were needed to ensure consistency. On a few occasions, our stock plates failed, halting our progress. Nonetheless, we overcame these difficulties through careful analysis and optimization of our experimental protocol, and seeing the dataset come together was extremely rewarding. Overall, the project gave me invaluable insight into the full research process and how experimental and computational approaches can be combined to address questions in cell biology.

Along with the BSCB, I want to thank Professor Yanlan Mao, Professor Robert Insall, and their wonderful team for making this such a rewarding and inspiring experience. This October, I will start my second year of Biological Sciences at Imperial while continuing my Mathematics degree with Spain's National Distance University (UNED). The BSCB studentship has been an excellent experience, and I am excited to continue exploring science and building on the skills I have gained.

VEGF regulation of human cell proliferation

Eve Greenfield held a studentship with Dr Vas Ponnambalam at the University of Leeds

"For the third year of my Natural Sciences degree I want to study pathology, so I researched several university laboratories and found an interesting project at the Ponnambalam laboratory at University of Leeds. The work they are doing has strong links to cardiovascular disease and cancer which aligned well with my future pathology module.

After I was accepted by Vas, he suggested that I apply for BSCB funding as this would allow me to have an eight week, fully funded experience in the lab working on my own research project.

The research question was focused on uncovering how different growth factors control VEGFR1 signalling and how this has an impact on cell death, survival and proliferation. This would be evidenced through FUCCI, western blotting and various cell viability and apoptosis assays. I was supervised by PhD student Shiny Don and MSc student John Howard, who were both very generous and helpful in sharing their time and expertise and allowed me to build my confidence in practical skills and in the lab environment. The research project experience consisted of culturing HEK293 cells and treating them with various concentrations of five different growth factors, both with and without tetracycline. This allowed me to analyse the cellular signalling responses over time course with western blots, probing for different VEGFR1 signalling proteins to understand more the molecular detail about the angiogenesis response. I also analysed FUCCI data to determine what stage of the cell cycle the cells were in with respect to time and different growth factor treatments.

I enjoyed the variety of the practical work and I was able to carry out experimentation over a longer period than I have previously experienced in undergraduate teaching labs. I became more independent in my approach to practical work and used problem solving skills to troubleshoot experiments that weren't working. However, one frustrating element was trying to troubleshoot the apoptosis assays, as each experiment took several days to carry out and often the data collected wasn't reproducible and sometimes positive controls failed, showing how important it is to have several positive controls in such assays.

I had the most success with the western blotting and, after a few trials, I was able to blot for all desired proteins and quantify this data to uncover the signalling pathways of VEGFR1 and relate this back to cell death, survival or proliferation. I found that the growth factor VEGFB seemed to elicit strong p38 signalling, which can lead to cell cycle arrest or apoptosis. On reflection it would have been more beneficial to do the western blots first and therefore easier links could have been made between the signalling and cell fate.

I am now going into my final year of a Natural Sciences degree at Cambridge where I will complete a research project in cancer biology. The skills I have gained this summer, achieved through the BSCB funding, have given me invaluable practical and theoretical knowledge for my own research project

The funding from the BSCB gave me the freedom to live near the university and really get the most out of this project. I would like to thank Vas, Shiny, John and the lab for making me feel so welcome and I am so appreciative of their support, time and knowledge."

Investigating the Role of GATA6 in Human Primitive Endoderm Specification

Yiping Zhang held a studentship with Prof Kathy Niakan and Dr Riley McMahon at the University of Cambridge

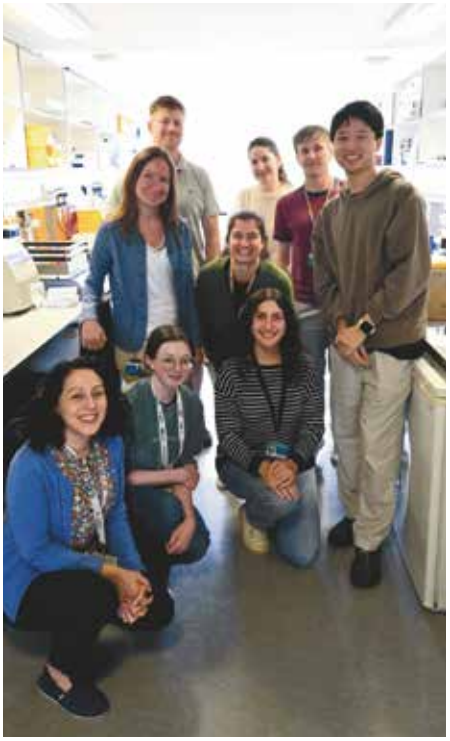
"During my undergraduate studies in Biological Sciences at UCL and through past internships, I was deeply fascinated by cell biology and inspired by the enormous potential of stem cell research to transform human health and medicine. This passion led me to apply for the BSCB summer studentship, which gave me the invaluable opportunity to join Professor Kathy Niakan's lab at the University of Cambridge, which the group is well recognised for uncovering the key molecular mechanisms governing cell fate decision of human pluripotent stem cell in early human embryonic development.

My project aimed to investigate the role of GATA6 in the specification of the primitive endoderm (PrE) using human embryonic stem cell (hESC) models. GATA6 is a key transcription factor that drives primitive endoderm fate specification in mouse embryos by activating lineage specific genes such as SOX17 and PDGFRA, integrating FGF/ERK signals, and opposing NANOG driven epiblast identity. However, its role in human embryo development remains poorly understood. To address this, we first applied two published differentiation protocols to drive naïve hESCs toward the PrE lineage to test their efficiency and reproducibility. Then, we evaluated the expression of pluripotency and PrE markers in both differentiated wild-type and GATA6-knockout cells using immunofluorescence and Western blotting.

After gaining proficiency in culturing naïve hESCs and mouse embryonic fibroblasts (MEFs), we began benchmarking the differentiation protocols by differentiating wild type naïve hESCs into primitive endoderm using two published strategies. The first was RACL medium described by Linneberg-Agerholm et al. (2019), a seven-day protocol employing three small molecules while the second was the 7F medium developed by Okubo et al. (2023), a three-day protocol that uses a combination of seven factors. Following five days of differentiation in RACL medium or three days in 7F medium, the naïve hESCs in both media lost their original compact, dome-shaped morphology, instead, they transformed into a flatter and more spread out epithelial like appearance under the microscope. To further

characterize the primitive endoderm identity, we collected and fixed cells at Day 5 and Day 7 of differentiation in RACL medium and at Day 3 and Day 5 in 7F medium for immunofluorescence staining. Confocal microscopy revealed strong expression of PrE markers (GATA6, GATA4, and SOX17) and only low levels of pluripotency markers (NANOG, OCT4, and SOX2) expressed in differentiated naïve cells under RACL conditions. In contrast, cells differentiated with the 7F protocol retained high expression of pluripotency markers, with only limited expression of PrE markers, which contrasts with the outcomes reported in the literature. We suspected this discrepancy might be due to residual MEFs in the culture, which should normally be eliminated in the 7F differentiation protocol. To address this, we attempted to remove remaining MEFs either by depleting them on gelatin or by replating cells onto Matrigel prior to 7F differentiation. Following Matrigel replating, naïve cells differentiated under 7F conditions showed strong expression of PrE markers and reduced expression of pluripotency markers, whereas gelatin depletion appeared ineffective.

Following the benchmarking of differentiation protocols, we started differentiated the both wild type and GATA6 knock-out (KO) naïve cell lines. The differentiation of Matrigel replated spCas9 edited GATA6-KO hESCs under 7F conditions retained high level of expression of pluripotency markers while shown very low PrE markers expression, which completely reversed with the expression of wild type hESC. In parallel, I genotyped new generated adenine base-edited GATA6 naïve hESCs and selected two successful edited clones along with spCas9 edited cells and wild type cells for RACL differentiation. Due to time constraints, immunostaining and confocal imaging for this could not be completed. As a next step, RNA should be collected from differentiated wild-type and knockout cells for bulk RNA sequencing to identify differentially expressed genes and predict potential direct targets of GATA6 during human PrE specification.



In summary, the BSCB summer studentship gave me an invaluable opportunity to apply my passion for stem cell biology in a world leading developmental biology lab. Through this experience, I developed key technical skills, including tissue culture, Western blotting, and immunofluorescence, while also strengthening my ability to work both collaboratively and independently and to think critically about biological research. These skills will be highly valuable for my following undergraduate research project and my future studies. Most importantly, this internship reinforced my determination to pursue a career in cell biology research and inspired me to continue exploring how stem cell biology can be harnessed to advance human health."

Imaging RNA transport during viral superinfection exclusion

Hamish Black held a studentship with Dr Jens Tilsner at the University of University of St Andrews/James Hutton Institute

"Wanting to gain lab experience in a research environment, particularly including advanced microscopy, I applied to the BSCB to conduct a summer research project on plant viruses with Dr. Tilsner at the James Hutton Institute, Dundee/University of St Andrews. Although I study neuroscience, this was an opportunity to get trained to use a confocal laser scanning microcope independently, and besides being an invaluable opportunity to immerse myself in a different field of biology, the lab's focus on live cell RNA imaging also has parallels in axonal mRNA transport.

The project focused on virus superinfection exclusion (SIE), a phenomenon where two closely related viruses are unable to infect the same host cell. SIE has major agricultural and medical implications and is central to understanding viral evolution (Folimonova, 2012). We aimed to investigate whether the replicase (enzyme responsible for replication) mediates exclusion within potato virus X (PVX). Plant leaves were transiently transformed by Agrobacterium infiltration to express the desired protein/s, such as the PVX replicase. Three days later, PVX infection was introduced into single cells via particle bombardment. Five days after inoculation, infection sites were imaged. Our results showed that expression of the PVX replicase prevented infection by PVX. Another part of the project involved molecular cloning to generate various tagged viral constructs for orthogonal dual-colour RNA imaging of PVX infections during SIE, and imaging of viral replication and translation.

My experience of the project was very positive. Despite many of the techniques (particularly within cloning) being new to me, I quickly adapted to working in the lab. Over the course of the project, procedures such as bacterial transformations and plasmid purifications began to feel second nature. Weekly lab meetings helped to consolidate my understanding of the research and improve my scientific communication. On the confocal microscope, I was able to image infection sites independently without supervision. From these, infection spread was later quantified to assess the effects of the different combinations of transiently overexpressed proteins.

Cloning was more challenging, with experiments often failing at first attempt. However, through problem-solving and troubleshooting I successfully cloned a large plasmid (>10kb) containing an infectious clone of TMV tagged for translation

imaging by three-fragment Gibson assembly. I also cloned tags for translation and RNA imaging, as well as proximity labelling, which were to be inserted into PVX infectious clones. Apart from Gibson assembly, I also used Gateway recombination and standard restriction-based cloning.

Looking ahead, I am now in my final year at St Andrews and shortly I will be conducting a research project in cognitive neuroscience. I plan to build upon my microscopy knowledge through an advanced microscopy module this semester. Next summer, I hope to undertake a research project within neuroscience that is still centred around microscopy and imaging. The BSCB funding allowed me to immerse myself in the project by supporting my living costs in St Andrews. This experience has been incredibly valuable, and has given me the confidence required to pursue further research opportunities, something I had been unsure of before."

Folimonova, S.Y. (2012). Superinfection Exclusion Is an Active Virus-Controlled Function That Requires a Specific Viral Protein. Journal of Virology, 86(10), pp.5554–5561. doi:https://doi.org/10.1128/jvi.00310-12.

Building a biomarker for high resolution imaging of retrograde axonal transport

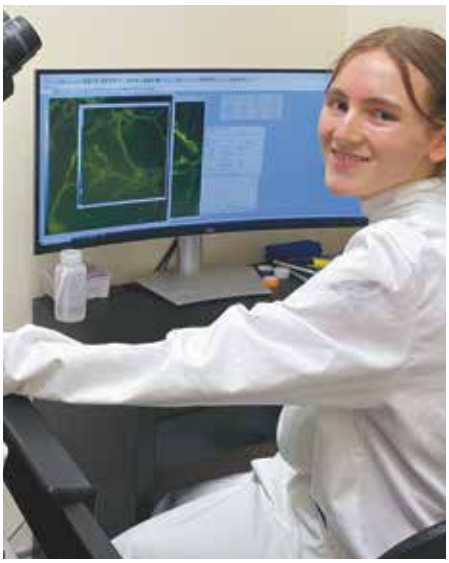
Megan Lowe held a studentship with Dr Alison Twelvetrees at the University of Sheffield

"Being fascinated by the mechanisms that facilitate the function of living organisms is ultimately why I chose to study Molecular Biology. I have relished the opportunity to learn about these processes during my degree, but have been eager to gain hands-on experience in a lab environment where people are actively working to fill in the gaps in human knowledge. With this in mind, I set out to gain some research experience over the summer. This led me to Dr Alison Twelvetrees' lab whose focus is on the cellular transport processes that underpin neuronal function and have been linked to neurodegenerative diseases when disrupted. Neurons are a particularly interesting type of cell, with their unique morphology and extensive lifespan, so I was drawn to the prospect of getting involved in this work and reached out to Alison who suggested that we put together a project and apply for a BSCB studentship.

Specifically, my project aimed to develop a biomarker which could be used for high resolution imaging of retrograde axonal transport - the movement of cargo from the synapse to the cell body. Existing methods for the study of this process often lack the sensitivity needed to follow transport at high spatial and temporal resolution, so our goal was to build a marker which could be used for single-molecule tracking. This would be a valuable tool when studying neuronal transport in the future, hopefully contributing to our understanding of the dynamics of retrograde transport in health and disease.

Fundamentally, the biomarker is based on the HCT fragment of the tetanus toxin (TeNT). This domain is responsible for the toxin's ability to bind to specific receptors on neurons and be internalised but, importantly, it is not involved in the disruption of communication between the nervous system and muscles. Therefore, when isolated, the HCT fragment loses the toxic properties of TeNT but retains the ability to undergo retrograde axonal transport. By then fusing this to several bright and stable ATTO fluorophores, it is possible to visualise the process without harming the neurons.

To achieve this, the first stage of the project was to express and purify the HCT fragment. This involved the transformation of E. coli with a plasmid encoding HCT alongside a GST tag which later enabled affinity purification of the fusion protein. During this time, I gained confidence in my wet lab skills and had the opportunity to learn how to use the AKTA Pure chromatography system. Inadvertently, I also developed my ability to problem solve at this stage as, when the first purification failed, I had to troubleshoot and optimise the protocol to maximise removal of impurities. Next, I labelled the purified HCT fragment by taking advantage of the reaction between maleimide groups on the fluorophores and cysteine amino acids in the protein to bind the two together - confirming the success by looking at the in-gel fluorescence. Finally, I applied the biomarker to a culture of rat cortical neurons and used a TIRF microscope for live imaging to assess whether we had met our aims.

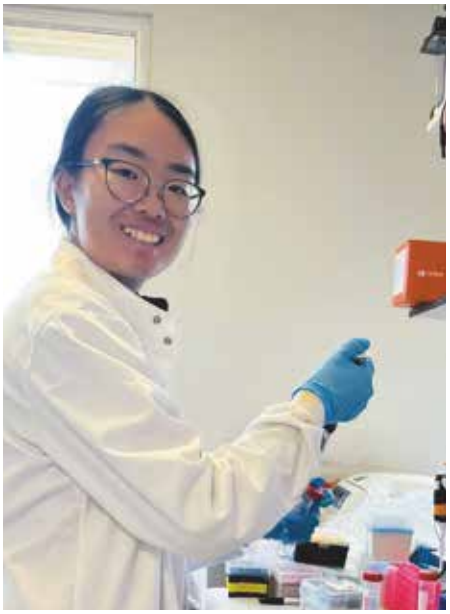


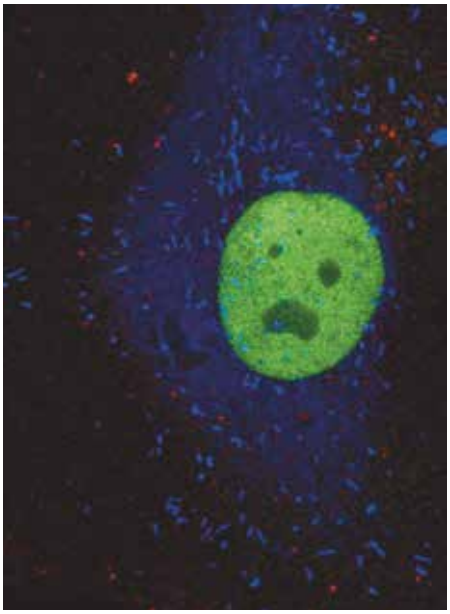
We found that the biomarker is very effective at labelling the neuronal surface membrane - a property which is itself useful, for example, in visualising axonal growth. Excitingly, there was also some evidence of bulk retrograde transport occurring. This is the first step towards being able to use the biomarker to track single molecule transport and, hopefully, with future optimisation this will be possible.

My main takeaway from this experience has been that research is not straightforward - experiments often don't work the first time, making resilience and adaptability underrated yet critical skills. Though initially frustrating, I really enjoyed this challenge and now feel far more prepared for my third year research project. I have become much more comfortable with lab work and familiar with the process of following a project through from start to finish. This experience has confirmed that I would enjoy a career in research and solidified my decision to pursue a Masters. I cannot thank Alison and the other members of the Twelvetrees lab enough for bringing me into their group and giving me the opportunity to develop such a broad range of skills which will undoubtedly be useful in my future."

The impact of senescence on the storage and release of VWF

Rachael Lam held a studentship with Dr Tom Nightingale at QMUL





"The aim of my project was to explore the impact of senescence on the storage and release of Von Willebrand Factor (VWF). I induced Human Umbilical Vein Endothelial Cells (HUVECs) with Palbociclib, a CDK4/6 inhibitor, as well as hydrogen peroxide to create two different in vitro models of senescence. Senescence is a hallmark of aging, with multiple triggers. It has physiologically beneficial effects such as wound healing, tumour suppression and embryogenesis, but also pathological effects such as promoting cardiovascular disease and metastasis. The link with VWF is important due to the pro-coagulant effect of senescent cells and VWF's role as a major protein in the clotting process.

I chose this project because I wanted to experience a lab-based research project on senescent cells, as I was aware of the new therapeutics based on senescence. During my project, I observed an increase of VWF in senescent cells compared to non-senescent cells, using immunofluorescent confocal microscope imaging. This led me to consider whether the increase could impact secretion, which I explored via secretion assays and dot blots. As part of an exploration of WPB turnover, I was also able to learn how to complete a cycloheximide time course in order to understand VWF turnover in non-senescent cells.

I really enjoyed my time at the lab, learning new techniques and it helped me visualise how a career as a clinical academic could be like. As a medical student, I had not been able to spend time in a research facility as part of my course, so I wanted to use this opportunity to explore research as part of my medical career. With a clear project aim and outline, I was able to explore the project fully, developing my understanding of both the techniques and the background.

I was able to replicate my experiments, but there were some difficulties with the hydrogen peroxide induction of senescence, with a fine line between inducing senescence with its stable cell cycle arrest and causing cell death of my HUVECs. Therefore, I was not able to complete analysis of some of my earlier hydrogen peroxide experiments. With hindsight, I would have replicated the hydrogen peroxide experiment more times to account for the cells affected by the initial hydrogen peroxide induction. Despite this, it was exciting to see the increase of VWF in both models of senescence compared to their controls, suggesting that senescence rather than the specific chemicals I added are important for this increase.

The funding from the BSCB has allowed me to realistically assess my suitability for research and complete this project without financial worries. I have developed so many transferable skills such as critical thinking, organisation and adaptability. This has shown me how applicable research is to a medical career and how it can be incorporated into my clinical training. It has encouraged me to consider intercalating during medical school and exploring clinical academia as a career further."

Above: Rachael's image of a Palbo-induced "sad" senescent cell (Green – p21; Red – PECAM; Blue – VWF)

Investigating dysregulated axonal transport in novel humanised ALS models

Philo Lloyd held a studentship with Prof Giampietro Schiavo at UCL

"Amyotrophic lateral sclerosis (ALS) is the most common type of Motor Neurone Disease (MND) and is a rare disease with an incidence of only 1.5 to 3 people per 100,000 in the US. It is a neurodegenerative disease that presents with muscle weakness and twitching eventually progressing to paralysis. However, it does not impact cognitive abilities.

Over the summer, I have focused on exploring the defects observed in both upper and lower motor neurons. More specifically, I have been using induced lower motor neurons to explore the potential role of axonal transport deficits in ALS pathology.

MND is an area that has fascinated me since I first discovered neuroscience, both for personal and academic reasons. Since the age of 15, I have fundraised for MNDA in memory of my grandmother by running long distance races (marathon hopefully coming soon!) In my first year of my Neuroscience BSc, we learnt about advancements in the understanding of mechanisms driving MND but never delved deeply into the research that led to these advancements. The research project was focused on axonal transport deficits in humanised models of ALS, so part of my attraction of this project was the cell biology aspect which meant I got to work on

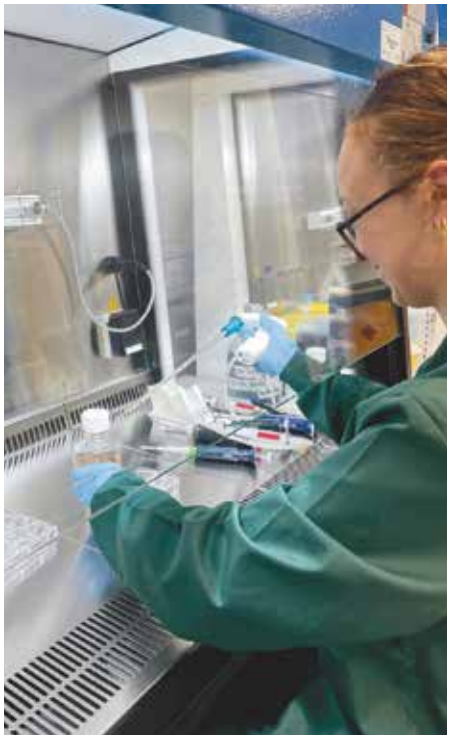
iPSCs, organoids and neurons! It was incredible to actively partake in and observe research processes I had heard so much about in lectures.

One element of axonal transport dysregulation we investigated was the speed of retrograde transport, separated into the transport of endosomes and mitochondria. Firstly, I made tripartite microfluidics (MFCs) which I then seeded with neurons by adding them to the somatic compartment. These neurons were then treated with HcT and a ZEISS confocal microscope was used for live imaging of the axonal compartment microgrooves to capture the retrograde signalling endosomes. I then used ImageJ software to determine whether there was a significant difference in speeds by tracking the organelles in these videos frame-by-frame.

After live imaging, I fixed the MFCs with formaldehyde (PFA) and stained them with fluorophores for immunofluorescent imaging, again using the confocal microscope. Once again using ImageJ, I quantified the stains in the resulting images from the immunofluorescence experiments. On top of observing how a confocal microscope works, these protocols allowed me to develop a host of new skills.

Part of the experience that I loved was seeing the collaboration between labs – I observed many experiments led by members of other labs showing Alya their techniques. For example, a PhD student also from the Schiavo lab walked us through RNA extraction, RT-PCR and gel electrophoresis - the results of which were used to determine whether certain cryptic exons (specifically UNC13A) were expressed in TDP-43 M337V neurons. Throughout my 6 weeks in the Schiavo lab, I have gained theoretical and practical knowledge of the field, with the practical element spanning from data analysis to wet lab skills.

Inspired by my work on iPSCs with Alya, I have chosen to study a module next year on Applied Tissue Engineering which focuses on the development of alternative model systems to address the 3 Rs of animal research. One of the central parts of the BSCB ethos is increasing education around cell biology careers, something I intend to implement in the Neuroscience Society as Vice President this coming year. Overall, I aspire to continue putting my new skills into practice, hopefully by completing another internship next year, and I will chase every opportunity to further immerse myself into the field.



On top of thanking the BSCB for providing me with the means to fully immerse myself into the lab for the first time, I thank everyone who welcomed me with open arms - I have experienced the community that exists in a lab, including all their quirks, and leave with mentors I treasure in Alya Masoud Abdelhafid and Giampietro Schiavo."

Society Business

BSCB funding to support members throughout their careers

Full details of all schemes are on the BSCB website (https://bscb.org/).

The BSCB Honor Fell and Support Grants schemes continue to be popular and we ask that applications are uploaded at least 6 weeks ahead of time to allow for assessment and transfer of funds to successful applicants. We expect all successful applicants to acknowledge BSCB funding using our logos found on our website.

Honor Fell Travel Awards

Sponsored by the Company of Biologists, the Honor Fell Travel Awards provide financial support for BSCB members at the beginning of their research careers to attend meetings and courses. Applications are considered for any meeting or course relevant to cell biology.

BSCB members may apply for funds for both an online and in-person conference in the same calendar year (these together will count as 1 travel award only). The amount of the award depends on the location of the meeting or course. Awards will be up to £500 for travel within the UK (except for BSCB Spring Meeting for which the full registration and accommodation costs will be made), up to £700 for travel within European and up to £1000 for meetings and courses in the rest of the world.

The application form and complete information about the scheme are available at https://bscb.org/competitions-awardsgrants/travel-bursaries/honor-fell-company-of-biologists-travel-awards/

Company of Biologists Support Grants

These grants are available for independent group leaders/PIs with no current funds for travel to attend meetings, conferences, workshops, practical courses, PI laboratory management courses and courses to re-train. BSCB will also consider applications to attend virtual and online scientific meetings, conferences, workshops and courses.

For detailed information and to apply please see https://bscb.org/competitions-awardsgrants/cob-support-grants/

Childcare Award

The BSCB now accepts applications to provide financial help with childcare or care for dependents when the applicant is presenting at a scientific meeting. For example, these claims can be for:

- Home-based childcare/dependent care expenses incurred because of meeting attendance (funds may not be applied to normal ongoing expenses).
- Travel of a relative or other care provider to your home to care for your child(ren) or dependent while attending a meeting.
- Travel of a care provider to the meeting with you to care for your child(ren).

For more information and to apply please see: https://bscb.org/competitions-awardsgrants/travel-bursaries/childcare-award/

BSCB Imaging competition

THE BSCB runs an annual competition to shows the best of your research images.

Prizes: 1st Prize £200; 2nd Prize £100; 3rd Prize £50. Winners will be published on BSCB webpages and will also be used in the Magazine and other promotional material. Copyright will remain with the creator- if you do not agree that the images may be used as stated, you must state this on the entry form.

Submission: Entrants must supply their name, address, email address, and BSCB membership number on entry. Entries must be sent by email (10 x 11.96 cm 300 dpi) to stephen.robinson@quadram.ac.uk). Only one entry per person is allowed. The subject matter of competition entries is flexible but must reflect current research in cell biology.

Further details are at: https://bscb.org/competitions-awardsgrants/image-competition/image-competition-rules/

BSCB Science Writing Prize

The BSCB Science Writing Prize aims to encourage writing skill development in young researchers on topics of key relevance to cell biology. Entrants have either communicated their own research projects or science stories in the literature, in a clear and concise way aimed at a non-specialist audience, or written essays that were not be limited to research per se, but tackled a bioethical or science policy issue. The winner receives a prize of £500 and has their winning entry published in the BSCB magazine and online (both on the BSCB website and, subject to editorial acceptance, on the excellent www.lablit.com website).

Each year shortlisted entries are judged by an external expert. In previous years we have enlisted the kind help of Tim Radford (Writer and former Science Editor at The Guardian), Viv Parry (Science Writer and Columnist), Tania Hershman (Science writer, former science journalist and writer-in-residence at Bristol University), Dr. Jenny Rohn (a cell biologist at UCL, who is also a science writer, novelist, blogger, broadcaster, the editor of LabLit.com and the founder and chair of *Science is Vital*), and Barbara Melville (science writer, former writer-in-residence at the MRC Centre for Regenerative Medicine and board member with the Association of British Science Writers).

Remember: You must be a BSCB member to enter. The full rules and how to enter can be found at https://bscb.org/competitions-awardsgrants/science-writing-prize/

The British Society for Cell Biology

Statement of Financial Activities for the Year to 31 December 2024

	Unrestricted Funds	Restricted Funds	Total 2024	Unrestricted Funds	Restricted Funds	Total 2023
Income from:	£	£	£	£	£	£
Grants	35,000	62,500	97,500	35,000	40,000	75,000
Investments	2,413	-	2,413	255	-	255
Charitable activities						
Subscriptions	26,110	-	26,110	26,610	-	26,610
Other income	-	-	-	1,715	-	1,715
Total income	**63,523**	**62,500**	**126,023**	**63,580**	**40,000**	**103,580**
Expenditure on:						
Charitable activities						
Grants payable:						
CoB	-	48,486	48,486	-	47,500	47,500
Other grants	6,244	-	6,244	871	-	871
Studentships	30,107	-	30,107	34,320	-	34,320
Medals	4,732	-	4,732	-	-	-
Costs of meetings	9,596	-	9,596	2,707	-	2,707
Website expenses	539	-	539	1,873	-	1,873
Newsletter costs	4,845	-	4,845	4,734	-	4,734
Membership fulfilment services	26,924	-	26,924	34,198	-	34,198
Examiner's remuneration	3,522	-	3,522	3,516	-	3,516
Miscellaneous	492	-	492	354	-	354
Subscriptions	773	-	773	242	-	242
Insurance	95	-	95	575	-	575
Total expenditure	**87,869**	**48,486**	**136,355**	**83,390**	**47,500**	**130,890**
Net (expenditure)/income	(24,346)	14,014	(10,332)	(19,810)	(7,500)	(27,310)
Transfer between funds		1,324	(1,324)	-	-	-
-						
Net movement in funds	(23,022)	12,690	(10,332)	(19,810)	(7,500)	(27,310)
Funds brought forward at 1 January 2024	208,242	28,022	236,264	228,052	35,522	263,574
Funds carried forward at 31 December 2024	**185,220**	**40,712**	**225,932**	**208,242**	**28,022**	**236,264**

BSCB Committee 2026

The Society is run by a Committee of unpaid volunteers elected by the Members. The Officers of the Society, who are all members of the Committee, are directly elected by the Members. The BSCB committee is comprised of office-holders (President, Secretary, Treasurer, Meetings Secretary, Membership Secretary, Magazine Editor, Award Co-ordinators and Web Co-ordinator) and up to 12 other ordinary members, including one PhD student representative, one postdoc representative and a schools liaison officer, who are coopted onto the committee.

The committee is always interested in hearing from cell biologists who wish to contribute to the society's activities. Members of the society are encouraged to nominate candidates for the committee or officers positions at any time. Formal nominations should be seconded by another member of the society. The committee is also happy to receive un-seconded informal nominations. Nominations should be sent to the BSCB Secretary.
The committee generally meets twice a year, at the spring meeting and in the autumn in London. Additional meetings are arranged from time to time. Items for consideration by the committee should be submitted to the Secretary prior to the meetings. The BSCB has charitable status (registered charity no. 265816). The BSCB AGM is held every year at the Spring Meeting.

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BSCB Ambassadors 2026

The BSCB Ambassadors – the society's advocates in the UK cell biology community – should be your first point of call for information about what the society can do for you and also how you can get involved. They should also be the people readily available to ask about sponsoring you for membership. Anyone who wishes to volunteer to become a BSCB ambassador at any Institutes not represented in the list below please contact the BSCB.

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The BSCB Magazine is published once a year in winter in hard copy. News is updated frequently through our website and BSCB Twitter feed. Follow us at @Official_BSCB

Submission

If you have an idea for an article, please e-mail the editors a brief outline first. It is preferable to send all articles, reports and images by e-mail (though alternatives can be arranged after contacting the editor). Attachments for text can be in txt, rtf or doc format. Please send images as 300dpi JPEG, TIFF or PSD files. Submission of articles and images should be made to Dr James Brown (james.brown@ul.ie) and/ or to Dr Matta Finelli (Mattea.Finelli@nottingham.ac.uk).

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BSCB Subscription information

The online application form can be found at www.bscb.org. The annual fees are:

BSCB Individual Full £45.
BSCB Individual direct debit £35.
BSCB Student £50 (3-year membership) or £70 (4-year membership).

Membership runs from January – December. If you join after October 31st you will not be asked to renew until the January after next. Eligibility for some funding schemes requires 1 year membership or 1 membership renewal – whichever comes sooner.

Membership enquiries

To become a BSCB member please go to: https://bscb.org/members/become-a-member/

If any of your personal details have changed please login to the BSCB members area online and update your information. bscb.org/members/become-a-member/

Please email HG3 to report any difficulties with the membership page: bscb@hg3.co.uk

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Journals

BSCB members are entitled to a range of discounts from journal and book publishers. Members should check www.bscb.org for the latest information.

Rules of Attraction

CERTIFIED BIO-CERT® QUALITY

The right surface makes all the difference. Whether you need cells to stick or float, we've got the solution for the perfect environment.

Managing cell adhesion or maximising conditions for spheroid formation has never been easier. Our surface treated microplates by BRAND® are perfect for your stem cell applications. These sterile plates are available in a choice of well formats, shapes and colours.

Choose the right BRANDplates® for your cell culture needs to deliver optimal performance.

Maximum Adhesion

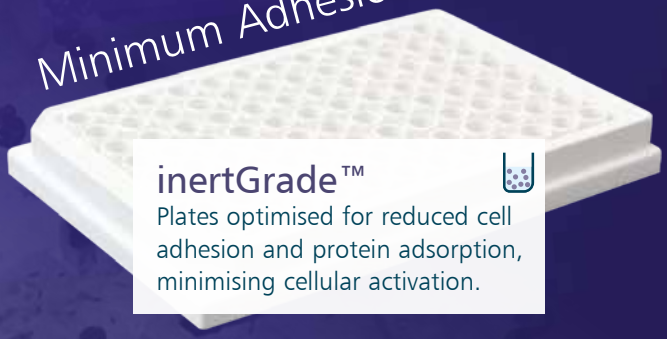
cellGrade™

Surface treated plates with freely accessible carboxyl and hydroxyl groups that enable easy cell adhesion. Created specifically for the cultivation of adherent cell lines.

Maximum Adhesion

cellGrade™ plus

Contain amino acid groups enabling direct attachment of cells suitable for cultivation of sensitive cell lines and reduced serum media.



Minimum Adhesion

inertGrade™

Plates optimised for reduced cell adhesion and protein adsorption, minimising cellular activation.

Maximum Adhesion

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An ideal alternative to a biologically coated surface, equivalent to Poly-D-Lysine. Optimal adhesion that withstands frequent washing and supports serum-free protocols.

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