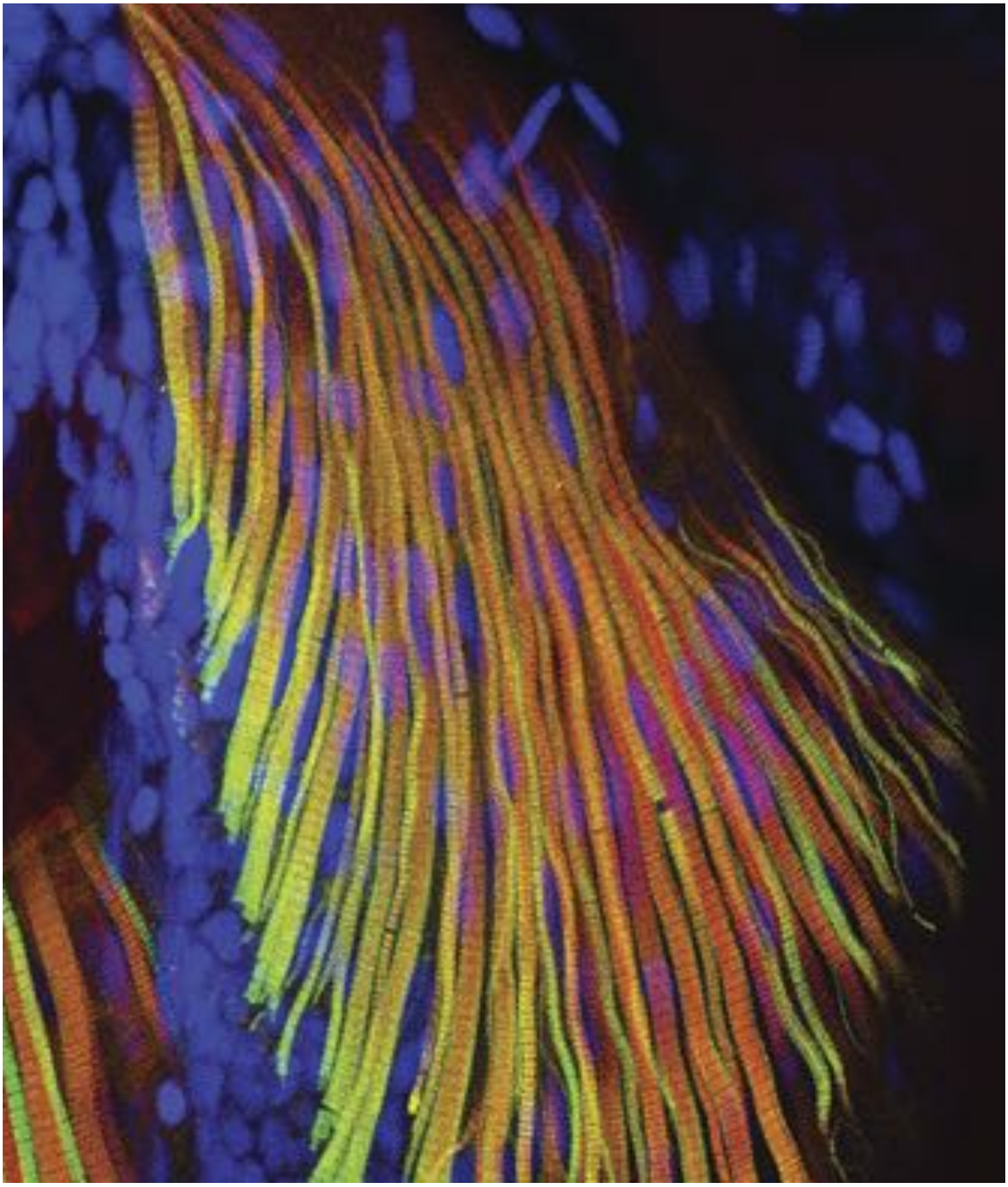
2019

BSCB Magazine

BRITISH SOCIETY FOR CELL BIOLOGY

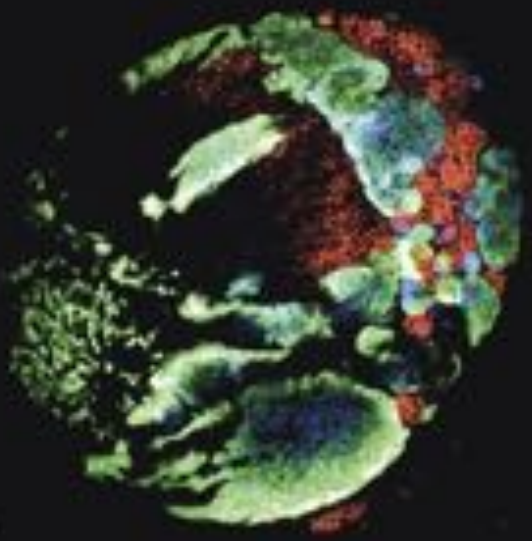


BSCB BSDB

BSCB/BSDB Joint Annual Spring Meeting

7th-10th April 2019 / University of Warwick UK

Peter Andrews (UK)
Monica Bettencourt-Dias (POR)
Mina Bissell (USA)
Kirsten Bomblies (UK)
Jeremy Carlton (UK)
Pedro Carvalho (UK)
Guillaume Charras (UK)
Ulrike Eggert (UK)
Yasuyuki Fujita (JAP)
Cedric Gaggioli (FRA)
Aga Gambus (UK)
Jesus Gil (UK)
Stephen Goff (USA)
Karen Guillemin (USA)
Volker Haucke (GER)
Eva Hoffmann (UK)
Anne Huttenlocher (USA)
Gwyneth Ingram (FRA)
Henrik Jonsson (UK)
Claus Jorgensen (UK)
Alexandra Joyner (USA)



Stephanie Kermorgant (UK)
Madeline Lancaster (UK)
Prisca Liberali (CH)
Ilaria Malanchi (UK)
JP Martinez-Barbera (UK)
Maria Mittelbrunn (SPA)
Denise Montell (USA)
Sean Munro (UK)
David Pellman (USA)
Eugenia Piddini (UK)
Jeffrey Pollard (UK)
Adrienne Roeder (USA)
Leonor Saúde (POR)
Shankar Srinivas (UK)
Claudio Stern (UK)
Aurelio Telleman (GER)
Sally Temple (USA)
Xavier Trepat (SPA)
Gia Voeltz (USA)
Stephen West (UK)

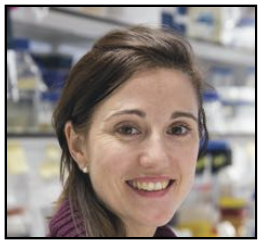
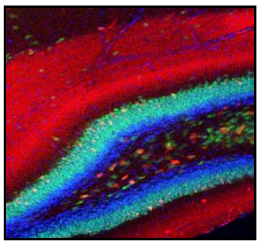
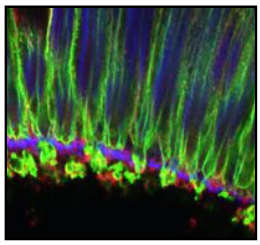
Topics: genome integrity and regulation, migration and invasion, trafficking and membrane biology, cell-cell communication, mechanics and morphogenesis, germline and early embryogenesis, growth regulation, organelle biogenesis and disease

Scientific Organisers:
Susana Godinho, Sally Lowell, Tristan Rodríguez,
Victoria Sanz-Moreno, Anne Straube, Rita Sousa-Nunes

Find out more at www.bscb-bsdb-meetings.co.uk

Image by Andrea Chai

BSCB Magazine 2019

Editorial

Welcome to the 2019 *BSCB Magazine*! This year Susana and Stephen are filling in for our Newsletter Editor Ann Wheeler. We hope you will enjoy this year's magazine!

This year we had a number of fantastic one day meetings sponsored by BSCB. These focus meetings are great way to meet and discuss your science with experts in your field and to strengthen your network of collaborators within the UK. You can read more about these meetings in the magazine. If you have an idea for a focus one day meeting, check how to apply for funding on page 4. Our ambassadors have also been very busy organising events at their local institutions to recruit new members to the society. Ambassadors play an extremely important role in advertising BSCB meetings, the science writing and image competitions, and promoting the society in general. Thanks to all of them!

We truly enjoyed the 2018 BSCB Spring meeting Dynamic Cell III, which took place in Manchester Conference Centre from 18th-21th of March and was jointly organised by BSCB and the Biochemical Society. A big thanks to BSCB committee member Anne Straube for doing a great job putting this meeting together. As usual, this meeting was a success amongst cell biology aficionados, covering several topics from cytoskeleton, mitosis, cell-cell communication and lots of cool imaging! We had fantastic talks, including the ones by our own Hooke Medal prize winner Andrew McAinsh and WICB prize winner Meritxell Huch. If you want to know more about either of them, check out their interviews with Journal Cell Science and our Postdoc committee rep. Congrats to BSCB Young Cell Biologist of the year, Cerys Currie (University of Warwik) and runner up Mustafa Aydogan (University of Oxford), as well as to BSCB postdoc poster of the year winners Dr Anna Caballe (University of Oxford) and Dr Agata Gluszek-Kustusz (University of Edinburgh).

In 2019, we will have our jointly BSCB-BSDB Spring meeting at Warwick University from 7th–10th April, organised by BSCB members Susana Godinho and Vicky Sanz-Moreno. The programme for this meeting, which usually provides a broad spectrum of themes, has a focus on cancer biology: cell migration/invasion, organelle biogenesis, trafficking, cell-cell communication. While most sessions will run in parallel, will also have one joint session between both societies on genome integrity and regulation. Two inspirational scientists will be giving the plenary lectures: Mina Bissel and Sally Temple. For more information about this conference please go to www.bscb.org. Information about travel awards can also be found on the BSCB website.

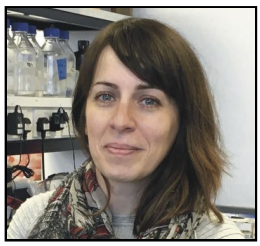
This year we have several articles written by undergraduate students who carried out research in BSCB members laboratories. This programme has been a success with so many students developing great research and a passion for cell biology! Visit our website for information on how to apply for these studentships!

Get in touch with us if you have ideas for an article. We are always happy to hear from you!

Looking forward to seeing you at Warwick University in April!

Susana Godinho, Stephen Robinson and Ann Wheeler, BSCB 2019 Newsletter Editors

Front cover: microscopic structure of pectoral fin and hypaxial muscles of a zebrafish *Danio rerio* larvae at four days post fertilization. The immunostaining highlights the organization of fast (red) and slow (green) myosins. All nuclei are highlighted in blue (hoechst).



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Website: www.bscb.org **Printer:** Hobbs

Society News

BSCB President's Report 2018

This has been an exciting and busy year for the BSCB. It started in March 2018, when we shared our annual meeting with the Biochemical Society in Manchester. 'Dynamic Cell III' was a great success, with capacity audiences of over 200 in the lecture theatre. This was the third of what has now become a series of meetings entitled 'The Dynamic Cell', which started in Edinburgh in 2009, followed by a joint BSCB/Biochemical Society 'Dynamic Cell II' in Cambridge in 2014. We are really grateful to our Anne Straube (BSCB meetings secretary) and her co-organisers for their hard work in putting the 2018 programme together.

Highlights for me included the two BSCB Medal Lectures from the 2018 BSCB Hooke Medal winner Andrew McAinsh (University of Warwick), and the BSCB Women in Cell Biology Early Career Award Medal winner Meritxell Huch (Gurdon Institute, Cambridge). Andrew gave a fascinating movie-filled talk on his work investigating how microtubules interact with kinetochores to drive mitosis. Meritxell's talk demonstrated the power of organoid cultures to discover how liver tissues regenerate, and how this can go wrong in diseases such as cancer. Watch the BSCB website or follow us on Twitter or Facebook for announcement of the two 2019 Medal winners (and please note that any BSCB member can nominate a UK cell biologist for either award).

Our 2019 annual meeting will be at the University of Warwick (7-10 April), this time shared with the British Society for Developmental Biology (BSDB). This meeting will include sessions on cell biology topics including cell migration, trafficking and organelle biogenesis, with an impressive list of top international speakers. PhD students and postdocs get the opportunity to meet the speakers one evening in the bar, and also have a career workshop before dinner on 7 April. I hope that current BSCB PhD students and postdocs will register for the meeting and encourage their colleagues to attend too.

In addition to our annual meeting, the BSCB sponsor one-day focused meetings on a variety of cell biological topics, which are organised by BSCB members. Several of them have been running annually for over 10 years, including the 'Actin meeting', 'Microtubule meeting' and 'Endocytosis meeting'. Our aim is to serve the UK cell biology community through these meetings, particularly through having PhD students and postdocs give most of the talks. By providing sponsorship, the BSCB helps to keep the meeting costs down so that whole laboratories can afford to attend.

The committee welcome new proposals for meetings in areas of cell biology that are not currently covered by a sponsored meeting, and indeed the BSCB committee has recently agreed to fund a new sponsored meeting in 2019. Please visit the BSCB website for information about future BSCB-sponsored meetings and how to apply for meeting sponsorship.

The BSCB would not exist without the BSCB committee, who all provide their time voluntarily to organise BSCB meetings, administer the finances, communicate with BSCB members, and run the travel awards and summer studentships. Each person commits to being on the committee for three years, which can be extended to a maximum of six years. This year we said thank you to Melanie Pagnini (PhD student rep), and welcomed Joyce Yu as our new PhD student rep. We also welcomed Folma Buss, Jason King and Carine de Marcos Lousa as new committee members. In addition, our BSCB committee member Jenny Rohn has taken on a new role for the committee as Science Advocacy Officer. She is our link with the Royal Society of Biology (RSB), a professional body representing many societies and organisations in the area of biological sciences. The RSB carries out public communication of science, education outreach, and informs and lobbies the government on behalf of its members and member organisations. If you are interested in any of these areas, please contact Jenny and she can help you to get involved.

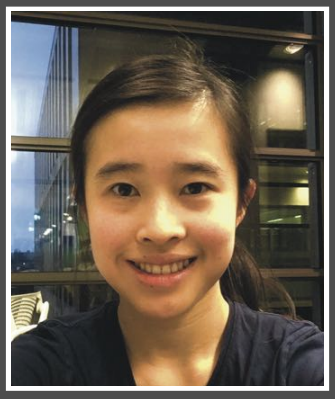
We are very grateful to the BSCB Ambassadors, who act locally within their Institute/University to promote the BSCB, BSCB meetings, and the values of BSCB membership. This year Andrew Carter (BSCB Membership Secretary) and Carine de Marcos Lousa sent BSCB marketing packs to all Ambassadors, which we hope were helpful in encouraging new PhD students and postdocs to join the BSCB. If you are interested in being a BSCB Ambassador, please contact Andrew Carter and he will send you details.

The BSCB is generously funded by the Company of Biologists, which allows us to fund summer studentships for undergraduates to gain experience in working in a BSCB member's laboratory, as well as provide travel awards for BSCB members to attend meetings, as well as workshops and courses to learn about new techniques. If you are a PhD student or postdoc, you can apply for travel funds towards any meeting or course relevant to cell biology. Group leaders who do not currently have any travel funds in their grants are also eligible to apply. Please do check out our website to find out what is available and how to apply.

The BSCB committee looks forward to meeting many BSCB members in 2019 at our annual meeting in Warwick and/or at one of our sponsored meetings. Please look out for our stand at these meetings to talk to members of the committee and find out more about the BSCB.

Anne Ridley
BSCB President

New Student Rep – Joyce Yu



Hello! I'm Joyce, your new PhD student representative. I am taking over from Melanie Panagi, who has done an amazing job in the past two years!

I am a third-year student in Jean-Paul Vincent's group at the Francis Crick Institute, London. I am originally from Hong Kong, but have been studying in the UK for the past 8 years. Before starting my PhD, I did Molecular and Cellular Biochemistry in the University of Oxford and during my master's year, I worked on mRNA localisation in the *Drosophila* neuromuscular junction.

My current research interest is Wnt signalling in the *Drosophila* wing, studying the role of Evi/Wntless in Wnt secretion, as well as investigating the redundancy among the various Wnts expressed in the developing wing. In my spare time, I enjoy helping out at various public engagement events at the Crick. I have previously volunteered at local schools as a science tutor in Oxford, and also had fun organising and designing summer science courses for primary school students.

My role as the BSCB student rep is to organise the student/postdoc careers roundtable, and the students' symposium in the coming joint BSDB-BSCB Spring meeting. I am also keen to advocate for more prizes and opportunities for PhD students working in cell biology, as well as more online resources specifically for PhD students on our BSCB website. To any PhD students with ideas for the society, feel free to contact me at joyce.yu@crick.ac.uk. I am looking forward to seeing all of you at the 2019 Spring meeting!

Schools news: 'Teenagers don't get cancer – it's a disease of older people, true or false?'

If this were an exam question the answer would be both 'true' and 'false'. 'True', because the incidence of cancer generally rises with age. False' because young people can get cancer. The number diagnosed is relatively low with an average number of 17 cases per day in the UK.

When a young person is diagnosed with cancer they generally have little knowledge of disease or illness, or how to cope with it as anyone who has seen the film 'The Fault in Our Stars' will testify. Teenagers are shocked emotionally, especially with regard to their social life, their future, and the thought of not being able to "keep up with their mates". Teenagers are also at a stage when they are finding out about themselves and looking to the future. They question knowledge and events. If a teenager is diagnosed witj cancer they want to know something about it, how they got it, how it might affect their future and, to quote a teenager, "what my cancer looks like" and whether they can "pass it on".

Society is now more open about discussing cancer and it is now a topic in some 'A level' (or

Journal of Cell Science most-read articles of the last 12 months

Research articles

High-content tripartite split-GFP cell-based assays to screen for modulators of small GTPase activation.
Faten Koraïchi, et al.
J Cell Sci 2018 131: jcs210419
doi: 10.1242/jcs.210419

Effects of mutating α-tubulin lysine 40 on sensory dendrite development.
Brian V. Jenkins, et al.
J Cell Sci 2017 130: 4120-4131; doi: 10.1242/jcs.210203

A novel fluorescent reporter detects plastic remodeling of mitochondria–ER contact sites.
Zhaoying Yang, et al.
J Cell Sci 2018 131: jcs208686
doi: 10.1242/jcs.208686

NudE regulates dynein at kinetochores but is dispensable for other dynein functions in the *C. elegans* early embryo.
Patrícia A. Simões, et al.
J Cell Sci 2018 131: jcs212159
doi: 10.1242/jcs.212159

Reviews and Cell Science at a Glance posters

Actin assembly mechanisms at a glance.
Klemens Rottner, et al.
J Cell Sci 2017 130: 3427-3435; doi: 10.1242/jcs.206433

Amyloid assembly and disassembly.
Edward Chuang, et al.
J Cell Sci 2018 131: jcs189928
doi: 10.1242/jcs.189928

Formation of COPI-coated vesicles at a glance.
Eric C. Arakel, Blanche Schwappach.
J Cell Sci 2018 131: jcs209890
doi: 10.1242/jcs.209890

Maintaining centrosomes and cilia.
Sascha Werner, et al.
J Cell Sci 2017 130: 3789-3800; doi: 10.1242/jcs.203505

equivalent) biology courses in England and elsewhere. Teenagers diagnosed as having cancer will find many answers and helpful suggestions in the Teenage Cancer Trust excellent publication 'A Young Person's Guide to Cancer'. More detailed information about the biological aspects of cancer is being provided by material being written for the 'softCELL' e-learning section of the BSCB website. The material is written at two levels. Level one provides a general overview. Level two gives much more detail. The e-learning site is for students, teachers (many of whom did not encounter the subject in any detailed way during their college years), and anyone interested in the biology of cancer. The material is being produced with the guidance of Professor Mel Greaves, FRS, Director of the Centre for Cancer and Evolution at The Institute of Cancer Research (ICR), and the kind help of others.

David Archer

BSCB focussed one-day meetings

In addition to the annual meeting at which the BSCB awards the Hooke and WICB medals and holds its AGM, the BSCB sponsors a number of focussed one-day meetings. Amongst those regularly supported are the Bristol-based Actin meeting, the Edinburgh-based Microtubule meeting and the London-based Endocytosis meeting. These meetings attract more than 100 participants from the UK cell biology community, are relatively informal with speaking opportunities mainly for students and postdocs, and have very low registration fees. Thus these meetings allow early career researchers to become part of the scientific community in their field of research without the need for a large travel budget.

If you like the idea, but there is not yet a one-day meeting for your field, why don't you organise one? To get started, first gather support from colleagues in your field to make sure there is demand and a minimal number of participants guaranteed. Find a suitable date and venue and then apply for funding from the BSCB and other sources. We would expect the BSCB to be the main or one of the main sponsors and that the society contribution is acknowledged accordingly.

An application form is available on the BSCB website, please submit these at least 6 months before the meeting to one of the two deadlines: 1st March and 1st October for consideration by the BSCB committee. When we decide sponsorship applications, we use the following criteria:

1. Topic of the meeting falls within the remit of BSCB and does not overlap with other sponsored meetings.

2. The meeting provides presentation opportunities predominantly for early career researchers and is open to the entire UK cell biology community.

3. It is a small one-day meeting and the BSCB is the main sponsor.

4. BSCB sponsorship is clearly indicated - ideally by attaching BSCB to the name of the meeting.

5. BSCB members benefit from reduced registration rates and a small exhibition stand for BSCB is provided that will be manned by a BSCB committee member attending the meeting.

6. The meeting presents value for money – many BSCB members benefit.

Anne Straube
BSCB Meetings secretary

BSCB Sponsored or allied meetings 2019–20

February 2019

Host-bacterial interface meeting, 11 February 2019, Francis Crick Institute, London

April 2019

SBCF meeting "Membrane Biophysics of Exo-Endocytosis" 3-6 April 2019, Cannes-Mandelieu, France

BSCB-BSDB Joint Annual Meeting, 7–10 April 2019, Warwick University, Coventry

Jacques Monod Conference "Mitotic and Meiotic Cell Cycle control and executions", 8-12 April, Roscoff, France

May 2019

British Microtubule Meeting, 13 May 2019, National Museum of Scotland, Edinburgh

SBCF Symposium "A day at the cell centre with Michel Bornens", 17 May 2019, Institute Curie, Paris, France

Journal of Cell Science Meeting "Cell dynamics: organelle cytoskeleton interface", 19-22 May 2019, Pestana Palace Hotel, Lisbon, Portugal

December 2019

ASCB-EMBO annual meeting, 7-11 December 2018, Washington DC, USA

September 2020

BSCB-SBCF Joint Meeting "Building the Cell", 23-25 September 2020, Institute Pasteur, Paris, France

BSCB Ambassadors News

Visiting the Advanced Imaging Facility at Janelia

After hearing a talk from Teng-Leong Chew, at the Facility Manager's meeting in January this year (run by the Royal Microscopical Society), I mentioned a possible project to him, that might benefit from using the iPALM, one of the 4 specialised microscopes on offer at the AIC. Leong is the current Director for the Advanced Imaging Centre (AIC). That quickly led to writing an application to access this microscope (application deadline was looming!) followed by Skype discussions about the project and finally acceptance, and a date fixed for July this year, where myself and two members from my lab spent two weeks using the microscope.

The AIC currently has 4 microscopes, the iPALM (Interferometric Photoactivated Localisation Microscopy), Lattice Light sheet Microscope, SiMView Light Sheet Microscope (multi-view light - sheet microscope with adaptive imaging capabilities) and a Multifocus microscope which can capture data from 9 focal planes with one exposure. None of these microscopes are available commercially. The microscopes are supported by a team of specialists both for the hardware itself, and the software to analyse the images once collected. We were supported by Jesse Aaron and John Heddleston, who worked with us on the iPALM, and Satya Khuon, who helped us with our tissue culture preparations, shipping etc. They were really helpful and supportive and we had a great experience.

The AIC is based at the Janelia Research Campus in Ashburn, Virginia, which is about 6 miles outside of Dulles Airport, near Washington. It is an autonomous research campus of the Howard Hughes Medical Institute (HHMI); the buildings were designed by the architect Rafael Viñoly (who incidentally also designed the building at 20 Fenchurch Street in London, nicknamed the 'Walkie-Talkie'), and it was opened in 2006. It is set on what used to be a farm (Janelia Farm), and the original farmhouse is still present. The building itself is built into a hill below the original farmhouse, and is one long arc-shaped building, with a lake in the front. Beyond that is the 'hotel' which looks out onto a second lake, with a resident heron. There is a range of other accommodation for hosting visitors nearby, and everything you need is on site, making it easy to devote time to experiments.

We very much enjoyed our 2 week stint using the iPALM. It was challenging, and not everything went as planned, but we came away with a clear understanding of the challenges, some promising images, and plans for the next visit! Staying at Janelia and using the microscopes is all free. All we had to do was find the travel money to go and visit. If you have a project that you think might benefit from one of the AIC microscopes, then why don't you contact the AIC team, and talk to them about it? All you need to know is here: http://janelia.org/aic

Michelle Peckham, Alistair Curd, Ruth Hughes

Are you Leading on the Edge?

I'll begin with a moment in time. It had been another bad night's sleep. Too many thoughts. The list of things I hadn't got done. My overflowing inbox. Requests coming in thick and fast, for things I simply couldn't say no to. I was already working long days, endlessly. I had nothing left to give. How would I get this all done?

And the gnawing dread. That people would think I wasn't up to this. That I couldn't take the pressure and the responsibility. That they were looking for the moment I'd show them that I just wasn't competent or capable enough. That I would let myself, them, everyone around me down.

When I stepped up, taken on greater responsibility, I'd quickly realised how isolated and out of my depth I felt. I had some support, but there was someone I was working with who was an essential part of the team, who had influence, who I began to realise was undermining me. Nice enough to my face, but not listening to me. Giving contradictory messages to others. Pushing forward their thoughts, ideas, opinions in relation to my work, the areas I was responsible for. I couldn't stop it. I felt like a puppet dancing to their tune, when it should have been me defining the tune and the dance. And people were starting to pay a lot more attention to them not me for how this work was going to move forward.

As I walked into my office, I was already weighed down by the prospect of what the day would bring, my shoulders were tight, my neck tense and there was a knot in my stomach. It would be another long, exhausting day…

Does this sound familiar?

You've got into something you care about. It intrigues you, you want to know more, understand better and use your understanding to make a difference. You're well published and have established yourself. You're known by your work, your reputation.

And you're rewarded – funding, people, resources, promotion, wider responsibilities.

But your workload is overwhelming, you're being pulled apart by everyone's expectations that you'll get involved, help, support, drive forward a myriad of initiatives and a whole range of projects, none of which are yours. You're having to face difficult people and wishing you could just walk away from dealing with them. And somehow, you've lost what it was that you really enjoyed doing.

So, if you're finding yourself in this position, overwhelmed and perhaps feeling isolated and unsure how to cope with it all, what can you do?

It's worth saying that changing your situation and how you're feeling will take time and effort. There isn't a quick or easy fix. But small steps, however small these might feel, will make a difference and lead to something bigger changing.

First, acknowledge the situation you're in and accept that this is how you feel.
What do I mean? I was back at my GP surgery to get a repeat prescription. My migraines had

become so frequent that my GP was suggesting long term medication as a preventative treatment. Of course, there would be side-effects. We had already discussed reducing my stress levels as it was clear stress was a major contributing factor to the onset of my migraines. This was the moment when I realised something had to change. I didn't want to be on long term medication. And I didn't want to carry on like this.

This realisation helped me understand the situation I was in and accept that it was only going to change if I did something about it.

So, ask yourself: What would you gain if something was to change? What would you lose if nothing changes?

Now assess what (e.g. tasks, activities, people, organisations, expectations) is leading you to feel this way.
Because I had so much going on, I was finding it more and more difficult to focus on any one task or area. My mind kept flitting and so rather than thinking things through, coming to conclusions and making decisions, I was overloaded by too many thoughts leading to constant anxiety. By separating out what I was working on, who was involved, I was able to prioritise what I felt I should focus on. It also made me realise who/what was making me feel particularly stressed. I ended up drawing this out on paper as it helped me visualise all that I had going on, more clearly. Through this, even though my workload felt overwhelming, I knew that I somehow had to deal with the person who I felt was undermining me, as this was causing me significant amounts of stress.

If there is one thing you could change, what would it be?

Determine what and who could help you.
In working with the person who I felt was undermining me, I had already realised that I was uncomfortable around them but that it was important that I build a relationship with them as they had influence. So I had met them, started to build a relationship to get to know them better and tried to understand their point of view. But this wasn't working.

I'm not one for moaning about colleagues and so felt deeply uncomfortable about the idea of talking about this person negatively. But I had to do something differently. So after carefully and discreetly sounding out a few people I found someone who I felt I could be open with, who'd keep what I said confidential. It was speaking with them openly that helped me realise what I could do to change the situation I was in.

So, who could help you see what options you have? Remember that there are always options.

Set yourself 3 actions you're going to take!
After speaking with the person I confided in, I had a plan. But it was big. It was about changing what I thought were people's perceptions of me. Getting their attention and demonstrating to them I knew what I was talking about. I had to build my reputation. But just doing that seemed too huge to comprehend. I went away lost for a while and did nothing. It took more conversations for me to realise that it would only happen when I did something. And then I did. I identified who I should speak with. I arranged meetings and set the agenda so it was on my terms. I wanted to ensure that on the critical decisions I could see coming up, I had their full attention to put across my point of view, understand their opinion and to influence the direction the work would take. And over time this started to make a difference, I finally began to feel that people were listening and that they respected my opinion.

What steps, however small, are you going to take?

Make your actions SMART
SMART is Specific, Measurable, Achievable, Realistic, Time-bound. Even though on paper it seems simple enough – work out who I'd speak to, set up a meeting with them, tell them what it's about – I still came up with a myriad of excuses as to why I didn't do it immediately. They're too busy, I'll be wasting their time, I don't have time today/tomorrow/next week, what do I really want to say to them…

For me the actions that I was thinking of doing fitted the SMART criteria bar one. I didn't set a deadline. So days slipped by. When I finally did set a deadline, I got it done.

How SMART are your next steps?

Tell someone you trust, to help you do what you have decided to do.
What helped me set my deadline was that the person I was talking to asked me how things were going. I felt a twinge of shame. I'd not done anything. So I agreed with them by when I'd do it. Suddenly I was accountable to someone else for getting the actions I had decided on, completed. And I did.

Who could hold you to account, to help you accomplish what you've decided to do?

Once you've completed your 3 actions, re-evaluate where you are and choose your next 3 actions.
During my first set of meetings I agreed with each individual on how we would continue to discuss our work. So my next action was ensuring that this happened and that I continued to set the agenda. I also reassessed who I should develop closer ties with and started to build those relationships on a one-to-one basis. Inevitably more meetings meant that my time was even more squeezed. Eventually I looked at the things I simply wasn't getting done and decided I had to have an honest conversation with the people involved.

Several months later, I'm back

with my GP we're revisiting the conversation about long term preventative medication for my migraines. I decline. I feel a little more in control and am prepared to see how things go. I still feel overloaded with work, but I'm better at identifying the small steps that keep me progressing. And the meetings I've continued to have with everyone are paying off. I've been building my reputation and feel more confident people are listening to me and respect the decisions I make. Whilst the person who I felt was undermining me is still talking and behaving in the same way, I feel less affected by it and this makes me realise that by changing how I did things I've been able to put myself in a better place.

So let me know how you get on. It would be great to hear your story.

Siân Taylor

After working at AstraZeneca on late stage drug discovery projects, Siân returned to academia managing a range of translational research projects and recently oversaw and managed a >£6m National Institute for Health Research Patient Safety Translational Research Centre focused on primary care. She obtained her qualification as an executive level coach and now focuses on making a difference for scientists who have just taken on greater leadership and management responsibility, so that they become self-assured leaders.
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Getting a feel for excretion.

Excretion may not be the most glamourous of topics. It may not even be the most exciting. So, in the absence of excitement and glamour, what is left? Importance.

Whilst an appreciation of excretory mechanisms is a pre-requisite for understanding human physiology and medicine, it is relevant also to a myriad of fields, from anthropology to zoology. The ability to expell the waste products of metabolism, neutralise ingested toxins and clear out other unwanted molecules is fundamental to all organisms on Earth. Excretion is not simple, it is governed by thousands of genes, a multitude of organs and tissues, hundreds of cell types and any number of environmental insults that can tip the balance in favour of biological chaos. Failures in excretory systems decrease fitness and increase mortality.

In this brief section, I'll introduce you to the invertebrate nephrocyte, a relatively obscure cell type that has gained notoriety as one of the most important model systems for the study of human kidney disease.

Nephrocytes have been described in numerous invertebrates from spiders to bivalves to fruit flies. They are typically groups of large, non-motile single cells, that reside within an organism's open circulation system. Nephrocytes are bathed in the circulating hemolymph (invertebrate blood) and able to internalise any molecules they see fit for destruction. They are powerhouses of endocytic activity - having one of the most veracious appetites of any cell type in nature.

Viewed at the electron microscopic level, it is apparent that each nephrocyte is packed with endocytic vesicles and lysosomes, ready for the mass destruction of anything they ingest. At its surface are uniformly distributed slits acting as molecular sieves, filtration barriers allowing smaller molecules into the interior of the cell, whilst excluding larger material. This combination of filtration slit and endocytic function is integral to the nephrocyte's function – they are machines for filtering and cleaning hemolymph.

The functions of the invertebrate nephrocyte have been conserved during evolution and up-scaled and up-cycled in the mammalian kidney. The filtration slits are now expressed by podocytes (cells that very elegantly envelop capillaries entering the kidney glomerulus), whereas the endocytic function is expressed by proximal tubule cells residing immediately downstream of the glomerulus. The slits still perform their filtration function and are crucial component of the kidney's glomerular filtration barrier (GFB), whereas the endocytic function is a crucial aspect of mopping up and recycling any blood proteins that pass the GFB. Although in separate locations in mammals, Nature has not changed the genetic blueprint of these functions.

This simple (yet landmark) observation – that aspects of kidney function are conserved between species – allows us to disrupt nephrocyte filtration genes in the fruit fly Drosophila (the geneticists' favourite model organism) and extrapolate the consequences to human kidney function. The reverse is also true, mutations thought to cause devastating kidney disease in people can be tested for their ability to affect nephrocyte biology in flies. Findings from the fly model can then support the argument that the mutation is causal in the human condition. Genetically speaking, this is powerful stuff.

Scaling up from fruit flies in the research lab to blue-bottles (Calliphora) in the teaching labs is allowing us to run student practicals that would otherwise be extremely challenging (dissecting fruit flies is somewhat of a miniaturist artform). By using the larger Calliphora, students get to more easily dissect an insect, see its beating heart and, with certain coloured dyes, see also the endocytic function of the nephrocytes. That's a lot of biology for a small investment and possibly one of the only undergraduate demonstrations of human kidney function outside of vertebrate models.

So, the next time you wipe your windscreen free of bugs, take a moment to marvel at the evolutionarily conserved biology drying in the sun – a lot of it runs your own body.

Paul S. Hatley.
University of Bournemouth;
Department of Life and Environmental Science.

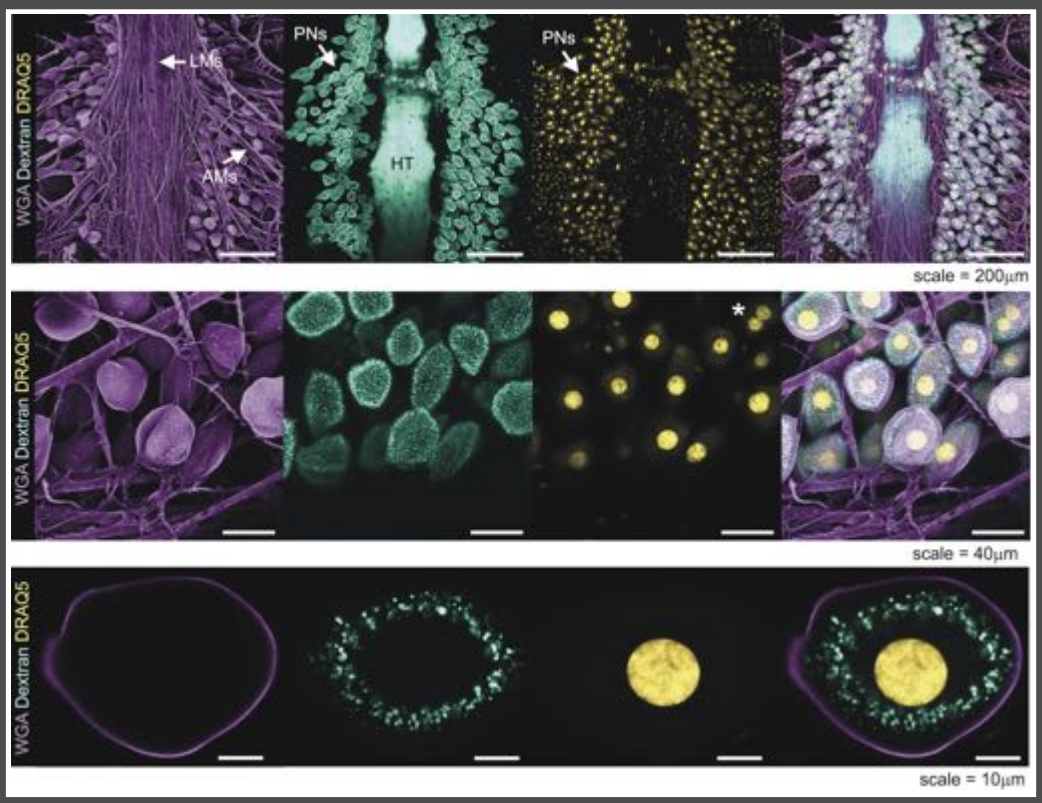
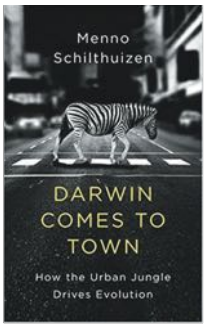


Figure1. Nephrocytes in the blue bottle (Calliphora). The heart (HT) of an insect aligns with the midline and pinned to the cuticle via Alary muscles (AMs). Runing parallel with the Heart are longitudinal muscles (LMs). Either side of the heart are groups of single Pericardial Nephrocytes (PNs) which endocytose and filter the hemolymph. In this instance their endocytic function has been visualised with fluorescently labelled dextran (which accumulates within the nephrocytes' endocytic vesicles and the heart's lumen). Occasionally nephrocytes will be binucleate (asterisk). A similar anatomical set up is seen in the fruit fly Drosophila. Wheat germ agglutinin (WGA, a general cell counterstain); DRAQ5 labels nuclei.

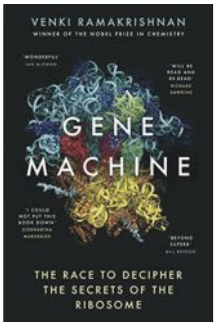
Books: This year's great biology reads round-up

DARWIN COMES TO TOWN
Menno Schilthuizen
Quercus (2018)



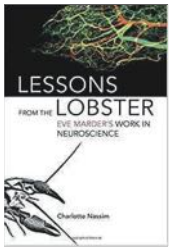
Menno, a professor of evolution at the University of Leiden, is interested in the effect of urbanization on biodiversity. Intriguingly, it's not all bad news: although the incursion of humanity and its infrastructure has caused a number of species to die out, other plants and animals are slowly adjusting to a world of steel and concrete, evolving clever adaptations that their wilder counterparts could only dream of. From pollutant-resistant feathers to seeds that are better at establishing without soil, the flora and fauna of the modern world are just getting on with the business of survival in a way that Darwin would surely find at once strange and familiar.

THE GENE MACHINE
Venki Ramakrishnan
Oneworld Publications (2018)



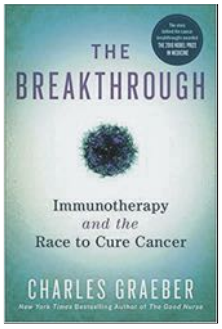
Everyone knows about DNA, but what of the equally important but largely unsung humble ribosome? The Gene Machine recounts the race to solve the structure of this key enzymatic complex, told by the man who shared the Nobel Prize for its elucidation. Ramakrishnan, a group leader at the Laboratory of Molecular Biology in Cambridge and currently serving as the President of the Royal Society, is well-placed to deliver not only the scientific details but, perhaps more interesting, the inside scoop about the personalities, twists and turns, politics, conflicts and egos involved.

LESSONS FROM THE LOBSTER
Charlotte Nassim
The MIT Press (2018)



This engaging book describes Brandeis University professor of neuroscience Eve Marder's love affair with a key network of thirty neuronal cells lacing the stomach of crustaceans such as lobsters and crabs, which she has spent four decades investigating in fine cellular detail. From this scrutiny, Marder has managed to extrapolate a host of rich information whose implications reach far beyond digestion into human thought and consciousness. Along the way, Nassim paints the molecular anatomy of a careful and intuitive scientist at work.

THE BREAKTHROUGH
Charles Graeber
Twelve (2018)



We find ourselves on the cusp of a new era, when the cells of our immune system will hopefully be used routinely to fight cancer better than radiation or chemo. Given how good our immune systems are at ferreting out and destroying invaders, many may well have been thinking, "What took us so long"? Bestselling author Charles Graeber delivers the answer with great style and form, taking us through the exciting story to date – the human as well as the scientific – and predicting where this brave new world is heading.

MYOTUBULAR TRUST 2019 CALL FOR PROJECTS (OPEN TO INTERNATIONAL APPLICATIONS)

The Myotubular Trust is holding a 2019 call for research grants. We will require completed applications by 1700 hours GMT Friday 15 March 2019.

We anticipate making awards in late June / early July.

We are looking to fund further projects that will help find a cure and/or a treatment for any form of centronuclear and myotubular myopathy (congenital X-linked recessive; congenital autosomal recessive; autosomal dominant), focusing on research that would not generally be funded by public or industrial funding sources. This call will be open to research bodies internationally.

We will be looking for the following types of application:

1. A project grant applied for by a Principal Investigator to fund a project for 2-3 years duration to be carried out by a Post-Doctoral researcher, or PHD student

2. A Myotubular Trust fellowship – basic science (3-4 years duration), where the scientist has identified a group that he or she wants to work with. Award is made to a named individual.

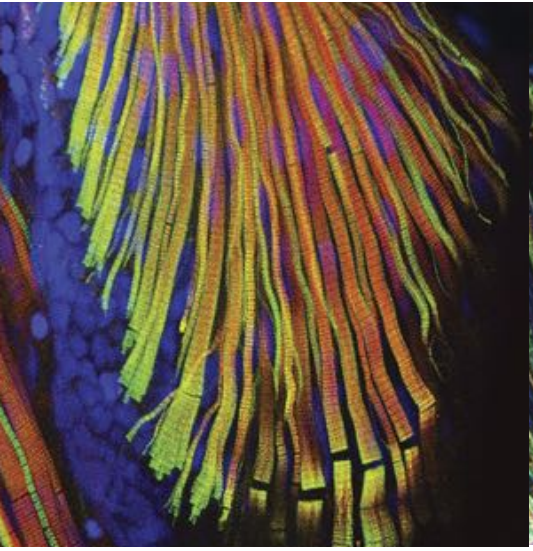
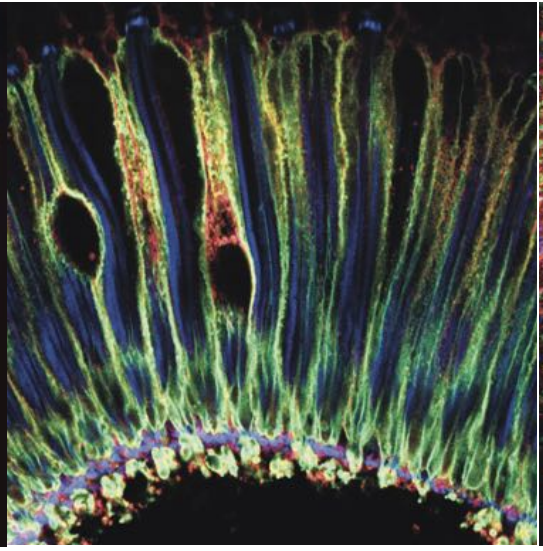
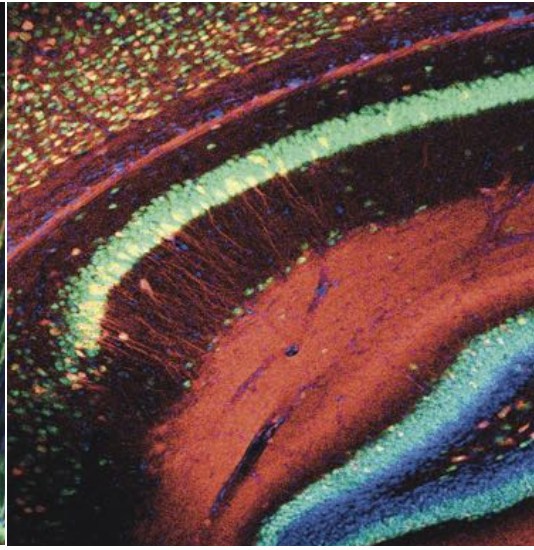
In particular, we would like to encourage the application of new technologies to research into centronuclear and myotubular myopathy; interventional trials; and those which may involve collaboration between different medical disciplines and / or different research institutions.

We are also willing to consider applications which involve joint funding with other organisations.

Myotubular Trust's Scientific Advisory Board (SAB) is chaired by Professor Francesco Muntoni of The Institute of Child Health, University College London. The SAB makes recommendations to the Myotubular Trust Trustees on which projects to fund, based on scientific assessment and peer review.

Further guidance and application forms can be found on the website myotubulartrust.org/research/grants-process/

To learn more about the Myotubular Trust, please see our website www.myotubulartrust.org or email research@myotubulartrust.org

BSCB Imaging competition 2018

First: Massimo Ganassi; King's College, London

I obtained my PhD in Molecular and Regenerative Medicine, founded by the Italian Government, in Dr Susanna Molinari's group at University of Modena and Reggio Emilia (Italy). Soon after completing my PhD project on zebrafish embryonic muscle development, I joined the laboratory of Prof Serena Carra, founded by a AriSLA fellowship (fondazione ricerca Sclerosi Laterale Amiotrofica) to study the role of small heat shock proteins in cell-stress response and in the pathogenesis of Amyotrophic Lateral Sclerosis. I am now a postdoctoral researcher in the laboratory of Prof Simon Hughes at King's College London. My project aims to define and understand the molecular and cellular processes contributing to skeletal muscle formation and development using zebrafish.

This confocal image shows the microscopic structure of pectoral fin and hypaxial muscles of a zebrafish *Danio rerio* larvae at four days post fertilization. The immunostaining highlights the organization of fast (red) and slow (green) myosins. All nuclei are highlighted in blue (hoechst).

Second: Alessandro Bossio; University College, London

I graduated with a BSc in Biological Sciences from the University of Florence (Italy) in 2013. I then moved to the UK where I completed the MSc Neuroscience at University College London (UCL), working on the characterisation of the blood nerve barrier under the supervision of Prof Alison Lloyd.

I am currently in the final year of the MRC LMCB PhD programme at UCL, where am I am working in the lab of Prof Patricia Salinas studying the role of Wnt signalling in the brain. My project focuses on understanding the role of Frizzled receptors, the main receptors for Wnt ligands, in synapse formation.
This confocal image of a sagittal section of the mouse brain (P15) shows the architecture of the hippocampus, a region of the brain important for learning and memory. Cell nuclei are labelled with DAPI (blue), mature neurons with NeuN (green) and axons and dendrites from cells infected by intraventricular injection of AAV1 are stained for mCherry (red). Note a couple of blood vessels spanning the whole hippocampus and a thick layer of neuronal progenitors (NeuN negative, DAPI positive) in the curve of the dentate gyrus. This image was taken whilst working with Prof Patricia Salinas.

Third: Sonia Muliyil; University of Oxford

After completing my undergraduate degree in Chemistry, I moved to the Tata Institute of Fundamental research, Mumbai for my Integrated Masters and PhD degree. My PhD work in cell and developmental biology was focused on understanding the complex cross talk between mitochondrial remodeling, stresses and apoptotic signals, using a model for wound healing. I was awarded the HFSP and EMBO fellowships for carrying out my Post Doctoral research in Prof. Matthew Freeman's lab at the Sir William Dunn School of Pathology . The aim of my project in the Freeman lab has been to uncover the functions of a pseudoprotease in the nervous system, and to investigate its molecular role in protein quality control.

Waves in the retina (Snapshot of a *Drosophila* adult retina): This confocal image shows a tangential section of the Drosophila adult retina comprised of multiple photoreceptors and inter-ommatidial cells. Phalloidin (blue) marks the photoreceptor light sensitive membranes, also known as the rhabdomeres, present apically while Na+-K+ ATPase (green) marks the baso-lateral membranes of the Photoreceptors. This section is also co-labeled with an anti-caspase antibody (red).

Hooke Medal winner 2018 – Andrew McAinsh

Andrew McAinsh received the 2018 Hooke medal, established to recognize an emerging leader in cell biology. The Hooke medal is awarded at the annual spring meeting of the British Society for Cell Biology.

Andrew McAinsh received his PhD from the University of Cambridge, UK, working in the laboratory of Steve Jackson on DNA damage and repair mechanisms in yeast. He then joined the laboratory of Peter Sorger as a Jane Coffin Childs Fellow to work as a post-doc on kinetochore biology at the Massachusetts Institute of Technology, Boston, USA. In 2005, he returned to the UK to establish his independent laboratory at the Marie Curie Research Institute, Surrey, before moving to the University of Warwick in 2009 to co-found the Centre for Mechanochemical Cell Biology (CMCB). Subsequently, Andrew was appointed Professor of Cell Biology and became a Wellcome Senior Investigator, and was awarded a Royal Society Wolfson Research Merit Award. He co-directs the MRC Doctoral Training Partnership in Interdisciplinary Biomedical Research, and in 2017 became Head of Division of Biomedical Sciences at Warwick Medical School. Andrew is interested in understanding how the chromosomal multi-protein complex, the kinetochore, ensures error- free chromosome segregation.

What inspired you to become a scientist?
To be honest, I didn't find biology very interesting back in school – I was much more into art and design. However, I did like science per se, because it has this artsy side to it as well. Then, during my A- levels, our teacher brought some *Drosophila* stocks to school and showed us the different phenotypes, such as eye colour. He was really good and taught us everything about the antennapedia mutation and the genetic basis of it. At that moment I thought: now, that's really cool – this is something I could actually do. I guess the combination of good teaching, actual practical work and seeing things amazed me, and I decided to go to Manchester University to do my undergraduate degree. There, I started reading genetics, but as soon as I attended the courses on molecular biology I realised that I was less interested in genetics, but much more in the molecular basis of phenotypes, so I swapped my course.

Back then, it was certainly a great period to look at the molecular biology behind genetic mutations... Yes, we had all these mutants and their phenotypes, and we were starting to see how this was working. I had a great time at university; another very important moment was the cell cycle course with Iain Hagan. He gave these fantastic lectures and would show us real data, actual research papers. A lot of students said that it was too difficult, and that they simply wanted nice lecture notes, but Iain insisted on looking at the experiments and the data. I loved it and was very keen to go to Iain's lab because I wanted to do a PhD, working on fission yeast and all these exciting new cell cycle mutants. To my surprise, Iain said 'No, you shouldn't come to my lab.' He explained that I needed to move around, to go to different places, and see different things.

Iain then recommended Steve Jackson, who was building a lab in Cambridge to work on DNA repair. Steve was working on DNA-dependent protein kinases, and ATM had appeared as being a critical mediator of DNA damage signalling. This was also very exciting for me, and I joined Steve's lab in the end.

Followed by a post-doc with Peter Sorger (Harvard), and the work from your own research. Would you say that you nonetheless drifted back towards what motivated you to join Iain's lab?
Did I go 'full cycle'? Yes, I think there's some truth in that. Steve's lab was an exciting place at the time, there was just so much going on and I learned a lot. Next door, Jonathon Pines had started live- cell imaging and was injecting fluorescent proteins into live cells. I loved the look of that – to be able to look at both the spatial and temporal control of cell cycle and cell division. Peter Sorger's lab just had a paper in *Cell* out at that time, looking at budding yeast kinetochores, and they had started imaging the localisation patterns of kinetochores. From there onwards, they were able to identify other new kinetochore components in yeast. It was beautiful, and I thought I'd love to do something like that.

What questions are you trying to answer in your research group?
It's a story of my interests and the constant influence of the people around me. Rob Cross next door has always been a mentor, and he's looking at single-molecule mechanics. Rob has always been keen on understanding exactly how one protein (kinesin) works in detail. I've been working on the multi-protein machinery that are kinetochores, and it just seemed like a completely intractable situation in comparison to Rob's approach. But we started thinking more and more about the kinetochore as a machinery, and its mechanics, as it needs to deal with forces and generate and sense forces. Thus, if we imagine it as a protein machine, how do the parts move, what are the requirements, how do these link to function?

It's quite tough to get at, because measuring the force on a single molecule is simpler than that on a kinetochore. But now it has started to happen, and some labs have done wonderful biophysical experiments on purified kinetochore particles, for example Sue Biggins (Seattle). Again, I like that in science we can witness such things happening in the research community. Our focus therefore is on the response of kinetochores to force. How is their behaviour, their movements and attachments to microtubules influenced by this? In the end, it's about how kinetochores prevent erroneous attachments to the spindle, and thus errors in mitosis.

We also work on molecular motors that are implicated in this process, but this is a side line for the lab. Again, this is Rob's influence – I always followed his single-molecule experiments and thought that was just great fun, and it's therefore a personal interest really, and a collaborative effort. The main thing for us is kinetochores in somatic cells.

You've also developed an interest in meiosis, right?
Yes, a recent effort is to look at human meiosis. Being in Warwick helps with that, because we have a reproductive clinic here, and there's the possibility to get human oocytes. We'd like to take all the tools and live-cell imaging we've developed for studying dynamics in mitosis and apply this to meiosis I and II. How does it all work and how do kinetochores behave in this? And why is there so much aneuploidy in human embryos? It's counterintuitive. Because human oocytes are difficult to work with for various reasons, the right image-analysis tools and quantitative approaches are going to be needed to make this accessible.

Nigel Burroughs is our collaborator in the Mathematics department, and it's been great fun working with him on kinetochore dynamics in mitosis. To go out of your comfort zone is important in order to understand the problems you're facing, and this has enabled us to develop more advanced tools. It has also been essential for our research to have people in the lab who can do both the computing and the bench work.

You put your recent manuscripts on the preprint server bioRxiv. What's your take on preprints?
It's taken me a long time to do it, I have to say; I've been worrying about depositing a preprint quite a lot. Not for the reason that somebody else might see what you're doing – transparent science is great. In fact, I really value the peer review process. It's an imperfect situation and it's much talked about, but in the end, I think I'd be hard pressed to find any paper I've ever published where one reviewer hasn't made a really good contribution to the science. That's really worth something.

Yes, there are issues with peer review, but we shouldn't forget that you often get some very insightful comments, great suggestions for experiments that will substantiate what you found or change the direction slightly. That's the scientific process in my view; you get to a certain point and then you try to improve and retest your ideas.

Overall, the review process makes papers better. That's why I was slightly worried about putting a paper out there that had not been through that process – you find yourself worrying even more, internally, about the work.

Would you advocate commenting and reviewing on preprint manuscripts in order to make it better?
Yes, I like the idea of constructive feedback. I'm not quite convinced I'd want to conduct the reviewing process fully in public, but if somebody made some helpful comments, I would certainly be there writing back to that person. I'd go offline to have that conversation and then think about it further.

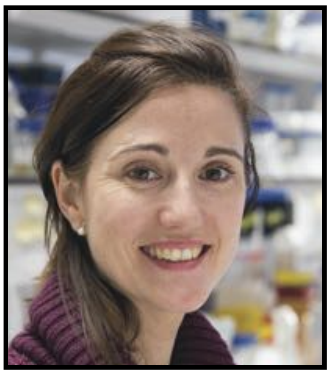
Regarding your own career: you started at the Marie Curie Research Institute (MCRI), moved to Warwick and co- founded the CMCB. Now you're Head of Division of Biomedical Sciences at Warwick Medical School and the Hooke Medal Winner 2018. How do you feel about your journey?
A lot has happened. When the MCRI closed down, there were a couple of options for what to do next; then, the opportunity arose to go to Warwick, together with Rob and Anne Straube to continue our collaborations. This was fortuitous – to have the possibility to be involved in designing the new laboratory space that we're sitting in now, and to take part in thinking about the CMCB and where it should go as an interface between cell biology and biophysics. At the time it wasn't the obvious thing to do, as there wasn't a large cell biology community in Warwick, but it was a very exciting time. Over the years, we brought people in and now we have a great research community, including an environment that is provided for the students and the post-docs.

The CMCB has built its extension in 2016, and now you have a lattice light-sheet microscope. It's certainly one of the best places to do cell biology in the UK nowadays? Well there are brilliant scientists at several places around the UK, and looking at them I find myself thinking 'I wish I could do that experiment', but it's certainly a great place to come to. You can be a student, a post-doc or career development fellow and build a successful career in a great environment here. We can and want to attract more people, and one of the challenges is to expand and diversify. My job as Head of Division is also to pursue these visions now. It'd be good for Warwick; you project the science and the campus twenty years into the future, and we'd like to see it thrive.

Andrew McAinsh was interviewed by Manuel Breuer, Features & Reviews Editor at Journal of Cell Science. This piece has been edited and condensed with approval from the interviewee.

Women in Cell Biology Early Career Medal 2018 – Meritxell Huch

Meritxell Huch was awarded the BSCB Women in Cell Biology Early Career Medal 2018. This annual honour is awarded to an outstanding female cell biologist who has started her own group in the UK within the last 6 years.



After completing her BSc in pharmacology at University of Barcelona, Spain, Meritxell Huch pursued her PhD in the laboratory of Cristina Fillat at the Centre for Genomic Regulation (CRG) in Barcelona. Wanting to move into more basic research, Meri trained as a postdoc with Hans Clevers at the Hubrecht Institute in the Netherlands. In her postdoctoral research, she successfully established a liver organoid culture that earned her the National Centre for Replacement, Refinement and Reduction of Animals in Research (NC3Rs) prize in 2013. Meri joined the Gurdon Institute in February 2014 and is currently a Wellcome Trust Sir Henry Dale Research Fellow. She is interested in the mechanisms responsible for adult tissue regeneration in the liver and the pancreas, particularly in identifying stem cell populations that respond to damage and the intracellular mechanisms regulating their activation.

What inspired you to become a scientist?
The first thing I recall is that, as a child, I could not understand how an aspirin worked; how does this pill know that it has to go to the place that is painful and do its job? That puzzled me so much that I decided to study pharmacology. What pushed me into research is that I always wanted to understand more-and-more how things work, and the lectures were not enough to cover my curiosity in that regard.

What motivates you now?
Every time you do an experiment, you realise that it actually brings you to another question. So you find something out, but it's never complete – there's always another question you want to answer. It's this constant curiosity of trying to understand everything as a whole, when you know that actually it will be very difficult.

What elements, inside or outside the lab, have been key to your success so far?
I'm a very persistent person; I just keep going until I understand something, which means that I can stay in the lab until midnight and I don't even realise the time. I also had very good mentors during my PhD and postdoc. Cristina, my PhD mentor, opened my mind to seeing things and asking questions that I hadn't thought about. Hans, my postdoc adviser, taught me how to ask the question that is important at the moment that it is important. My husband has also been a key to my success. He is my angel, constantly giving me support, and I would not have managed without him. Of course, my parents also played an important role: when I was a kid, my father once told me: "it doesn't matter what you want to be, an actress, a ballerina, a scientist or a musician, but whatever you do, just do it well and do it from the bottom of your heart" and that was one of the best pieces of advice I ever heard.

What challenges did you face when you started your lab that you didn't expect?
The surprising challenge was that the UK has a lot of regulations. Maybe I didn't notice them in The Netherlands since I was a postdoc and in an established lab, so all the regulations on how to work with human material or with mice were already in place. Here, I had to set it up from scratch. It was even harder because the institute wasn't working much with human tissue and I'm working with liver, which has additional implications, like potential pathogens (although we don't actually accept any tissue from infected patients). It took a lot of educating myself and my colleagues about these things, and sometimes I found it exhausting. Now, after having all these

regulatory issues taken care of, answering the questions we are interested in is the biggest challenge, but that is how it should be; at the end of the day, we are scientists because we try to understand the world around us.

What challenges do you think you will face in the near future?
One challenge is to be fast, and a related challenge, one I will probably face soon, is funding. If you don't get a publication, you don't get funding, and to get a publication, you have to be fast and avoid being scooped. At the end, the one that gets recognised is the one that got there first, but as a small lab it's very difficult to be fast. You don't have the infrastructure that big labs do, with lots of technicians, postdocs and PhD students. There are also so many brains in the world that at least one other person could be thinking of the same types of questions as you. If you knew the person, you could try to collaborate, but if not, you are in a race with someone who might not even exist. So you need to be fast, and you need funding, good people and all your energy and the will to understand the questions you have.

What is your advice on establishing successful collaborations?
Collaborations are not easy whether you are young or established, although when you're established, you have a reputation, which is like your business card, so when you go to someone they will want to collaborate with you. But when you're starting out, nobody knows you. People may know me because of the NC3R prize and papers, but they still don't know me as a person. They don't know if I'm a good collaborator, and establishing this trust is not easy. I've also been contacted by many PIs who want to use the system we have, and I haven't always had the impression that it was mutually beneficial. My advice is that it's good to collaborate with someone who is not completely in your field, but who has a huge enthusiasm for science and loves what you're doing. You must admire what they're doing as well. It's also good to have a collaborator at the same level.

What is the best science-related advice that you ever received? The best advice I got was from Hans, which is that you have to do your best with the people that you have. Sometimes, the first time you hire, you think that the person is a clone of yourself, but it will never be the case. It's a very common error that I also made in the beginning. Learning someone's best skills is the most difficult part of being a supervisor. So try to identify people's strengths and play in that direction, so that they can develop their maximum potential and grow, which will allow your lab to grow at the same time.

Do you still do experiments in the lab?
Yes, whenever I can, although it tends to be less than what I would like to do. But I have a very good example in this institute, because John Gurdon does many of his own experiments – I see him walking around with his white ice box. I definitely want to follow him as a model. I also like to see the raw data and I like to understand how the experiment has been done. I would not like to arrive at a point when there is a technique in my lab that I don't understand, and I think the only way is to stay in the lab.

How do you balance going to meetings with being in the lab?
That's extremely difficult. I get invited to several meetings, and most of them I cannot say 'no' to, because it's considered an important meeting, but I decided that I'm not going to a meeting more than once a month unless it's essential, because otherwise I would never be in the lab. But deciding when to go and when not to go is a very difficult task. As a young PI, you need to know what other people are doing, and despite the fact that people tend to present already published or at least accepted data, there are always several people who will present unpublished data. But I've sometimes been to meetings that are too far away from what we do. Knowing when my time investment is worth it is still a learning process.

How do you achieve a work–life balance, especially at the early stages of having your own lab? Ah, it's impossible! [laughs] I think at the beginning it's difficult, because you have a small lab, you want things to move forward, but you are used to a different pace from your postdoc and you can't match even half of that speed. You think: 'if I did it by myself, I would be here,' but the truth is: 'if I did it by myself in my former lab, then I would be here. But if I do it by myself in my own lab,I may speed it up a bit more, but I would still not be there.' That means you have to put a lot of time into the lab. I've been lucky that I've managed to recruit good people whom I can trust. Although I have a bit more work–life balance now, I still feel that the lab needs me a lot. This feeling will never disappear, but at some point you learn how to achieve a balance.

Could you tell us something about yourself that you wouldn't put on your CV?
There's a lab story from my PhD. My lab mates said that they would never tell me when they were going for lunch, because whenever they said "we are going in 5 min" I would say "I will be ready", and I would never be ready. There was always some extra experiment I wanted to do. In the beginning they would wait for me, and sometimes they waited for half an hour! They also said "you are like Einstein, your time stretches! Your 5 min are always at least 30". At some point they decided they would just go, and I could join them if I could. And I'm still the same, so people in my group just come and say "we're going for lunch" and then they just go.

For the above interview, Meritxell Huch talked to Anna Bobrowska, Editorial Intern at Journal of Cell Science. The piece has been edited and condensed with approval from the interviewee.

How would you describe your research to a general cell biology audience?
We're interested in understanding the process of regeneration- and why certain organs like the pancreas regenerate so poorly, and others, like the liver, so incredibly well. We approach this question by investigating the mechanisms cell use to sense damage, activate a progenitor programme, proliferate, and finally sense when to stop proliferating and differentiate into regenerated tissue. We use two parallel approaches- animal models and organoids,

which are surprisingly good at mimicking many aspects of regeneration. We're also interested in understanding the consequences of chronic disease- the liver does not have an unlimited capacity to regenerate. Moreover, failure can take two forms: a loss of function, fibrosis, but the tissue can also amplify mutations and cause cancer during the actual regeneration phase. You might have a dormant mutation in a single liver cell with no phenotype since the organ is not actively proliferating- until the tissue is damaged and forced to regenerate, resulting in a clonal tumour. This is still a hypothesis- but the indirect evidence is that damage involves sometimes fibrosis, and sometimes cancer.

Did this research programme evolve naturally from the work you carried out during your postdoc?
Yes, in some ways! The intestine and the stomach are very similar- able to maintain a highly proliferative state homeostatically- and this allowed us to study and understand mechanisms that underlie the simultaneous proliferation and differentiation of epithelial tissues in these organs. Then we moved on to the liver, which does not really proliferate until it is damaged. One of the things we discovered during my postdoc was that Wnt signalling was a key player in both intestinal and stomach proliferative pathways. We then found that Wnt signalling is upregulated upon damage in the liver. This was one of the key pieces of data that led us to hypothesize that liver regeneration could be mimicking constitutively active pathways in other organs.

Did this finding come as a big surprise?
Not quite, but we managed to provide the proof! What was unexpected, however, was that we were able to produce the conditions to maintain stomach cells from primary tissue in culture, for long periods in time. It was even more surprising that this approach worked for the liver- because we were working with healthy cells that were not proliferating at the moment of isolation. Under the culture conditions I had developed, we were able to get them to induce and maintain the proliferative state.

Back in the present- you've had your lab for a few years now- is there one aspect of academic life that you find especially challenging?
Yes- it is sometimes challenging to be able to do everything at the same time. You have to be able to attract funding, supervise students, produce good science, communicate it to the world- while combining all of these activities with family life. Somehow you have to pull this off without becoming obsessed with or too focused on just one aspect to the exclusion of all others. I for one find it difficult to not get obsessed with the science!

Do you find striking a balance is easier now than when you were younger?
Yes! In a way, it's like training your muscles. The day after you go to the gym for the first time, you're destroyed. But after 2 weeks of regular visits, you're not destroyed, and in fact you're capable of going further than you could before. When I was a student, I was stressed, but it was so much more overwhelming than now. When I look back, in fact, I can no longer even see the reasons for the stress! Science makes you stretch yourself every day, and even though it feels like you're doing the absolute maximum at each moment in time, when you take a step back you find you're capable of more and more.

How do you unwind outside the lab?
Outside the lab, I am the wife of my spouse, the parent of my child, the daughter of my parents. Non-lab time is family time. I love playing the piano and dancing- but I've stopped practicing and therefore I've stopped playing entirely- I can no longer stand to listen to myself! Once a week at least I find an hour or so to listen to classical music, and while it's certainly not the same as being in the theatre, I still love it.

The BSCB award for women in cell biology is an implicit acknowledgement that the odds have historically been so stacked against success for women in academia. Have you seen evidence for this imbalance in the institutions that you've been associated with, and do you feel things are headed in the right direction?
Personally, I've never felt like I was treated differently because I was a woman. I've never felt like that. That said, I've noticed obviously that as you climb the academic pyramid, you see women getting left behind. You see equal numbers at the PhD and postdoc levels but you see fewer female leaders, fewer senior female speakers at conferences, even in fields where this could be avoided. I think things are changing for the better- I've seen a change from when I was student until now, for sure.

Any parting advice for postdocs looking to follow in your footsteps?
This is not a regular job- you need to have the energy and persistence to accept a huge amount of failure along with the success. If you don't have passion for the scientific questions you're trying to answer, I think you might be setting yourself up for failure. On the other hand, if you're asking good questions, that you care about, you have some idea about how you will answer these, and you can attack them persistently- I'd say you're in great shape to start your own lab and take it in exciting directions.

For the second interview, Meritxell Huch talked to Gautem Dey, BSCB Postdoctoral committee representative, University College London.

Science Writing Prize Winner 2018 – Alex Binks

Of Monsters and Genes: using AAV as a tool in the fight against childhood blindness

Alex Binks (@binknabel) is a final year PhD student at the University of Glasgow, currently completing the remainder of his studies on a secondment at Imperial College London, supervised by Prof Iain McNeish. Alex's research revolves around oncolytic ("cancer-killing") viruses and how the mechanisms of killing that these viruses employ can impact the immune system. Outside of the lab, Alex enjoys finding ways to engage the public with his science via articles and videos.

For many, the mundane act of tucking your child into bed at night can present as quite an ordeal. Settle them down, get them in their PJs, check the wardrobe for monsters, read a bedtime story, check for monsters again, lights out. This issue of monsters needs to be taken seriously: even with some tactically placed night-lights, and a NERF gun at the ready, sometimes darkness and the supernatural prevail and the parental bed gains an extra guest for the night. Thankfully, most of us outgrow our negative relationship with the dark, but for some children, these nights are only the beginning.

Children living with Usher syndrome never escape the darkness. For them, darkness only grows with time. Nights become blacker as they lose all ability to see below certain light levels. Those night lights and NERF guns may as well be gone as objects become harder to make out. Eventually, the darkness begins to visit them during the daytime as they see their peripheral vision close in, tendrils of blackness creeping in from every angle.

Thankfully, Usher syndrome is extremely rare, affecting approximately one in 10,000 people. In addition to the gradual onset of blindness, sufferers are also deaf from birth, which can immensely impact their abilities to learn and communicate. Usher syndrome is a genetic disease, which can be caused by a mutation in a gene called CDH23. There are currently no treatments or cures, which is leading researchers to explore some inventive approaches, such as gene therapy.

Much like changing a flat tyre on your car, the premise of gene therapy is simple: if a gene is broken, provide the cell with a new one that works. Despite this, these days it's clear that achieving successful gene therapy is perhaps more akin to rolling a tyre down an assault course with fire pits and swinging axes and hoping that when it gets to the car, it has the manners to hop onto the axel itself.

While difficult, there are still ways to make the process of throwing genes at cells a little more elegant. For starters, targeting areas of the body that you can reach with a needle (e.g the eyes) substantially reduces the number of swinging axes our new genes come up against. Using biological tools which can stand in for qualified mechanics can also make the end switch much more possible. Enter viruses.

Viruses are fascinating objects of nature. In many cases, consisting of just some genetic information and a protein coat, viruses roam the expanses that are our bodies, seeking cells that they can hijack for their own nefarious needs. Viruses enter cells and take over their machinery, convincing them to read the viral genes as if they were the cell's own. This means that cells are tricked into producing and assembling a new generation of viruses, each ready to head off and find their own cellular fools.

While viruses can be troublesome and, in some cases, deadly, the traits that make them great biological spies are exactly the traits that make them outstanding tools for gene therapy. By cleverly switching out some of the key genes for making viruses, and replacing them with, say, CDH23, we can in one quick motion remove the ability of the virus to cause harm and prime it for repairing our broken eye cells.

One of the more popular viruses used today is called 'adeno-associated virus' or 'AAV'. AAV is a great gene therapy virus because it's extremely safe and can infect cells that aren't dividing – like many of the cells in our eyes. One unfortunate drawback of AAV is that it's so tiny. Clearly, all viruses are tiny, but AAV dwarfs many of these by a long way. AAV has a genome length of just 5,000 base pairs. This means that of all those As, Gs, Cs, and Ts that code for our genes, there are only 5,000 in a line from start to finish. To put that in perspective, that's 25x smaller than say, the chicken pox virus genome, or 600,000x smaller than the genome of humans. Unfortunately, this means that not only can you not fit many genes inside of AAV, but some genes won't fit at all. This includes the Usher syndrome gene, CDH23, which is 10,100 base pairs long.

The scientists behind a recent study published in Cell have valiantly taken this problem on. They reason that if a gene won't fit into a virus's shell, then why not chop it into pieces? Imagine a family of very tall people all trying to fit into the same Mini. If there's not enough legroom to go around, it makes much more sense to take separate cars. In the same vein, researchers took the CDH23 gene, and placed it into three separate AAV vectors.

The key to making this work was finding a way to get the three gene pieces to assemble back together again once inside the cell. This involved flanking each gene piece with special 'recombinogenic' and 'splicing' sequences. The recombinogenic sequences are used for the sticking; like two Velcro pads at either end of a

piece of fabric, the cell uses these sequences to assemble the gene into one. However, this leaves rough sequences in the middle of the gene, making it impossible to read. This is where the splice sites come in. These sequences tell the cell to chop out the intervening recombination parts, much like instructing someone to diligently sew together the Velcroed fabrics, leaving one uninterrupted, readable sequence.

The researchers showed that when these viruses were injected into the retinas of mice with the CDH23 mutation, levels of full-length CDH23 protein were shown to increase. Unfortunately, this system cannot show whether the increase is enough to reverse any of the effects of the disease.

This research hopefully provides some light at the end of the tunnel for children suffering from Usher's. Maybe one day, AAV will be just another weapon in the fight against monsters in the dark.

We are living in uncertain times

We are living in uncertain times, a situation which affects us both as citizens and scientists. Here in the UK, we are about to embark into uncharted waters as we prepare to leave the European Union, which has provided a source of peace and prosperity for this country. What's more, the EU has given us a net-gain scenario in terms of science funding, and it's still not clear whether that dividend will be replaced. Meanwhile, more internationally, we have all seen the trend towards disregarding the experts and a wholesale turning away from truth and evidence-based thinking in favor of unverifiable sources, 'fake news' and conspiracy theories. This trend is a threat to our society, to our way of life – and in all likelihood to our planet.

In the face of all of this bleakness, it is tempting to want to hide away in our labs and bury ourselves in the familiarity of our research. But the last thing we should be doing is turning our backs on society. We have to raise our voices and fight on the side of rationality, defending both the funding and processes that keep our research ecosystem healthy, and advocating for the evidence-based policies that will see us through the great global crises that loom ahead: climate change, dwindling fossil fuels, antimicrobial resistance to name but a few.

Can one person make a difference? Can one learned society? I am convinced that individuals, as well as the wider scientific community, can, if they work together and think strategically.

This is why I was so pleased to have been appointed the Science Advocacy Officer for the BSCB – a new role on the Committee. But what does this actually mean?

In essence, the role will provide a link between the BSCB and the broader community lobbying for science in the UK. As many of you will know, the BSCB is a member of the Royal Society of Biology, a charity umbrella group that seeks to be a unified voice for the life sciences and which incorporates a number of learned societies. The RSB has as its mission to influence and advise the Government on policy, to facilitate education and career development, and to engage with the wider public about biology. Influencing politics in particular is an aim that is much easier to achieve with one voice and with a large number of people behind that voice. The BSCB could attempt to influence on its own, of course, but we will be much more effective if we stand shoulder to shoulder with many others in the scientific community and send a united message.

So I will be liaising with the RSB on matters that affect our own community. One of the most powerful things we as a society can do to help the RSB's mission is to feed into their submissions to various Parliamentary calls for consultation on scientific matters. These consultations crop up regularly, and I will be working with Judith Sleeman, our Web and Social Media Officer, to bring these items to your attention so you can let us know what matters to you and how you would like us to respond as a cell biology community.

One key aspect of lobbying the Government will of course revolve around science funding. In recent years the Government has given UK science some badly needed cash infusions to help reverse the lingering trend of real-terms cuts in spending that had been in place for some time. In fact, the Government has committed to a target of investing 2.4% of GDP in research and development by 2027 (and eventually, up to 3%). This ambition came as a relief to UK scientists, as for many years we've been sorely in need of a longer-term vision and budget to allow researchers to be strategic rather than reactive. This 2.4% goal sounds lofty, but it needs to be underpinned by actual commitments to increasing science funding year on year to meet the target. The Autumn Statement back in October did not seem to deliver on this, so we need to keep an eye on Government and hold them accountable. With austerity still seemingly with us, and with Brexit uncertainty in the wings, it's certainly not a good time falter on investing in science, which research has shown reaps a substantial return towards economic growth.

The BSCB is also dedicated to other missions that the RSB aims to facilitate, including diversity in science, science education and more effective public engagement. Equality and diversity will help keep the scientific community not only a fair place, but also a more healthy and creative one. And education and engagement will be absolutely crucial in what is shaping up to be an epic battle between rational citizens and the forces of untruth.

I hope you can join us on this worthy mission.

Dr Jennifer Rohn

Meet the BSCB committee: Susana Godinho

Susana Godinho is a Lister Prize Fellow and Senior Lecturer at Barts Cancer Institute. She joined the BSCB Committee in XXXX.

1) What's your role on the committee?
I was elected as a BSCB member in 2016, and I am one of the junior members. I participate in the scientific committee meetings where we discuss all issues related with the society, including conference organision, Hooke and Women in Cell Biology medals, finances, our magazine etc. It has been quite rewarding to be able to have a voice in this society, which I value so much.

2) Over the next year what will be you be up to for the BSCB?
This last year I took on board extra responsibilities at the BSCB. I am co-organising the next BSCB-BSDB joint spring meeting in April 2019 with BSCB member Vicky Sanz-Moreno, our meetings officer Anne Straube and our counterparts at the BSDB. This has been quite a fun experience, putting together the progamma, inviting the speakers. We have a great line up for 2019 and I am really exicted about that. In addition, together with my fellow BSCB committee member Stephen Robinson I am helping putting together the material that goes into the BSCB magazine. This magazine goes to all BSCB members and it is a good way to keep up to date with what the society does.

3) Aspirations for the BSCB?
My main aspiration for BSCB is for the society to continue its fantastic work in promoting cell biology, diversity and support for researchers. What I would like to see is a better reach of the society and its activities to a wider audience.

4) Could you describe your research in a nutshell?
My lab works on morphological abnormalities that occur in cancer cells and how these impact cell physiology. We are particularly focused on centrosomal abnormalities that occur mainly in tumours. We study their impact on cell division and how they contribute to tumourigenesis.

5) What inspired you to come into Cell Biology?
Actually my interest in cell biology started when I was in college and learned about bacteria physiology, how they adapt and survive to harsh conditions. I thought it was fascinating to learn about the biology of these cells. Many things changed since then but not my love for cell biology (small and big cells!).

6) What's been your best moment as a Cell Biologist?
That has to be when we are able to understand how something complex works based on your findings. It does not happen often, and most of the times it takes many years and the work of many people, but when it does is truly magical.

7) What do you feel are the biggest challenges facing Cell Biology?
Unfortunately the importance of Cell Biology is often underestimated by funders and policy makers. It is easier to explain more applied studies to a broader audience. However without the fundamental science there is no applied science. In my opinion the biggets challenge for Cell Biology is to attract a healthy stream of funding so that progress can be made.

8) If you were to start your PhD today what would be the emerging topic you would like to focus on?
I recently became fascinated with cell–cell communication. We know so little of about cells communicate and how this communication imparts on their biology and response to stresses. This is also very important in cancer as it is clear the tumours behave a bit like an "ecosystem". In my opinion we need to undertand the biology behind cancer cell communication if we want to eradicate tumours.

9) At the BSCB meeting where would we be most likely to see you?
That's an easy one! Either checking our posters (always fantastic science at poster sessions) or at the bar having a pint and chatting!

10) What's your favourite cell and why?
I like all cells, even bacteria. But as cell biologists I like big cells that I can culture *in vitro* like the retinal epithelial cell line RPE-1. They also have beautiful mitotic spindles, so that's a plus. In the body, I really like the multiciliated epithelial cells. I think they are cool!

Meet the BSCB committee: Julie Welburn

Julie Welburn at the Wellcome Trust Centre for Cell Biology, University of Edinburgh, is the BSCB's Honor Fell/COB Coordinator.

1) What's your role on the committee?
I am responsible for the Honor Fell Travel awards, Course awards and PI fund awards. Funnily enough, Honor Fell was an undergraduate at the University of Edinburgh (1918–1922). Her portrait is up in one of our corridors.

2) Over the next year what will be you be up to for the BSCB?
I give travel awards and awards for care/childcare out. The winter and spring are really busy as many people apply to attend spring and summer meetings. I am also sorting out the BSCB merchandise, so watch out for those BSCB mugs and pens.

3) Aspirations for the BSCB?
The BSCB is there to support all UK-based cell biologists and help people know each other and work together. All the committee works hard to provide this support and with a rotating community, many people have an opportunity in their career to get involved. I hope we can continue to support Cell Biology in the UK.

4) Could you describe your research in a nutshell?
We are interested in the role of microtubules and motors in cell organization and transport at the molecular level. We use the mitotic spindle as a model, as it is highly spatially and temporally regulated by microtubule motors. However I am also interested in looking at how microtubules and motors provide cell organization in other cell types. Very little is known about how molecules are transported to the right place to build functional cells.

5) What inspired you to come into Cell Biology?
I always liked sciences and did a degree in Biochemistry. I did some undergraduate work and a PhD in Structural Biology. At the end of my PhD, I wanted to be able to test our structural models. That's why I went to a cell biology lab for my postdoc. Now we do both cell biology and structural biology!

6) What's been your best moment as a Cell Biologist?
As a PhD student, after one year of taking crystals to the synchrotron that did not diffract, I got diffracting crystals to 2.5Å. It was pretty emotional moment! Then during my postdoc, there was a key experiment I did. Iain, my postdoc adviser made me come on the 1st January to develop the Western blots. I was not very happy, but got this great result, which was key for our *Mol Cell* paper. And then in my group, my postdoc presented key results in a group meeting – she waited until then to tell me. It was a great surprise!

7) What are the biggest challenges facing Cell Biology?
Cell biology and science in general are becoming more and more competitive. We all face the same problems, competing for grants, competing for students – we have to fight to survive for limited resources. Competition is healthy but too much competition is counterproductive and destructive. The other challenge is the fact most funding bodies want to fund applied science; basic science and blue sky research are not valued as much. Diversity and basic science in research is essential for downstream applications! There are many examples of that out there – look at CRISPR. Originally it was discovered studying the defense mechanism in bacteria against viruses and hard to get funding for. Now CRISPR is revolutionizing how we do experiments and has potential for therapies on day.

8) If you were to start your PhD today what would be the emerging topic you would like to focus on?
My problem is that there are so many interesting things to find out. I would look into ecosystems and how they adapt to climate change. The planet needs to be looked after. I love Eric Karsenti's Tara project to study ocean and climate change. And he used to work on the cytoskeleton before!

9) At the BSCB meeting where would we be most likely to see you?
I would be talking to some PhD students and postdocs about their work, their life in their lab and exchanging fun stories with them. It's also great to meet with UK colleagues and catch up over breakfast and lunch. At Dynamic Cell, I really enjoyed meeting the students and postdocs that I have given Travel awards to. It is nice to put a face to a name.

10) What's your favourite cell and why?
Bacteria and Sf9 cells! Because they help us make proteins to do biochemistry.

1909 Bedside to Bench, 2018 Bench to Bedside

The Institute of Cancer Research, London is collaborating with The Royal Marsden NHS Foundation Trust.

Even if you have nothing to do with cancer research you will likely have seen in both the higher education and scientific press that The Institute of Cancer Research (ICR) came 1st of 154 institutions in the 2014 Research Excellence Framework (REF). More recently, in 2017, the ICR was ranked in joint 5th place out of 1,200 higher education institutions in the world across seven research criteria. So where is the ICR located and how does it operate?

Location
The ICR has two sites; one in traditional style buildings in Chelsea, London and the other in newer buildings in Sutton, S. London. At both sites the ICR has firm links with The Royal Marsden Hospital and it is through these links that a unique synergy has been developed. Some ICR staff are consultants at The Royal Marsden.

Bedside to bench
In 1851 Dr William Marsden founded a hospital in Chelsea. Following the death of his wife from cancer, Marsden, with a few friends, established an institute for the free treatment of cancer diseases. In 1909 it became the 'Cancer Hospital Research Institute'. A research building was opened in 1911 with the intention that the Institute should "conduct research on a broad front and in which every branch of science bearing on the subject of cancer should be represented." – This was a very important statement and is mirrored in the current operation of the ICR. 1939 saw a move of the Institute to a new site in Fulham Road, Chelsea and renamed as 'The Chester Beatty Research Institute'. In 1954 there was a change of name to 'The Institute of Cancer Research' (ICR).

Bench to bedside.
With the formation of the NHS in 1948 the ICR became independent of The Royal Marsden and in 1956 set up an additional and new site in Sutton, S. London where it also partners with the second site of The Royal Marsden. In 1999 the Chester Beatty Laboratories in Chelsea were redeveloped and extended to incorporate the 'Break-through Toby Robins Breast Cancer Research Centre' (now renamed 'Breast Cancer Now').

The ICR in 2017
The ICR is currently led by a Leadership Team of Chief Executive and President, Professor Paul Workman, Chief Operating Officer Dr Charmaine Griffiths, and Academic Dean, Professor Clare Isacke, a past President of the BSCB. Corporate services to ICR are provided by twelve groups covering areas such as Human Resources and Development.

Research at the ICR is formally divided into eight Divisions. Those for Breast Cancer, Cancer Biology and Structural Biology are at Chelsea. Cancer Therapeutics, Clinical Studies, Genetics and Epidemiology, Molecular Pathology and Radiography and Imaging are based in Sutton.

An example of a Division is that of 'Cancer Biology' where Professor Jon Pines, ex Membership Secretary of the BSCB, is Division Head. The Division has a focus on genome stability and encompasses research on cell division, cell signalling, cell shape, and cell migration. Each Division is a collective of Teams, each with a Team Leader. In addition there are numerous Centres, Units and Initiatives. Some of these are established within Division but others cut across Divisions and drive and promote collaboration, as envisioned by the first Director in 1909. This philosophy is keenly promoted by the present Chief Executive, Professor Paul Workman in its modern concept as 'Team Science', and is integral to research at the ICR. An example of a Team is that for Dynamic Cell Systems and led by Dr Chris Bakal who is a current member of the BSCB Committee. Ex BSCB Postdoctoral Representative Dr Alexis Barr works in this team as Senior Researcher on cell cycle G1/S transition and its dysregulation in cancer cells. Chris Bakal's Team is also is part of the Integrative Network Biology Initiative. Initiatives can exist across teams.

An example of a Centre is that of Professor Mel Greaves' Centre for Evolution and Cancer which is investigating amongst other things, the evolutionary resilience of cancer. Units can focus on one issue, such as Dr Amanda Swain's 'Tumour Profiling Unit' set up in 2013, or the 'Enterprise Unit' which is responsible for manoeuvring enterprising ideas from the whole of the ICR into the market-place and hospital.

In 2003, the ICR became a full college of the University of London and recognised for research and postgraduate teaching. The ICR has as its mission

statement 'Making the discoveries that defeat cancer'. Since 2005, twenty drug candidates have been discovered with prostate cancer drug abiraterone now used worldwide.

The current research strategy is built upon four pillars:
[1] Understanding cancer's complexity,
[2] Innovative approaches,
[3] Smarter kinder treatments
[4] Making it count.

The Learning and teaching strategy is structured around three pillars of activity:
[1] Providing world-class degree programmes,
[2] Teaching tomorrow's leaders today's discoveries,
[3] Partnering with our peers and the public.

At the start of the 2016/17 academic year, 61% of the intake were females, 39% males. 117 were registered as PhD/ MPhil students and 24 on the MD (Res) course. Also available is a day release course for clinicians leading to an MSc in oncology.

ICR: The future

The ICR has a staff of 1027 people. 67% are located at the Sutton site and 33% at Chelsea. Plans are being prepared for the Sutton site of the ICR and The Royal Marsden to be expanded and developed into The London Cancer Hub with about 10,000 people employed within the Hub area. Recently a grant of £30 million has been awarded towards a new ICR Centre for Cancer Drug Discovery. To enable resources, skills and talent to be jointly used, the ICR have teamed up with Imperial College to create a virtual Cancer Research Centre of Excellence.

Compiled by David Archer on behalf of, and with the assistance of past BSCB Committee members, Professor Clare Isacke, Professor Jon Pines, and Dr Alexis Barr of The Institute of Cancer ResearchGrateful thanks are due to The ICR communications Team for kindly supplying information.

Hooke Medal and WICB awards, and Summer studentships

The **Hooke Medal** is awarded every year by the BSCB and recognises an emerging leader in cell biology. It is given to an individual who has made an outstanding contribution to UK Cell Biology. This is usually been within the first 14 years of establishing their own lab. The medal is presented annually at the annual Spring Meeting, after which the winner delivers their research talk.

BSCB Women in Cell Biology Early Career Award Medal. This will be an annual honour awarded to an outstanding female cell biologist who has started her own research group in the UK within the last 6 years, with allowances for legitimate career breaks. Applicants must also have published at least one senior author paper from their own laboratory.

Candidates for both awards can be nominated at any time but must be nominated by at least one BSCB member, should provide a full CV and a recommendation letter with a short summary of the candidate's major contributions to cell biology. Submission should be sent to the BSCB Secretary.

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 post-graduate research career in cell biology after their undergraduate studies. Applications must be made by the prospective supervisor, on behalf of a named student. Supervisors must be a BSCB member for a minimum of one year before, or on the date of, the application. The research project must be on a topic in the broad area of cell biology and must not form part of the student's normal degree work.

Meeting Reports

3rd British Microtubule Meeting

30th April 2018, Edinburgh

The sun was picking out the gorse shrubs on Arthur's Seat as delegates arrived in Edinburgh for the third edition of the British Microtubule Meeting. This year the meeting was organised by Julie Welburn (Edinburgh), Bungo Akiyoshi (Oxford), Andrew Carter (Cambridge) and Steve Royle (Warwick). The venue was the National Museum of Scotland, and the route up to the lecture theatre wound its way past an exhibit on Alexander Fleming. Venturing further into the museum the day before, I had discovered that while this was, first and foremost, a grand space for a scientific meeting, it also had the added bonus of being the home of Dolly the sheep.

Kicking off the first session of the day, Michelle Peckham (Leeds) introduced actin and tubulin affimers as an exciting alternative to antibodies in research, demonstrating their remarkable ability to access the cytokinetic furrow due to their small size. This was followed by diverse talks ranging from medical applications of modulating microtubule stability (Yiyan Zheng, Oxford) to functional studies of kinesins (Nida Siddiqui, Warwick).

Mathematical approaches were well represented at the meeting, which again highlighted its interdisciplinary feel. Aleksandra Plochocka (Edinburgh) gave a beautiful talk on how microtubules become aligned within cells in the developing *Drosophila*. She mathematically modelled microtubule dynamics to offer a solution to the chicken or egg problem of 'what comes first, cell geometry or microtubule organisation?' Meanwhile, Mustafa Aydogan (Oxford) presented the first evidence of sub-cellular oscillations regulating the growth of an organelle. He demonstrated how mathematical modelling is providing a deeper insight into the mechanism of this process.

After coffee, Victor Garcia from the Acton lab made his first foray into the world of microtubules, unveiling dendrites as initiators of microtubule network remodelling in fibroblastic reticular cells. Steering us from immunology to neuroscience, Dhanya Cheerambathur (UCSD/Edinburgh) revealed exciting roles for C. elegans kinetochore proteins away from the chromosomes. For the last two talks of the session, meiosis was very much the focus: Pierre Romé (Edinburgh) presented data suggesting a role for subito (which he showed is a γ-tubulin complex interacting protein) in meiotic microtubule nucleation, a process that occurs without centrosomes. Weronika Borek's (Edinburgh) work focuses on the other end of the meiotic spindle microtubules; she presented work

suggesting that the CCAN kinetochore subcomplex is necessary for proper meiotic kinetochore-microtubule attachment.

Thirty-two posters were exhibited over the lunch break, giving the chance for attendees to discuss the varied range of their work. The third session followed, offering three talks from the perspective of structural studies. Clinton Lau from the Carter Lab in Cambridge discussed how cryo-EM has revealed that dynactin can bind two dyneins, thus allowing faster movement along microtubules. Meanwhile, Joe Cockburn (Leeds) presented structural studies that uncovered how kinesin-1, another motor protein that moves in the opposite direction to dynein, is activated by cargoes such as JIP3. Exciting work from Szymon Manka (London) used cryo-EM alongside microtubule stabilisation with doublecortin (DCX) to capture the structural transitions that occur as microtubules destabilise. He posited that GTP hydrolysis promotes microtubule compaction,

strengthening the longitudinal interactions between the tubulin polymers that comprise the overall structure. Meanwhile, lateral contacts between tubulin polymers loosen, promoting microtubule peeling and a catastrophe event.

The meeting was closed by a talk from Amy Barker (Oxford), which focused on how the symmetry of the trypanosome flagellum is broken so that different proteins are not evenly distributed along its entire length. Trypanosomes are responsible for major human diseases such as sleeping sickness, and a parasitic theme was felt throughout the final session: work from preceding speakers Hanako Hayashi (Oxford) and Mohammad Zeeshan (Nottingham) focused on kinetochore proteins in other kinetoplastids and on the role of kinesin-8 in malarial parasite endomitosis respectively.

As the shadows began to lengthen, the attendees headed over to Pollock Halls for dinner, and the opportunity to take part in the end of conference quiz. Picture clues representing various famous scientists was a highlight, with 'Eye-sack Newt-on' being a particular triumph. With the meeting coming to a close, it was time to reflect on the fantastic science that we had heard and to look forward to coming together again next year!

Laura Hankins, PhD student at Oxford University

EMBL Conference: "Transcription and Chromatin"

25–28 August 2018, Heidelberg

{ I am hugely grateful for the PI Travel grant from the Company of Biologists and BSCB that allowed me to attend the 2018 EMBL "Transcription and Chromatin" conference high up on the hill behind Heidelberg. This biennial conference brings together over 400 scientists from around the world to discuss the latest findings in the field of gene regulation, focusing on the factors that regulate transcription and the influence of chromatin structure and modifications on them. }

The conference was scientifically intense, with over 60 talks and almost 300 posters to digest over three and a half days. However, there was ample time to chat about the science while digesting dinner in the esteemed EMBL canteen and supping post dinner drinks, enjoying the view from the Rooftop Lounge of the EMBL Advanced Training Centre. Obviously, there were too many presentations to cover here, but talks were well summarised on Twitter using #EMBLtranscript.

Hot topics at the 2018 meeting included investigating how the 3-dimensional arrangement of genes within the nucleus relates to their regulation, the unending quest to understand how Polycomb Repressive complexes are recruited to mammalian genomes, and the contentious role that phase separation may play in gene regulation.

In a field historically dominated by molecular biologists and biochemists, many labs are now using high resolution microscopy to visualize events associated with changes in gene expression. For example, Clodagh O'Shea from the Salk Institute talked about the development of FIREnano and ChromEMT techniques to visualize gene activation at the level of chromosomes in the very first plenary talk of the meeting. Mike Levine (Princeton University) presented visualization of the process of transvection (activation of a gene in trans by the regulatory region of its homologue) in living Drosophila embryos.

Another recent trend in the field reflected in talks at the meeting is that many biology labs are forming productive collaborations with colleagues from the fields of maths and physics to come up with novel insights into transcriptional regulation. For example, Jane Mellor (University of Oxford) presented work combining mathematical modelling and experimental data to give novel insight into the effects of antisense transcription on chromatin architecture and sense transcript dynamics. Leonie Ringrose (Humboldt Universität zu Berlin) presented a theoretical analysis leading to the proposal that "poised" chromatin is different from

"stable" chromatin due to switching state with higher frequency.

Other highlights included Patrick Cramer's presentation of chromatin the structure of the RNA polymerase II elongation complex at a resolution of ~3.1 Å using cryo-EM. Eileen Furlong presented the nifty use of Drosophila balancer chromosomes to demonstrate the relatively minimal effects on gene expression associated with multiple chromosome arrangements that disrupt Topologically Associating Domains (TADs). Finally, Paolo Sassone-Corsi (University of California, Irvine) presented convincing links between chromatin remodellers, metabolic pathways and the circadian clock.

The conference ended with Tony Kouzarides (University of Cambridge) declaring that the conference had been the "most successful transcription meeting ever" before we headed off to the conference dinner and disco.

Barbara Jennings, Principal Investigator at Oxford Brookes University

Report of the 10th Salk Institute Cell Cycle Meeting

26–29 June 2018

I travelled to the Salk Institute of Biological Sciences in San Diego via San Francisco for my first international conference and first conference solely focused on my field of interest. The meeting had invited speakers specializing in all aspects of cell division, it's role in cancer and the differences between organisms including one talk about starfish!

Tony Hunter from the Salk opened the meeting by giving a nostalgic history of how postdocs from his lab had transformed the field of cell cycle research beginning with my PI Jon Pines who began to characterize the fundamental role of cyclins in cell division. It was inspiring then to spend the next 4 days hearing a diverse range of talks that had in some way stemmed from this seminal research.

Controversially during a question and answer session a woman used her question time to point out that out of around 50-60 speakers only 12 of them were women and asked why this was. I wanted to mention this because I believe it is an important topic in science generally which must be discussed in order for it to be overcome. Tony Hunter and the other organizers promised to take this criticism on board for future meetings.

I then presented a poster on my research which focuses on quantitatively studying the Spindle Assembly Checkpoint in mitosis and how I intend to further our understanding of the SAC using gene editing and fluorescence correlation spectroscopy. I had a chance to discuss the challenges facing my project with experts in the field and in turn find out about new techniques and new discoveries in mitosis.

Overall it was a great meeting which allowed me to meet cell cycle experts from all around the world. It gave me many new ideas for my PhD and it also reinvigorated me to get back in the lab as soon as possible to test as much as I could. This trip would not have been possible without the Honor Fell travel award from the BSCB and the Company of Biologists and I would like to thank them for this opportunity

Jordan Holt

Keystone Symposium: Exosomes/ Microvesicles: Heterogeneity, Biogenesis, Function and Therapeutic Development

4–8 June 2018, Colorado

{ I travelled to the Salk Institute of Biological Sciences in San Diego via San Francisco for my first international conference and first conference solely focused on my field of interest. The meeting had invited speakers specializing in all aspects of cell division, it's role in cancer and the differences between organisms including one talk about starfish! }

In June this year, thanks to the BSCB's support, I attended the Exosomes/Microvesicles: Heterogeneity, Biogenesis, Function and Therapeutic Development Keystone Symposia. The conference was held in Breckenridge, a beautiful little town amongst the stunning rocky mountains of Colorado providing a beautiful location to enjoy the symposia in. The conference provided me with an incredible opportunity to present my work to an international audience, and to gain valuable insight into the movement of the extracellular vesicle field which is a fast moving and changing field. In recent years extracellular vesicles (EV) have emerged as critical mediators of intercellular communication, however the heterogeneity of these vesicles presents a significant challenge to determining the functional roles of these EVs. Attending this conference gave me the opportunity to listen to experts describe their most cutting edge science and gain insight into the newest and most successful methods of EV isolation and analysis which was immensely valuable to me and my work.

The jam-packed conference schedule included a wide range of talks that covered basic EV biogenesis to functional roles of EVs in disease. It began with talks on EV cargo and Delivery where key researchers in the field impressed the importance of understanding the basic biogenesis and functional delivery of EVs. One particularly interesting talk from Andre Leidal (University of California, San Francisco, USA) showed increased expression of the tetraspanin CD63 on EVs aided delivery of their cargo to the recipient cells whereas IFITM3, which can be robustly incorporated into EVs, prevents cargo delivery. We then switched direction into sessions focused on the functional role of EVs in cancer development and metastasis, where inspiring researches such as Clotilde Théry (Institut Curie, France) pointed out the importance of precise EV separation showing the distinct functional differences EVs of different sizes can have. Additionally, a key talk from David Lyden (Weil Cornell Medical Collage, USA) highlighted the role of EVs in cancer metastasis as well as novel discoveries, such as that of the exomere (the smallest EV to be discovered) that are advancing the field as a whole.

Afternoon lunch breaks provided time to take a stroll into the heart of Breckenridge, a lovely former gold mining town, or into the Rocky Mountains with colleagues, providing time to enjoy the breath-taking surroundings. The days were rounded off with dinner followed by poster sessions and drinks which provided an informal setting for students and established scientists to discuss new movements in the field and network. I was lucky enough to make some very rewarding connections and gained valuable insight into my own work during these evening sessions. A huge thanks must go to the scientific organisers Crislyn D'Souza-Schorey and David Lyden as well as the keystone symposia organisers for putting on such an informative and enjoyable conference. I look forward to attending similar events in the future!

Sophie Adams, PhD student at Barts Cancer Institute, Queen Mary University of London

Summer studentships

Hypoxia mediated effects on the activity of JmJC Family of Histone Demethylases

As I prepare for the 3rd and final year of my undergraduate genetics degree I feel confident taking on my dissertation having been given the opportunity to work with such a distinguished group of researchers at the university of Liverpool. I was funded by the British society of cell biology to work in a lab at the department of integrative biology with Professor Sonia Rocha, supervised by postdoctoral researchers Michael Batie, Julianty Frost and Mark Frost. Thanks to the guidance of Prof Sonia Rocha and Colleagues, I have gained a variety of technical skills and a greater understanding of many biochemistry techniques. I have long considered a career in research. However, after completing my 7-week studentship I am resolute in pursuing a PhD position.

Hypoxia is generally accepted as a decrease in oxygen availability. This causes a variety of changes to gene expression; these changes may be mediated by the HIF transcription factors. HIF are heterodimeric complexes with a constitutively expressed HIF-1 subunit and one of three HIF- proteins HIF-1 , HIF-2 or HIF-3 . The presence of the HIF- subunit is affected by the activity of dioxygenase enzymes FIH and PHDs which require oxygen to function. (FIH) Factor inhibiting HIF mediated hydroxylation of HIF- causes inactivation of the transactivation domain which subsequently prevents the binding of the coactivator protein p300.

During normoxia (PHDs) Prolyl hydroxylase domain containing proteins hydroxylate conserved proline residues of the Hif- oxygen degradation domain. This increases the binding site affinity of (VHL) Von Hippel Lindau tumour suppressor protein for Hif- . VHL is part of the E3-Ubiquitin ligase complex which targets Hif- for proteasomal degradation, preventing a hypoxia response. Hypoxia causes other changes in gene expression through chromatin accessibility. This is regulated through various mechanisms including (CRC) chromatin remodeller complexes, (PTM) post translational modifications and (ncRNAs) non-coding RNAs. PTMs include histone methylation and acetylation. Histone methylation is a dynamic modification of lysine and arginine residues of N-terminal histone tails. These modifications alter the binding site for chromatin binding proteins.

These methyl modifications are mediated by histone methyl transferases and histone demethylases which add and remove methyl groups respectively. Structural biology studies have identified various methyl demethylases. The class relevant to this project is the (JmjC) Jumonji-C containing histone demethylases. This class of demethylases are dioxygenases and therefore require 2-oxoglutarate, iron and oxygen for their catalytic activity meaning that reduced oxygen availability results in an increase in histone methylation marks since the JmjC containing proteins are unable to remove the methyl groups from their target histone methylation sites. The sites important to this project are targets of the KDM5 family of JmjC containing proteins, H3K4me3 (Histone 3 lysine 4 tri-methylation). I also investigated H3K9me3, H3K27me3.

Western blot analysis was used to investigate the activity of the KDM2, KDM4, KDM5 and KDM6 family of JmjC containing proteins after hypoxia and through reoxygenation. Well characterized mammalian cell types HeLa and HFF were exposed to hypoxia (1% oxygen) for 24 hours followed by reoxygenation for 10 and 30 minutes, 1, 2 and 4 hours. Different cell types HeLa and HFF show alternative changes in their methylation marks. These differences are determined by the activity of the histone demethylases after their reoxygenation.

Western blot assays for HIF-1 show low levels of protein in normoxia and have a significant increase after exposure to hypoxia for 24 hours. This is expected since the enzymes that control HIF-1 require oxygen to function. However, another major difference between the cell types is the rate that HIF-1 levels decrease during reoxygenation. HFF cells seem to decrease slower in relative HIF-1 protein compared to HeLa cells which seem to decrease beyond the point prior to hypoxic insult after 10 minutes of reoxygenation.

The most interesting difference between the cell types is the rate of change in the target methylation marks. H3K4me3 and H3K9me3 markers show similarities between them with differences between cell types. HFF cells show that levels of both markers begin to rise after 24 hours of hypoxia but continue to rise for up to 10 minutes after reoxygenation. This contrasts with these marks in HeLa cells which decrease after 10 minutes of reoxygenation. These markers also seem to rise again after 1 to 2 hours of reoxygenation while HFF cells seem to rise only slightly before falling again. There is a similar rate of change in the levels of the H3K27me3 marker between cells. HeLa cells rise to their highest levels and immediately begin to decrease steadily after reoxygenation begins. H3K27me3 marker levels in HFF cells begin rising after hypoxic insult and continue to increase until 10 minutes of reoxygenation before decreasing. Actin and histone H3 proteins were used as controls. It is currently understood that hypoxia does not affect expression of actin. Therefore, the level of actin should stay consistent after normalising the amount of protein using the Bradford assay.

Looking at the differences in the amount of protein and the rate of change of these specific histone demethylases target markers helps us to understand the activity of these proteins between cell types and how they respond to the introduction of oxygen to cells after hypoxia. Despite the short 7 weeks of my studentship I have gained more insight into the field of study that I may like to pursue.

I am sincerely grateful to Professor Sonia Rocha for giving me such an insightful and rewarding experience and to her research team for the constant support throughout my time in the lab. I would also like to thank the BSCB for the financial support which allowed me to undertake this studentship.

Shawn Cottrill

The role of actin nucleation and assembly proteins (ANAPs) in myosin-Va dependent organelle transport

I am a MSci Biochemistry and Biological Chemistry undergraduate student at the University of Nottingham. I have always had a vision of one day working in a research environment at the heart of new and exciting areas of science and this placement has sufficiently increased those desires. Over my 8-week placement with Dr Hume I have learnt many new laboratory techniques and have seen what it feels like to have a project of my own with the aim of working to a specific goal.

Before entering my third year of study and having a biochemistry and chemistry project of my own I wanted to increase my basic lab skills and this placement has surpassed my expectations, learning so much more than I thought I could in a short space of time. A big part of the placement I have enjoyed is having responsibility over my own experiments and working independently. However, Dr Hume and other members of his lab were always on hand to answer my questions and concerns making me feel welcomed and more inquisitive about all the work they are involved in. It has opened my eyes to how diverse and wide spread the field of scientific research is.

My project was focused around the 27 KDa protein, Rab27a. This Rab-GTPase has a crucial role in distributing melanosomes within melanocytes, giving pigment to the cell. Wild type cells show a dispersed distribution of melanosomes whereas when Rab27 is knocked down in cells the melanosomes become clustered indicating the importance of this small GTPase. I worked towards answering reviewer comments on a paper trying to make the mechanism of dispersion and the components that are involved in it clearer as the method by which Rab27 disperses melanosomes is not completely understood at the moment. The paper discloses that the GDP/GTP exchange factor for Rab27, Rab3GEP, has an important role in the activation of Rab27 but is not the sole component in this extensive mechanism of melanosome distribution.

As part of my project I tried to purify Rab3GEP to send to partners in Munich where exchange assays would be run on it with GTP and GDP. To purify this protein, I transfected HEK293a cells with adenovirus containing Rab3GEP bound to GFP. I used a GFPTRAP strategy to purify the protein in which the GFP tag binds to GFPTRAP beads whilst the other cellular proteins do not. To confirm the purity and quantity of my eluted sample I ran SDSPAGE gels which I stained and western blotted. I ran 6 experiments in total, each time trying to improve the yield and purity of Rab3GEP. I learnt I got a higher yield when performing small experiments in parallel instead of a bulk experiment and other such improvements to the protocol. Overall, I had three separate samples of purified Rab3GEP that will be sent to Munich.

Another aspect of my project was to investigate whether the exchange factors Dennd4b and RabIL3 had influenced the distribution of melanosomes. To do this, I transfected melan-a cells with siRNA that target the transcripts of these genes and controls. This allowed me to observe the effect of knocking down the production of the mRNA for these particular proteins inside the cell by viewing them on a confocal microscope. I then extracted mRNA from the cells to produce cDNA for taqman PCR to clarify the knockdown.

Another, more challenging, aspect of my project was trying to make an adenovirus allowing expression of a Rab27a fusion protein that would allow me to forcibly target other proteins to melanosomes, with the aim of examining their effects on melanosome transport. Unfortunately, I did not have very much success with this work due to problems that we eventually pinned down to our stocks of virus producer cells. This experience allowed me to realise not everything I do will work the first time but that what is important is to systematically troubleshoot these kinds of technical troubles.

My favourite part of the project was my cell culture work and everything related to it. It gave me a sense of responsibility to have my own cell flasks to look after and it was extremely fun to carry out experiments with the cells I was growing. I found the techniques used incredibly interesting as working that closely with cells is something I had not had the opportunity to do before. After completing the cell experiments, I enjoyed viewing them on a confocal microscope and seeing the different fluorescence the cells expressed as many of the experiments I carried out used molecules with either a GFP tag or an mCherry tag. Imaging the cells and then analysing the results was rewarding as I had gone through every step to get to the final image from growing the cells myself.

All this work would not have been possible without the help and guidance of Dr Hume and his team within the lab who went above and beyond to make my time in the lab an enjoyable one, so I would like to thank him greatly. The connections I have made over this summer are vast and talking to the other students in the lab such as ones undertaking their PhD allowed me to gain valuable knowledge to further help me decide the path I want to take after completing my degree. The biggest thank you is to the BSCB for granting me the funding to make all of this happen and giving me the most rewarding 8 weeks of my undergraduate life so far. Thank you!!

Sophie Twigger

Localisation and function of PP1 phosphatases during cytokinesis

I am a biological natural sciences student at cambridge about to start my third year. I hope to continue my education after completing my undergraduate degree; ideally by applying for a phd. In order to do this I need to obtain as much hands on research experience as possible. I was granted eight weeks of funding from the british society of cell biology which allowed me to live in cambridge and work in the universities department of pathology over the summer; I have been supervised by Pier Paolo D'Avino while working in his lab for the past 8 weeks.

The majority of my time has been spent using immunofluorescence microscopy to study the subcellular location of four proteins that were identified by a previous study in Dr D'Avino's lab as possible regulators of cell division. I was using the microscope to observe fixed cells undergoing cell division and to determine if and when these proteins localized to the midbody during the final stages of cytokinesis. In addition to this I have been using siRNA to knockdown two different proteins of interest and using the same methods of immunostaining to examine the surviving cells for unusual distributions of proteins during cell division. To do this I have had to learn how to grow and maintain HeLa cells in artificial culture; how to fix these cells with either

formaldehyde or cold methanol and how to prepare appropriate dilutions of primary and secondary antibodies to use as stains.

I feel I have learned a lot in my time in the lab. In particular I found it both interesting and challenging to be involved in all stages of the imaging process; starting with cells growing and continuing to edit and combine the channels and images after they have been taken to produce clear figures for each protein. I hope to be able to use this experience of working independently in any research projects I pursue in the future and am very grateful to the BSCB for giving me this opportunity to learn.

Matthew Bagley

How does actomyosin contractility affect nuclear shape and DNA damage in metastatic melanoma?

Over the summer, I had the opportunity to get hands-on experience in Dr. Chris Bakal's Dynamical Cell Systems team at the Institute of Cancer Research (ICR). Metastasis is the spreading of cancer cells from the original site, via the blood and lymph, to establish new tumours in other organs. This spreading makes its very hard to contain and treat metastatic cancer. During metastasis, cancer cells must squeeze through tight spaces resulting in dramatic changes in cell shape. This requires both actomyosin contraction and extreme deformation of the nucleus, which can result in DNA damage. Consequently, I investigated the interactions between DNA damage and the contraction of actin-myosin complexes in mammalian skin cancers, also known as melanoma.

Initial measurements of the baseline levels of DNA damage and actomyosin in melanoma cell lines of various genotypes showed large differences. These levels changed significantly and were correlated in response to various treatments, such as replication or actomyosin inhibitors. We tested how the DNA damage response, nuclear shape and actomyosin contractility were affected under different doses and combination treatments. In some cases, the data we gathered in our experiments were consistent with clinical observations of patients' response to these treatments.

I spent the first week conducting an in-depth review of the existing literature relevant to my project. This allowed me to hit the ground running when I started work-shadowing Dr Lucas Dent, a post-doctoral researcher in the lab. I was exposed to the entire workflow of typical experiments, from maintaining cell cultures to transfections and drug treatments; and from antibody staining to image analysis. He helped me to quickly progress from analysing previously gathered data, to planning and executing my own experiments followed by data analysis. As a result, 8 weeks on, I have been equipped with valuable practical skills and specialist knowledge in mammalian cell signalling, increasing my confidence in pursuing a PhD to explore these concepts further.

The studentship was further enhanced by discussions during numerous seminars on the cutting edge research of others at the institute. I also had the opportunity to present the literature review I had conducted to members of my host lab, helping me to improve my presentation skills through their constructive criticism.

I'm thankful for the BSCB Summer Studentship bursary to allow me to expand the horizons of my degree beyond structured labwork and into the arena of more independent, articulate and novel research. I look forward to leveraging this experience while looking for post-graduate roles in cell biology research.

Hansa Shree

Investigating the cellular complexity of drug-induced cardiotoxicity by fingerprinting single cell mass spectral information using Time-of-flight Secondary Ion Mass Spectrometry (TOF-SIMS)

I am currently studying Natural Sciences at the University of Cambridge and am about to enter the final year of my degree. With aspirations for a career in scientific research, I was keen to obtain some research experience which could provide more realistic insights into the field than those offered by university practical classes and lecture courses. I was fortunate enough to have the opportunity to complete a BSCB-funded eight-week studentship in the Cunningham laboratory at the Strathclyde Institute of Pharmacy and Biomedical Sciences, University of Strathclyde.

PZ-128 is a peptide mimetic (pepducin) clinical trial candidate (NCT02561000) which antagonises the protease-activated-receptor-1 (PAR1) activity in platelets via an allosteric mechanism at the receptor-G-protein interface [1,2]. It is at the clinical trial stage for use as an antiplatelet drug in the prevention of thromboses associated with percutaneous coronary intervention [3]. The aim of my project was to investigate potential cardiotoxic side effects of the drug using TOF-SIMS, which permits the detection of changes in levels of molecular species at the single cell level. Human coronary artery endothelial cells (HCAEC) treated with PZ-128 were analysed using TOF-SIMS and Principle Component Analysis (PCA) was conducted on the resulting data sets.

It was found that chloride (assigned to peak 34.97u) levels increase significantly in HCAECs following treatment with PZ-128, as did cholesterol (assigned to peaks 385.36u, 771.64u). Arachidonic acid (assigned to peak 302.85u) levels also increased following treatment. Given the role of arachidonic acid in feeding into the cyclooxygenase and lipoxygenase pathways – and hence in the formation of prostaglandins, thromboxanes and leukotrienes-, this suggests that PZ-128 could be linked to inflammation, however additional studies would be required to investigate this further. Scanning electron microscopy performed on the samples following analysis by TOF-SIMS verified that the cells remained intact, and that damage to the cells did not account

for the observed differences between PZ-128-treated and untreated cells.

During my studentship I was given ample opportunity to gain experience in other techniques and across other laboratories including that of Prof. Gail McConnell at the University of Strathclyde. Mice organs were cleared with BABB reagent in an effort to optimise a clearing protocol in preparation for imaging using the Mesolens microscope [4], which would enable 3D imaging of the heart with subcellular resolution, complementing the cellular information obtained by TOF-SIMS. In addition, Western blotting was carried out to explore cell signalling events in cell lines.

As shown in figure 1, a method devised by adapting and combining existing protocols proved effective in clearing the heart and other organs [5,6]. The kidney was cleared and imaged first to determine the effectiveness of the clearing and staining procedure. The kidney images acquired using the Mesolens (figure 2) after staining with 100μM propidium iodide (PI) showed an intense fluorescent signal near the surface of the organ and much less in the centre. Hence, 50μM PI and a longer incubation time of 2 days was used to stain subsequent organs, in the hope that the dye would penetrate deeper into the organ and give a less intense signal at the organ surface. It was also noted that BABB, which has been reported to quench fluorescent signals [7], did not interfere with the fluorescent PI stain such that it would impede imaging with the Mesolens.

In other experiments, I had the opportunity to explore signalling cascades downstream of purinergic receptor (P2Y) activation in response to adenosine diphosphate (ADP). HMC3 cells (human microglial cells) were used for Western blotting and P2Y12 receptor signalling was explored in relation to it's Gαi-coupling to cAMP-related pathways. These pathways are also important in platelet function. The P2Y12 receptor is a target for gold standard antiplatelet drugs, so these experiments allowed me to build on the knowledge of anti-platelets that I had obtained from the PZ-128 study.

I have thoroughly enjoyed my studentship and would like to sincerely thank both Dr Margaret Cunningham and the BSCB for giving me this opportunity. I have learned so much over the past eight weeks, gaining experience in a variety of techniques including TOF-SIMS, organ clearing, microscopy, western blotting and cell culture. In addition to enhancing my knowledge of cell biology, I have been able to obtain a realistic insight into life as a PhD student and beyond, which has confirmed that my career aspirations do indeed lie in research.

Rebecca Gilchrist

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Analysing centrosome separation dynamics using a chemical genetics approach

I have completed 2 years of my Genetics BSc at the University of Sussex, which I have thoroughly enjoyed. This has sparked my interest in pursuing a career in scientific research. I decided to apply for the BSCB Summer Studentship to gain a feel for the research environment and provide me with experience before undertaking a masters degree and a PhD.

I was lucky enough to accepted into the studentship to work with Dr Thomas Stiff on the Centrosome Project for 8 weeks as part of the Hochegger Lab within the Genome Damage Stability Centre at the University of Sussex. Dr Stiff is currently investigating the balance of cytoskeletal forces that impact on centrosome separation in late-G2 phase. He is specifically looking at the role that nuclear envelope associated dynein, microtubules and actin structures play in this process. This area is important in terms of fundamental research, as the control of centrosome separation is still poorly understood, and it is also of interest biomedically as problems in separating centrosomes can lead to difficulties in mitosis, aneuploidy, cancer and cell death. Moreover, inhibitors of a key protein in this pathway, Eg5, are being trialled as anti-cancer drugs.

A major aim of my project was to quantify the speed of centrosome movement during separation in late G2 phase based on data from live cell imaging of synchronised U2OS cells. Imaging this process at high temporal and spatial resolution is difficult, because it lasts only 15-20 minutes and occurs once per cell cycle approximately every 20 hours. We used a mutant form of Cdk1 that can be inhibited by bulky ATP analogies to arrest cells in G2 phase, just before entry to mitosis. Additional inhibition of Eg5, the motor that drives separation, prevents centrosome separation under these conditions. Removal of the Eg5 inhibitor results in rapid initiation of the separation process that can be followed under the microscope. Using ImageJ, I also measured the centroid of the nucleus and cell, to allow comparison of the position of centrosomes relative to both centroids. The results of this analysis suggest that centrosome separation proceeds at a speed of approximately of approximately 0.1μm/min, and this is approximately 2.5 times faster, if Cdk1 is fully activated.

My analysis also suggests that the separation process is spatially coordinated with the nucleus rather than the cell centre. Dr. Stiff is currently testing a hypothesis that the actin cytoskeletal plays a pivotal role in the spatial coordination of centrosome separation.

I gained invaluable experience from this studentship, I have improved my laboratory skills and have learnt additional techniques that I have not been exposed to, yet which help me in my dissertation next semester and aid me in post-graduate study. Moreover, I have seen that research extends beyond the lab, with data analysis being just as important. Experiencing the research environment first-hand has led to me seriously considering a PhD and a career as a research scientist. I would like to thank Dr Helfrid Hochegger, Dr Tom Stiff, everyone at the Hochegger Lab and the BSCB for giving me the opportunity to undertake this project and for the support I've received throughout.

Harry Pink BSCB summer studentship 2018, Hochegger Lab, University of Sussex

An investigation into the coregulation of glycolysis at the level of mRNA localisation by various signalling pathways

I am about to start my third year at The University of Sheffield studying Biochemistry and Genetics. I wanted to gain experience working in a research laboratory to learn skills that would be invaluable in the next year of my degree, which will involve a research project. This placement has also assisted decisions about my future career as I have gained a greater understanding about how academia works and an insight into what working in a biological science laboratory is like. I was able to work with Prof Mark Ashe and the rest of the Ashe Lab to research mRNA localisation in yeast in my project.

Under stress, such as glucose starvation, stress granules and P-bodies form in yeast. Evidence suggests that these have roles in mRNA decay and storage. Previous work has found that glycolytic mRNAs are co-ordinately translated in granules and when exposed to stress, these granules coalesce to become P bodies. The aim of this project was to assess the organisation of these glycolytic mRNAs in mutants of the cAMP-dependent protein kinase (PKA) pathway.

The system used to visualise the localisation of these mRNAs involves a cleavage deficient Cas9 enzyme (dCas9) fused to GFP and a single-guide RNA (sgRNAs) that guides the dCas9-GFP to specific mRNA sequences.

First, sgRNAs for the dCas9 system were constructed by Gibson assembly. Primers were designed so that the sgRNA scaffold and the sgRNA sequence specific to the mRNAs of interest would be integrated into the linearised plasmid (figure 1). The sgRNAs generated were ARO3, SUP35, PDC1, PDC5 and FBA1.

The dCas9 plasmid was transformed into yeast, resulting in expression of the dCas9-GFP fusion in the nucleus. The constructed sgRNAs and NIP1 sgRNA were transformed into yeast containing the dCas9 plasmid and the localisation of mRNAs was visualised (figure 3).

NIP1 was also transformed into the YMK201 strain, a low PKA mutant containing a tpk2 wimp mutation and into the corresponding wildtype strain YMK199 (figure 4).

The sgRNAs guide the dCas9-GFP to the mRNA sequences of interest, these were visualised using fluorescence microscopy.

Due to time constraints, the microscopy data was not able to be quantified. However, from the images it can be concluded that all of the sgRNAs show localisation to granules apart from PDC1. The right panel of the YMK201 strain shows high dCas9 expression and similar pattern of localisation to the wildtype, whereas the middle panel shows a different pattern of localisation to the wildtype. This data is interesting as it shows that mutations in the PKA pathway can cause different patterns of glycolytic mRNA localisation. This could have implications on future work investigating the coregulation of glycolysis by the PKA pathway along with various other pathways.

I would like to express my gratitude to the Ashe Lab and BSCB for supporting and enabling me to carry out this project. It has been a fascinating and rewarding experience that has helped me enormously.

Katie Sharrocks

The *in vitro* purification of a stubborn protein

Two years through a Natural Sciences degree at University College London (UCL), I can understand the seamless ways in which areas of science overlap with one another. When applying to Prof Frances Brodsky's esteemed lab at UCL, I did not realise that my summer would be an extension of the interdisciplinary work so central to my academic life thus far. Indeed, I have already briefly experienced labs in cell & molecular biology, and organic & inorganic chemistry but this would be my first major endeavour in a laboratory setting.

Principally, the objective of my project was to develop a strategy for purifying an isoform of clathrin, CHC22. Clathrin is a coat protein implicated in shaping rounded vesicles - the isoform CHC17 is a heavy chain found in all cells but the CHC22 heavy chain is only found in muscle and fat cells. So far, CHC22 has been purified in vivo within HeLa cell systems, but successfully purifying CHC22 in vitro would explicate its structure, both as triskelion molecules and in cages, and elucidate more about its function - particularly pertinent given CHC22 is suspected to be an important regulator in glucose transporter trafficking.

Initially, I was introduced to the methods I would need to use by Dr Lisa Redlingshoefer. These included insect cell culture, expression and purification of the recombinant protein from the insect cell. While culturing cell lines, I quickly realised how vital sterility is and became extremely vigilant when working with any cells or viruses. We used a baculovirus system, that held the gene for the recombinant protein, to transfect the insect cell. After finding that 72 hour incubation of the virus yielded the highest amounts of CHC22, we began with purification.

Employing clathrin's intrinsic ability to form cages and in turn, disassemble into its constituent triskelia, we used a series of buffers and centrifugations to isolate the protein. In theory this seemed like a sensible proposal yet in practice, it was naturally not so simple. Firstly not enough clathrin was extracted from the cell during lysis, then there were problems efficiently pelleting assembled cages. Turning our attention to Nickel-affinity chromatography, we isolated recombinant CHC22 tagged with a C-terminal motif of histidine residues. This method was more fruitful as it eliminated steps in the protocol and left only the lysis and column chromatography stages.

After concentrating the product we remarkably were left with pure CHC22 and confirmed this using Coomassie staining and Western blotting. Furthermore, using the pure CHC22, we were able to demonstrate its physical association to an adaptor protein, GGA2, for the first time. Going into this project I was simply oblivious to the sheer level of problem-solving required on a day-to-day basis of a scientist. Now I have greater confidence in the lab and a fuller understanding of how interdisciplinary science actually is. I would like to thank Prof Brodsky for hosting me in her lab, the BSCB for providing their support and Dr Redlingshoefer for supervising this project and truly being the brains behind it all.

Nikhil Harsiani

The epidemiology of uropathogens in renal transplant recipients

I am an MSci Applied Medical Sciences undergraduate student at University College London (UCL) entering my fourth and MSci year studies. Over the summer, I was very fortunate to have been awarded an 8-week funded placement by the British Society of Cell Biology.

Scientific or medical research is not considered a popular profession by people from my ethnic background. However, I believe that there is no better way to improve people's quality of life. As a person who has always had a strong curiosity and desire to learn through the process of performing experiments and investigation, I decided to invest my skills and knowledge into a career in medical research.

My interests in this field are also personal: I was once a chronic Urinary Tract Infection (UTI) sufferer. When I was previously a regular visitor to my doctor for this condition, I felt a lack of support, as if my condition wasn't deemed to be that serious. The healthcare professionals I encountered had the firm belief that antibiotics successfully treat all bacterial infections, including UTI, but these same people failed to provide me with an answer when I asked "So why am I suffering from recurrent infections?" It was this mystery that drove me to want to undertake research in this area.

So I spent two months with Dr. Jennifer Rohn, Dr. Sanchutha Sathiananthamoorthy and rest of the Chronic UTI Research Group at UCL, trying to answer this question for myself. This team is interested in understanding why UTIs frequently recur, and it uses a combination of cell biology, microbiology, genomics, immunology and high-resolution imaging in the process. My project was to investigate the understudied epidemiology of uropathogens in renal transplant recipients. This work is crucial as kidney transplant patients are highly susceptible to recurrent UTI, and it's thought that UTI can ultimately lead to a higher chance of transplant rejection. Little is known about the host/pathogen interaction in this complicated group of patients, and we were interested in identifying any differences compared with individuals suffering from traditional uncomplicated UTI and healthy controls.

The midstream urine (MSU) culture is a reference standard procedure performed in hospital microbiology laboratories used to detect bacteria in urine at levels that are conventionally considered to indicate infection, but it has known limitations. My colleagues in the lab had published an alternative approach which involves concentrating the uroepithelial cells shed in urine by centrifugation. This method is intended to optimise the isolation of bacteria because they are highly evolved to bind, invade and colonise human epithelial cells, a behaviour that is thought to contribute to recurrent UTI. This improved protocol showed significant improvements over the standard MSU culture. For example, from one of the patient's urine samples, I found no colonies on plates prepared in the traditional manner, but many colonies revealed after culturing the cell sediment. This work underscores the hypothesis that the standard MSU protocol may not be suitable for diagnosing UTI in kidney transplant patients. Using advanced microscopy techniques, I have also made a start in studying the molecular host/pathogen interactions in bladder epithelial cells; in parallel, I have recovered DNA from these cells to facilitate a metagenomic approach to understanding the complex ecology of infection in these patients.

This has been my first summer placement working away from my home country, yet the best experience so far. This opportunity allowed me only not to learn different laboratory techniques such as immunofluorescence staining and a new DNA quantification method (Qubit), but also to improve my soft skills such as time management and problem-solving. Good communication also helped me to develop a stronger relationship with my colleagues as well as with clinical staff in the renal unit to create a healthy and relaxing working atmosphere. I would like to thank the BSCB and Dr. Rohn for making this placement possible, in fact, I will now continue the research as my MSci project in the coming year, and hope to be able to contribute meaningful result to this neglected but important area of research in the future.

Flora Cheng

Investigating the effect of zona occludens-1 in the stability of tight junctions in prostate acini and how this can contribute to prostate cancer progression

I am currently studying Biomedical sciences at St Georges University and having successfully finished second year I was eager to broaden my understanding of the "research world" and gain a greater understanding of what a typical day in the lab would consist of as I was unsure if I wanted to undertake a career in research. When the opportunity came, I seized the chance to work alongside Dr Valderrama here at St Georges and was lucky enough to be funded for an 8-week placement by the BSCB. Also, not having had the opportunity to work in a real working laboratory before, the studentship would allow me to build on essential lab techniques that I had learnt in my university course thus far.

The aim of the project was to investigate the effect of radixin (RDX) – an adaptor protein that links plasma membrane receptors to the actin cytoskeleton – on the localisation of zona occludens-1 (ZO-1), a protein found in tight junctions. Preliminary data in our laboratory show that radixin is able to affect the stability of adherens junctions by relocalising E-cadherin away from the plasma membrane. This process seems to also involve atypical protein kinase C (aPKC). The effect of E-cadherin relocalisation is a loss of cell-cell contact and the loss of epithelial organisation of prostate acini. Since aPKC is known to regulate tight junctions , my project aimed to investigate whether radixin would also influence tight junctions stability by controlling ZO-1 localization. In order to achieve this aim, the objective was to grow cell lines of varying degrees of malignancy and overexpress different forms of radixin to see if there would be any effect on ZO-1 localisation.

I was able to grow three cell lines: RWPE-1, WPE1-NA22 and WPE1-NB26. RWPE-1 are normal non-cancerous prostate epithelial cells, NA22 are mildly tumorigenic and NB26 are highly tumorigenic. During this time, I was introduced to cell culture techniques which I previously had no experience of, and at first I was very nervous about killing the cells. But with practise - which this placement allowed me to do - I was able to grow confidence with cell culture and only made me feel more ready to start my final year

research project. I also learnt other techniques such as 3D cell culturing, Western blotting, DNA extraction and Immunofluorescence. Unfortunately, due to difficulties with the antibody against ZO-1 not working well, it was difficult to even see ZO-1 under widefield or confocal microscopy.

Something I learnt from this placement however, was to not always expect positive results. I learnt to appreciate how much hard work it is and the many hours that people spend in the lab running experiments and having to repeat experiments when they do not work out as hoped for. It was amazing to see how persistent everyone is – it is definitely inspiring and I feel privileged and extremely grateful to have been given this opportunity by the BSCB and Dr Valderrama for allowing me to work in his lab.

Juma Akhtar

When base editors meet human embryonic stem cells

I began my MSci degree in Genetics at University College London 3 years ago and I have been always pleased with the opportunities I have received to gain theoretical insights into areas that always sparked my curiosity. However, as an undergraduate student I felt that my exposure to practical work remained limited and given my ambition to pursue a career in research, I knew it was important to develop the appropriate skills in benchwork. As a result, undergoing internships has always been a priority for me, and this summer, I had the wonderful opportunity to spend 9 weeks in Dr Kathy Niakan's "Human Embryo and Stem Cell" laboratory at the Francis Crick Institute and I was particularly fortunate to have been supported by the funding of BSCB.

Under the direct guidance of Dr Afshan McCarthy, and thanks to Dr Niakan's encouragement, I was given the freedom to develop a project using the new CRISPR-mediated base editing technology, which I am particularly interested in. Allowing single base pair changes without double stranded breaks, this technology may be a powerful alternative to traditional CRISPR/Cas9, which has the limitation of being linked to uncontrolled repair through non-homologous or microhomology-mediated end joining. I sought to test this "cleaner" gene editing technique in human embryonic stem cells (hESCs) for the first time. As a proof of principle, my aim was to introduce an early stop codon in POU5F1, with the hope of creating a successful knockout of this gene, whose role in early human embryogenesis was recently uncovered in the lab. I therefore went through the entire experimental process, from the initial design to selecting candidate sgRNAs and appropriate plasmids, followed by cloning steps and finally, nucleofecting and selecting both hES and HEK293T cells.

9 weeks was certainly not enough to complete my project, and like anyone working on their own project in a lab for the first time, I faced many challenges and setbacks. I am currently waiting on the deep sequencing results, that will tell me the efficiency of the base editing in both cell types. If encouraging, the results could open an avenue for further optimisation of base editing in hESCs, a tool that may become a new standard for both correction of disease causing mutations and induction of gene knockouts.

On my way to university, I used to pass in front of the Crick Institute, wondering whether one day, I would have the opportunity to work in such a prestigious institution, a cathedral for science, alongside the world's leading scientists. This summer, I gained more research experience than ever before. Not only have I learnt about stem cell culture and a wide range of molecular biology techniques, but I have also been exposed to a unique critical and rigorous way of approaching science in the lab, which is invaluable and will help me in my journey to becoming a scientist. I now feel more prepared to undergo my master's research project and I am more motivated than ever to pursue a career in research.

Jérémie Subrini

Using *Drosophila* to investigate gender-specific differences in the essential, conserved invadolysin metalloprotease

A childhood fascination towards the life forms around me sparked the passion of science in me. The more I tried to reach out into the field of science, the more it pulled me in. The processes like cellular signaling, neuronal transmission and homeostasis polished my passion into a dream. The quest to learn more about science brought me to IISER-TVM, one of the premier research institutes in my country, where I am presently at the final year of Integrated MSc. My first sam- pling of undergraduate research was when I joined Dr. Jishy Varghese's lab in 2016. I was intro- duced to Drosophila melanogaster, which still remains as my model organism of interest.

This summer I was fortunate enough to work in Prof. Margarete Heck's laboratoryat the Univer- sity of Edinburgh. I consider it as my utmost privilege to have gotten the opportunity to carry forth independent research in a lab of such calibre. The experience left me enriched with critical thinking skills, developed a stronger sense of confidence and further fueled my drive for science.

Invadolysin is a conserved metalloprotease discovered in the Heck lab, the only discovered pro- tease to be localized on lipid droplets. It has been demonstrated to have a role in cell migration, angiogenesis, adipogenesis and insulin signaling. Previous reports from the lab showed that dif- ferent forms of invadolysin are presentin males and females, similar to the forms expressed in their gonads. My project was to investigate the sex specific expression of invadolysin in Droso- phila hemolymph and gonads, and how mating and ageing affect expression. I was able to find that the gender specific expression is seen as early as from 2 hours of eclosion, and was depen- dent only on age, but not mating. Using the UAS-GAL4system to drive expression at particular times or tissues, I expressed protease-dead and lipase-dead forms of invadolysin (also invadoly- sin shRNA constructs)in a tissue specific manner. While these experiments were designed to address the roles of the catalytic motifs of invadolysin in lipid and glycogen metabolism, addi- tional experimental repeats would be required for the results to be considered statistically signifi- cant.

I owe my sincere gratitude to Prof. Margarete Heck for her truly inspiring guidance which was essential for completing this project. I am thankful to Ms. Linda Feng for mentoring and guiding me throughout the project. I also thank the British Society of Cell Biology for awarding me the studentship.

Anantha Krishnan S S

Application for Honor Fell / Company of Biologists Travel Award

Please complete, print out and send to Julie Welburn at the address below together with supporting information

Full name and work/lab address:

Email:

Age: BSCB Memb. No:

I have been a member for years

Years of previous Honor Fell /COBTravel Awards:

Degree(s) (dates):

Present Position:

Meeting for which application is made:
title/place/date:

Expenses claimed:

Travel:

Accommodation:

Registration:

Have you submitted any other applications for financial support?
YES/NO (delete as applicable)
If YES, please give details including, source, amounts and whether these monies are known to be forthcoming. Note we expect you to not claim the expenses twice from different sources.

Bank details
Sortcode:
Account number:
Bank:

Supporting statement by Lab Head:
This applicant requires these funds and is worthy of support. I recognise that in the event of non-attendance at the meeting, the applicant must return the monies to the BSCB and I accept the responsibility to reimburse BSCB if the applicant does not return the funds. Also, the student is not receiving the same reimbursement from another source.

Signature:

Name:

Applicant's Signature:

Name:

Have you included all the necessary information/documentation in support of your application?
All Applications must contain:
- A copy of the abstract being presented
- A copy of the completed meeting registration form

Send to: Dr Julie Welburn, Wellcome Trust Centre for Cell Biology
University of Edinburgh, Mayfield Road, Edinburgh EH9 3BF

- If proof of payment for ALL costs claimed is available at the time of application, successful applicants will be awarded a grant in advance of the meeting
- If proof of payment for ALL costs is not available at the time of application, successful applicants will be awarded a provisional grant and funds will be sent when BSCB have received the receipts.
- Incomplete applications will not be considered.

The British Society for Cell Biology

Statement of Financial Activities for the year to 31 December 2017

	Unrestricted Funds	Restricted Funds	Total 2016	Unrestricted Funds	Restricted Funds	Total 2015
	£	£	£	£	£	£
Income from:						
Grants	35,000	62,500	97,500	35,000	60,000	95,000
Investments	8,861	–	8,861	1,322	–	1,322
Charitable activities						
Meetings	–	–	—	–	–	–
Subscriptions	32,802	–	32,802	33,439	–	33,439
Total income	**76,663**	**62,500**	**139,163**	**69,761**	**60,000**	**129,761**
Expenditure on:						
Charitable activities						
Grants payable:						
CoB/Honor Fell travel awards	–	57,352	57,352	–	45,083	45,083
Other grants	900	3,000	3,900	1,165	500	1,665
Studentships	16,012	–	16,012	15,140	–	15,140
Costs of meetings	18,859	–	18,859	19,356	–	19,356
Website expenses	465	–	465	798	–	798
Newsletter costs	3,675	–	3,675	2,768	–	2,768
Membership fulfilment services	22,267	–	22,267	12,108	–	12,108
Executive Committee expenses	2,345	–	2,345	1,235	–	1,235
Examiner's remuneration	2,524	–	2,524	2,447	–	2,447
Subscriptions	1,279	–	1,279	2,221	–	2,221
Insurance	1,095	–	1,095	1,080	–	1,080
Total expenditure	**70,477**	**60,352**	**130,829**	**58,318**	**45,583**	**103,901**
Net gains/(losses) on foreign exchange rates	–	–	–	–	–	–
Net (expenditure)lincome	**6,186**	**2,148**	**8,334**	**11,443**	**14,417**	**25,860**
Transfer between funds	–	–	–	–	–	–
Net movement in funds	**6,186**	**2,148**	**8,334**	**11,443**	**14,417**	**25,860**
Funds brought forward at 1 January 2017	198,197	28,924	227,121	186,754	14,507	201,261
Funds carried forward at 31 December 2017	**204,383**	**31,072**	**235,455**	**198,197**	**28,924**	**227,121**

BSCB Committee Members 2019

Committee
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 eight office-holders (President, Secretary, Treasurer, Meetings Secretary, Membership Secretary, Newsletter Editor and Web Co-ordinator) and up to 12 other ordinary members, including one PhD student representative and one Postdoc representative.

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 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 .

The BSCB has charitable status (registered charity no. 265816) and has a constitution. The BSCB AGM is held every year at the Spring Meeting and all BSCB members are invited to attend.

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BSCB Ambassadors

Ambassadors are BSCB members who represent the society at their institution. Their role is to promote the society to the UK Cell Biology community and to provide a route by which members can communicate with the BSCB Committee. This year Ann Wheeler and Andrew Carter updated our list of Ambassadors and recruited some new ones for institutions that were not previously represented. Andrew will keep in contact with the Ambassadors in his role as Membership secretary. We would like to thank the ambassadors who have stepped down for their several years of service to the society.

We also extend a warm welcome to our new Ambassadors. We will look forwards to hearing more about what the BSCB has been doing locally.

If you have any questions about the society or ideas about what the BSCB can do for UK Cell Biology then please contact your Ambassador. If your university does not have an Ambassador and you would like to volunteer please also write to our membership secretary Andrew Carter. (cartera@mrc-lmb.cam.ac.uk).

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