WINTER 2006

BSCB Newsletter

BRITISH SOCIETY FOR CELL BIOLOGY

British Society for Cell Biology

B S C B

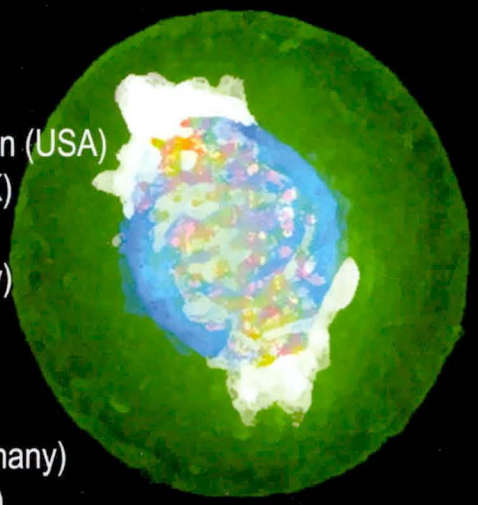
Spring 2007 Meeting

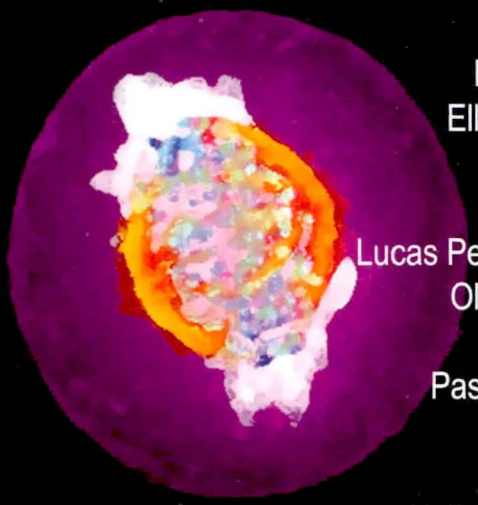
a joint meeting of the Genetics Society and the British Societies for Cell and Developmental Biology

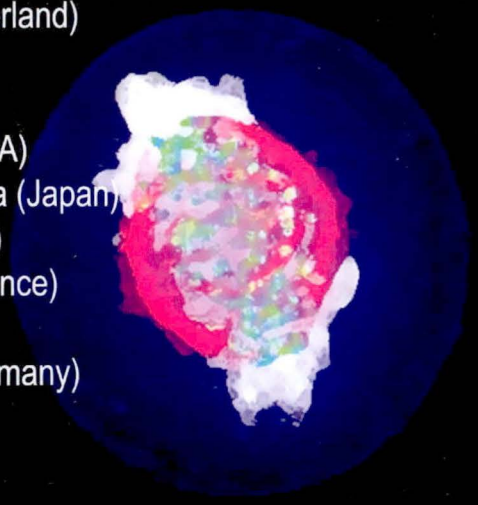
the genetics society | BSDB | British Society for Cell Biology | B S C B

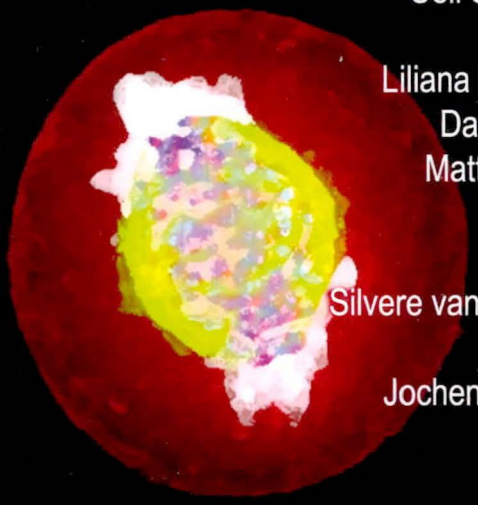
Heriot-Watt University, Edinburgh, 29th March - 1st April 2007

Michael Bate (UK)
Herwig Baier (USA)
Zhijian "James" Chen (USA)
Stephen Davies (UK)
Caroline Dean (UK)
Ivan Dikic (Germany)
Liam Dolan (UK)
Bill Earnshaw (UK)
Bruce Edgar (USA)
Jan Ellenberg (Germany)
Bob Goldman (USA)
Michael Hall (Switzerland)
Ron Hay (UK)
Doug Higgs (UK)
Laura Johnston (USA)
Ryoichiro Kageyama (Japan)
Angus Lamond (UK)
Thomas Lecuit (France)
Paul Lehner (UK)
Matthias Mann (Germany)
Jane Mellor (UK)









Barbara Meyer (USA)
Elliot Meyerowitz (USA)
Andrew Millar (UK)
Steve Oliver (UK)
Lucas Pelkmans (Switzerland)
Olivier Pourquié (USA)
Tomo Tanaka (UK)
Pascal Therond (France)
Mike Tyers (Canada)
Bill Schafer (USA)
Ueli Schibler (Switzerland)
Pam Silver (USA)
Liliana Solnica-Krezel (USA)
Daniel St Johnston (UK)
Matthias Uhlen (Sweden)
Helle Ulrich (UK)
Sylvie Urbé (UK)
Silvere van der Maarel (Holland)
Steve Wilson (UK)
Jochen Wittbrodt (Germany)

Programme includes

Protein Modification, Cell Growth, Ubiquitin, Trafficking and Signalling, Biological Clocks, Nuclear Dynamics, Genetics and Behaviour, Genomes, Chromosomes and Disease, Cell Polarity and Migration, Systems Biology, 'Omics and High throughput Screens: The future?

BSCB Honor Fell travel awards and BSDB travel awards available on application
For further information see www.genetics.org.uk, or www.bsdb.org or www.bscb.org

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BSCB Newsletter

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Editorial

Welcome to the new look newsletter for the BSCB. At the same time as several other cosmetic changes, Giles Newton at the Wellcome Trust and Jag Matharu have reworked the template to provide more visual impact. This of course ties in with the new BSCB logo that is shown on the cover and profiled in the 'News' section. David Archer, our schools liaison officer has spent considerable time and effort organizing this with the help and input of the rest of the committee. The logo is designed to be clear and effective on multiple media.

The final component of this redesign is the launch of the new BSCB website. This has been designed to make all aspects of your interaction with the BSCB easier and more efficient. This includes a new secure online membership application form to join the society, full details of all society meetings, Honor Fell travel awards, archived versions of the newsletters, and the highly popular softCELL eLearning pages.

The newsletter also includes a new section profiling new research institutes with a strong cell biology theme. The first two are from the Universities of Manchester and Edinburgh and it is our intention to profile a range of new infrastructure projects that relate to cell biology over the next few issues.

The news section also includes a call to arms from the society President Clare Isacke about the BSCB Annual General Meeting. This traditionally takes place at the annual Spring meeting and is your chance to directly influence the direction and roles of the society. As Clare emphasizes in her piece, attendance is open to all so we hope to see many of you at the AGM at Heriot-Watt in March 2007.

We are always on the lookout for your own material to be included in the Newsletter. If you have any news items, features, suggestions for cover images, or meeting announcements that you wish to be included then please contact me directly.

The Editor: David Stephens
(david.stephens@bristol.ac.uk)

The cover image shows a Jurkat T cell, transfected with GFP-Rif (a small GTPase of the Rho family involved in the generation of filopodia) and labelled with Alexa-594-phalloidin. The 2 ruffles actually cover and/or explore polystyrene beads which are not shown. The image accompanies the 'Actin 2006' meeting report in this issue and was kindly provided by Stéphanie Pellegrin and Harry Mellor (University of Bristol).

Newsletter editor: David Stephens **Production:** Giles Newton **Website:** www.bscb.org **Printer:** Hobbs

News

Why attend the Annual General Meeting?

The annual general meeting (AGM) of the BSCB is traditionally held during the spring meeting and this year is no exception in that it will take place on 30 March 2006 at Heriot Watt University, Edinburgh. However, in another way I want this year to be the exception that sets the trend for future meetings because I am urging all BSCB members who are at the meeting to attend the AGM.

Why would you want to? Because the AGM is where you as members of the society get a say in how the society is run. Do you think the meetings we run could be improved? If so, the AGM is the place to tell us what you think. Do you have suggestions for who should be on the BSCB committee? Tell us who you would like to nominate. Do you have a view on what a cell biology society should be doing for cell biologists?

We would love to hear your opinion. We can't represent you if you don't tell us what you want.

Finally, should you want to attend but feel that after a long day listening to talks, the lure of the bar is too great. Do not worry as refreshments will be provided. Please come and join us and help make the BSCB a society that truly represents its membership.

Clare Isacke
President, BSCB.

Further honours for past Hooke Medal winners

Three recent winners of the BSCB Hooke Medal have been given further honours recently.

Frank Uhlmann (Cancer Research UK London Research Institute) has been awarded the EMBO Gold Medal. On announcement of the award Frank Gannon, EMBO's Executive Director said that it was "in recognition of a decade of extraordinary work that has revolutionised our understanding of the cell cycle and opened the door to new possibilities in cancer treatment".

Andrea Brand (The Wellcome Trust/Cancer Research UK Gurdon Institute, University of Cambridge), winner of the Hooke Medal in 2002, has been awarded the Royal Society Rosalind Franklin Award for 2006. The award is made for outstanding contributions in any are of science engineering or technology.

The winner is also asked to undertake projects to raise the profile of women in science and Andrea will be organizing two lecture series to be delivered by outstanding women scientists in the field of cell and developmental biology; one to be delivered to school children in the Cambridge area, and the other to undergraduates and postgraduates.

The Royal Society has also honoured the 2003 Hooke Medal winner **Matthew Freeman** (MRC Laboratory of Molecular Biology, Cambridge) with election as a Fellow of the Royal Society. Matthew is also the current Chairman of the British Society for Developmental Biology.

New Wellcome Trust funding for early career scientists

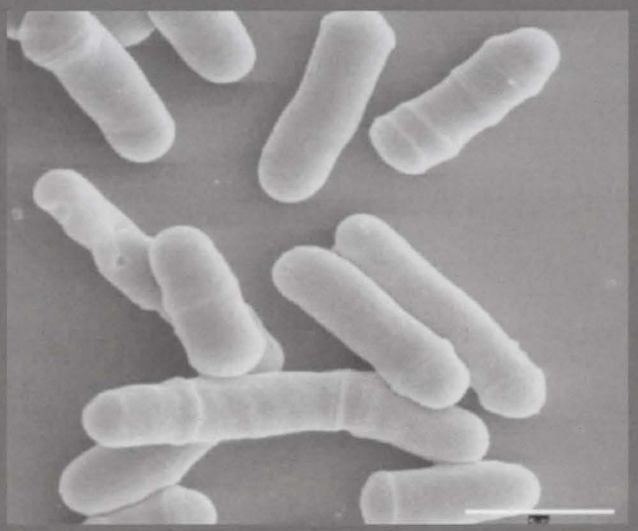
The Wellcome Trust has revised their fellowship provision for early career research scientists. A new scheme, the **Sir Henry Wellcome Postdoctoral Fellowships** provide funding opportunity for the best newly qualified postdoctoral researchers to work in top-class labs either within the UK or overseas. Applicants should be within 12 months of the award of their PhD and are expected to propose a deliver an independent research programme in an important biomedical research area. The first round of applications is now closed but the scheme is ongoing and further rounds of application will be announced on the Wellcome Trust website.

Wellcome has also announced a new scheme of **Flexible Travel Awards**. There are two variations on this theme. Travel Fellowships provide support for up to two years to be spent working on a new area or emerging area of research within the applicants own field. Sabbatical awards have also been launched for internationally competitive researchers to undertake a sabbatical to establish new collaborations or explore new activities that are not supported through other funds.

Further details of all these schemes can be found on the Wellcome Trust website at: www.wellcome.ac.uk

Cover images

Do you have any cell biology images of which you are particularly proud? The newsletter provides a great way to display your work to a wide audience. Any type of contribution within the field of cell biology will be considered. Following the recent redesign of the newsletter, these images are now displayed at a large size providing even greater impact for your work. If you would like a chance for you image to be included on the cover of the newsletter then please email it with a short description (either as TIFF or JPEG in CMYK and at high resolution, e.g. 600 dpi) to David Stephens (david.stephens@bristol.ac.uk).



The BSCB Logo has been refreshed

ICI did it and British Petroleum has done it several times. ICI refreshed their logo by reducing the number of wave peaks from three to two, so smoothing the 'wavy lines' in their roundel logo design. BP (or bp) did it by changing from an upper case font (BP) to an italic one (*BP*). Next in time came non-italic and lower case (bp), with different words added such as 'connect' and 'beyond petroleum'.

Organisations refresh their image and branding mainly for market led reasons so why has our society refreshed its logo?

There are several reasons:

1) There were two versions of the logo in use, one diagrammatic and very clear, and a coloured version which was clear when back lighting was used, as on a computer screen, but less clear in some applications. On poster presentations the coloured version was not eye catching and when copied or printed in black and white the clarity was poor.

2) Neither version of the logo informed the uninitiated who we were. Whilst this might be acceptable if you are ICI or bp, we are not in that league.

3) When the clear bi-polar single colour BSCB logo was devised in 1996 colour printing from personal computers was fairly new. Now that it is commonplace we needed to consider whether the logo should contain more colour but in a clear and reproducible way.

4) The BSCB website is being re-designed and the Newsletter is also being changed, so it seemed an appropriate time to re-visit our logo.

What were the criteria for re-designing the logo?

1) The total logo needed to be clear at all usual enlargements and reductions.

2) The logo had to be clear when printed or photocopied in colour or black/white. It had to be clear when back lit and when printed on opaque material.

European Research Council

The European Research Council is now a reality. Prof. Fotis Kafatos former Director General of the European Molecular Biology Organization has been elected as Chairman of its Scientific Council and the ERC intends to launch its first call for proposals, to be targeted at early stage researchers, early in 2007.

The first call for applications takes the form of early career awards called **Starting Independent Researcher Grants** (StG). These are designed to support "excellent researchers at the stage of establishing their first independent research team or to strengthen teams that have been recently created". Around 200 awards will be made each year. Calls for applications from more advanced scientists following later on. The ERC will be funded as part of the 7th EU Framework Programme with a proposed annual budget of 1.5 billion euros.

ERC website:
ec.europa.eu/erc/index_en.cfm
StG strategy note:
ec.europa.eu/erc/pdf/sir-grant-strategy_en.pdf

In brief...

MEMBER BENEFITS

Did you know that your BSCB membership includes discounted journal subscriptions (including *Journal of Cell Science*, Traffic), and discounts on Wiley and Oxford University Press books? One-off discounts are also available including for the newly released textbook 'Cells' by Benjamin Lewin *et al*. The book includes a chapter on 'Intermediate filaments' by former BSCB Secretary Professor Birgit Lane. 'Cells' will retail in the UK and Europe at about £38.99 but BSCB members can obtain it at the special price of £33.00 inc p&p. To take advantage of this offer please contact Christine Gribble at cgribble@jbpub.com or phone 01842 878586. These discounts more than compensate for society membership fees so do encourage your friends and colleagues to join. Students also benefit from reduced membership fees so do encourage any new postgraduate students joining you in the autumn to join the BSCB. Further details of all member benefits and the new secure online form for membership applications can be found at www.bscb.org

FUNDING FOR LOCAL MEETINGS

The Society is prepared to provide limited financial support for meetings organized by any local interest group relevant to cell biology. Request for funds should be sent to the Treasurer, Mark Marsh, accompanied where possible by a report of a previous meeting. If a meeting receives support, a report from that meeting will be required for publication in the Newsletter.

BSCB MEMBERSHIP DATABASE

The website contains the facility to search for members of the Society. However, under the data protection Act, we can include your details only if you specifically grant us permission to do so. If you wish to be included and are not, please contact Margaret Clements (bscb@biologists.com).

ARCHIVED NEWSLETTERS ONLINE

Previous versions of the BSCB Newsletter are now available on the BSCB website; so, if you lose your copy then you will still have access to all of the content. Further changes to the website will be taking place shortly as part of its re-launch. www.bscb.org

BSCB Ambassadors

The Society has representatives at each of the institutions listed below. The Ambassadors have agreed to promote Society activities and membership within their University or Institute. They disseminate advertisements concerning future BSCB meetings, promote the advantages of membership, particularly to new PhD students, and are available to sign application forms and answer any BSCB-related questions. If your institute is not represented and you would be willing to become and ambassador, please contact Jonathan Pines.

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	Patricia Salinas	p.salinas@ucl.ac.uk
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York	Dawn Coverley	dc17@york.ac.uk

The Queen's Medical Research Institute, Edinburgh

A new face for biomedical research at the University of Edinburgh.

The idea of the Queen's Medical Research Institute originated in the mid 1990s when Lothian Health made the decision to relocate the Royal Infirmary of Edinburgh to Little France. The University developed plans to replicate, at Little France, the symbiotic relationship of research, teaching and clinical practice which had evolved over a century at the old Royal Infirmary/Medical School site.

Development of the Chancellor's building was the first stage of these plans. However, the Research Institute vision was nurtured and developed until the advent of the Government and Wellcome Trust Joint Infrastructure and Science Research Infrastructure funds (SRIF) offered the opportunity to attract key core funding. That the Institute project attracted £11.2m from the Wellcome SRIF in 2001, matched by £11.2m from the University's SRIF allocation from the Scottish Higher Education Funding Council, is testimony to the commitment and confidence of those who carried the vision and to the quality of the research to be housed in the Institute. This quality was recognised in the 5* award for 'Hospital Clinical Subjects' in the 2001 Research Assessment Exercise. Indeed, the University of Edinburgh is one of the leading Universities for medical research in the UK and Europe. It is with great pride that the University occupied, in 2005, 'The Queen's Medical Research Institute'.

The £49m Queen's Medical Research Institute (QMRI) will accommodate over 600 clinical and basic scientists working in three interdisciplinary centres and investigating cardiovascular, reproductive and inflammatory disorders of the lungs, liver and kidneys. The MRC/University of Edinburgh Centre for Inflammation Research, the Centre for Cardiovascular Science and the Centre for Reproductive Biology (incorporating the MRC Human Reproductive Sciences Unit) are now together under one roof encouraging and facilitating collaboration between disciplines. Through this interdisciplinary approach we hope to accelerate the research process leading to swifter delivery of new means to prevent, diagnose, treat and monitor disease.

The QMRI will have collaborations with a wide range of scientific interests throughout the University, including Informatics, Biological services and Veterinary medicine. For example, specialists in Informatics are able to develop complex computing tools and models which help us to extract meaningful information from new technologies such as Magnetic Resonance Imaging (MRI), Positron Emission Tomography (PET), and infrared scanning.

Above: The Queen's Medical Research Institute at night.

Such collaborations will fuel the ongoing development of integrated human imaging facilities within the QMRI. The imaging facility, which has very recently attracted a major award (c.£5m) from the UK and Ireland Clinical Research Consortium, specifically for the study of humans, will significantly accelerate our research, and ultimately improve the quality and nature of care for patients.

Fund Raising

Fund-raising for the research institute began in 2000, headed until 2002 by Vice-Principal of the University, Sir David Carter, and Professor Colin Bird, Dean of the Medical School, working closely with Development and Alumni staff, and a wider team from within the University. Following their retirements, Professors Carter and Bird have remained actively involved in fund-raising efforts which are now led by Professor Chris Haslett, inaugural director of the QMRI, Professor John Savill, Vice Principal and Head of the College of Medicine & Veterinary Medicine and Vice-Principal Young Dawkins III.

Major gifts were firstly sought to ensure that this key project was viable. The first two commitments came from the allocation of £11.2m from the University's Science Research Infrastructure Fund resources, which matched an award of £11.2m by the Wellcome Trust, giving the project a healthy start of £22.4m. Further major gifts were pledged by donors including the British Heart Foundation, the Medical Research Council, the Wolfson Foundation, the Rayne Foundation, the Gannochy Trust, the Garfield Weston Foundation and the Robertson Trust. The European Regional Development Fund awarded the project £5.6m, acknowledging that significant contribution the research institute will make to the economy of the City of Edinburgh and the south-east of Scotland.

A number of organisations and individuals have made generous personal donations which are acknowledged on a donor board in the foyer of the Institute. Contributions by Mrs Gina Fyffe and the Mary Kinross Charitable Trust have given rise to meeting and break facilities, while contributions from Lord and Lady Trotman, the Robertson Trust, Gannochy Trust, Rayne Foundation and others have given rise to state of the art laboratory facilities.

In March 2004, the American Kresge foundation issued a $1,000,000 'challenge' award to the research institute project. Our subsequent success in unlocking this challenge commitment, the first of this size to be awarded to a UK University, was dependent on the project organisers being able to demonstrate to Kresge that the private sector, alumni and staff of the University supported the project by raising the final £2.64m required to complete the project. The call to action of the challenge was answered by many individuals and organisations that support the research institute project, including medical alumni, current staff of the College of Medicine and Veterinary Medicine and other well-wishers.

THE RESEARCH

Cells – The building blocks of tissues in health and disease

The QMRI has a unique and exciting focus – the cell biology of common diseases. Cells are the building blocks of tissues. Disease commonly arises from abnormal cell function and the disruption of 'conversations' between cells in a tissue, the 'meat' of organs such as the lung or kidney. Edinburgh researchers have discovered remarkable new insights into the behaviour of cardiovascular, reproductive and inflammatory cells in health and disease. Intriguingly, there are abnormalities of cell function that are common to apparently different disease processes. For example, very similar changes in white blood cell behaviour are seen in coronary heart disease, asthma and painful disorders of menstruation in women. So, by bringing together researchers in these fields, we are accelerating progress, improving the prevention, diagnosis and treatment of common diseases that affect millions of people in the UK. The Queen's Medical Research Institute, therefore, makes a very special and distinct contribution towards improving health.

Key research into three important areas of health is undertaken at the Research Institute:

Cardiovascular Science

Researchers in the Centre for Cardiovascular Science are addressing disorders of major importance, including coronary artery disease, the metabolic syndrome and hypertension. There is a major commitment to translational research that ranges 'from bench to bedside and back again'.

For example, researchers can now understand better the potential risk of a heart attack by assessing the atheroma 'plaques' which build up in coronary arteries. Among the plaques, a few become unstable and can cause thrombosis and heart attack. Edinburgh researchers have identified techniques to study these plaques. They are working on approaches which can demonstrate how such hardening of the arteries can develop over a period of time, which plaques are vulnerable, and what can be done to prevent plaques becoming unstable and causing blockage of coronary arteries.

Inflammation Research

The Inflammatory response, in which white blood cells are recruited to sites of tissue injury, evolved to protect us from infection and promote healing of damaged tissues. Unfortunately, inappropriately triggered or abnormally persistent inflammation causes a wide range of diseases such as asthma, lung fibrosis, liver cirrhosis and kidney failure. Researchers in the MRC/University of Edinburgh Centre for Inflammation Research are investigating the cellular mechanisms that underlie evolution and resolution of inflammation with the ultimate aim of developing new treatments.

For example, Edinburgh researchers have discovered that a natural type of cell death (apoptosis) is crucial for the normal resolution of inflammation. Examples of persistent inflammation including asthma, rheumatoid arthritis and atheromatous plaques in blood vessels, are thought to occur in part as a result of defects in apoptotic cell clearance. Workers in the Centre have recently identified a new class of drugs capable of specifically driving resolution of inflammation through induction of white blood cell clearance from inflamed tissues by apoptosis, with exciting potential for therapeutic benefit.

Reproductive Biology

Abnormalities in the human reproductive systems result in fetal and maternal mortality and in considerable illness and distress due to menstrual disorders, infertility, infections and cancers of the reproductive organs. Edinburgh is a leading international centre for research in the field, thanks to the world-renowned Centre for Reproductive Biology, which represents a longstanding collaboration between the University and the MRC Human Reproductive Sciences Unit. The Centre hosts the Jennifer Brown laboratory, which focuses on issues of paramount importance for mothers and babies, such as the causes of intrauterine growth retardation and complications of pre-eclampsia, a syndrome in which pregnancy induces high blood pressure and blood vessel injury in affected mothers.

Weblinks

The Queen's Medical Research Institute
47 Little France Crescent
Edinburgh, EH16 4TJ
http://www.ed.ac.uk/explore/places/buildings/mri.html

The University of Edinburgh / MRC Centre for Inflammation Research (CIR)
http://www.cir.med.ed.ac.uk/

The University of Edinburgh Centre for Cardiovascular Science
http://www.cvs.med.ed.ac.uk/

Centre for Reproductive Biology
(incorporating the MRC Human Reproductive Sciences Unit)
http://www.crb.ed.ac.uk/

New insights into menstrual disorders and reproductive cancers are being generated through collaboration within the Centre for Cardiovascular Science on the importance of local steroid hormones, and on mechanisms signalling responses to low oxygen levels (with colleagues in the Centre for Inflammation Research).

Future developments

The QMRI will form a nucleus for future exciting developments. In particular, the Institute's medical cell biology research serves as an ideal springboard for development of the University's new Centre for Regenerative Medicine, directed by Professor Ian Wilmut, FRS.

It is envisaged that the Centre for Regenerative Medicine will be an early development within the Centre for Biomedical Research. This is a collaboration between the University, Scottish Enterprise and Edinburgh City Council that will deliver 1.4 million square feet of research space to allow companies to translate discovery science into new drugs, diagnostics and devices, with the aim of delivering both health and wealth gain to Scotland. The QMRI will serve as a crucial research engine for driving this exciting project forward.

Lastly, perhaps the most important contribution of the Institute will be to serve as a magnet to attract young scientists and clinicians from all over the world into training for a career in medical research in Scotland and beyond.

Margarete Heck, The Wellcome Trust Centre for Cell Biology, Institute of Cell and Molecular Biology, University of Edinburgh. Margarete.Heck@ed.ac.uk

The Michael Smith Building, Manchester

In 2001, the University of Manchester was awarded £15M from the Joint Infrastructure Fund (JIF) initiative. This grant, supplemented with more than £30M of University funds, was used to create a large facility for biological and biomedical research, the Michael Smith Building.

Named in honour of an alumnus of the University and Nobel Laureate, the research complex is sited at a central location in the University's biomedical corridor, adjacent to Medicine, Chemistry, the Manchester Incubator Building, the Core Technology Facility and the Wellcome Trust Clinical Research Facility. The 10,000 m^2 building is made up of four wings arranged around a central quadrangle, and provides research laboratories, core facilities, offices and meeting rooms for ~800 staff in up to 100 research groups. Scientists worked closely with the architects in order to provide a building that would encourage collaboration between research groups and facilitate access to a wide range of core equipment facilities.

In the last decade, a major programme of cell-biological research has been established at the University of Manchester. Concomitantly, the University has developed into a national centre for bioinformatics and genomics research. Scientists have been recruited with interests in complementary areas of cell biology, including adhesion and matrix assembly, protein trafficking and targeting, determination of cell fate, gene expression, and signalling. Other research areas, such as developmental and evolutionary biology, immunology and tissue repair, have also developed during this time. The Michael Smith Building was designed to integrate all of these research groupings, allowing them to address fundamental, holistic questions at all levels of enquiry, from molecular and cellular biology, through tissues to whole organisms.

The second major driver behind the development of the Michael Smith Building was the provision of technological resources essential to pursue comprehensive and integrative forms of experimentation. Contemporary technologies for bioimaging, structural analyses, genomics and proteomics are evolving rapidly and are dependent on sophisticated equipment and a skilled technical interface. The building clusters core facilities to provide easily accessible resources for researchers. Vertical 'hubs' of specialised secondary space at the junctions of the wings of the building aare configured to maximise access by all scientists. The main core facilities available in the Michael Smith Building include Bioimaging, Biomolecular Analysis, Electron Microscopy, X-Ray Crystallography, Microarray, Fermentation and Flow Cytometry. These facilities are available to all staff and students, and are maintained by fulltime expert staff.

The building houses about one third of the Faculty of Life Sciences' academic staff. Principal investigators are linked with one or more 'research groupings'. These are general areas of research activity, each one bringing together researchers with related interests.

Organelle Function in Health and Disease

Research in this grouping examines the mechanisms that promote the correct folding and assembly of newly synthesised proteins, and ensure that these molecules are transported to their appropriate compartment within a eukaryotic cell. Many diseases are related to defects in one or more of these processes, and work is focussed on protein biogenesis (examining the folding and assembly of proteins at specific subcellular locations and the translocation of these polypeptides into and across the membranes that define these organelles) and protein transport (defining the molecular machinery and mechanisms responsible for the sorting and transport of proteins between the different compartments of the endomembrane system). Both themes use a collaborative, multidisciplinary approach using a combination of biochemical, genetic, molecular genetic and biophysical techniques in both higher and lower eukaryotes. The objective is to understand more fully how the individual steps that delineate these processes occur and to define the mechanisms by which they are regulated. This will provide an intellectual framework within which specific diseases can be studied. An MRC co-operative award for the study of "Protein assembly and trafficking in eukaryotic cells" underpins the work.

Molecular Cancer Studies

Cancer cells differ from normal cells in a number of ways. They grow and divide in the absence of proliferative signals; they tend to be insensitive to anti-proliferative signals and evade normal apoptotic cues; they are immortal and have highly abnormal genomes; in addition, they can invade other tissues and hijack blood supplies. Consequently, understanding the mechanisms which result in the initiation, progression and maintenance of tumours requires an understanding of many basic biological processes. Molecular Cancer Studies brings together researchers who have an interest in these processes in relation to tumourigenesis. Under the umbrella of the Manchester Cancer Research Centre, this grouping has ties with other cancer scientists in Manchester including members of the Faculty of Human and Medical Sciences and the Paterson Institute. Research in this grouping is focussed on three main themes. Cell cycle: understanding the mechanisms regulating progression through the cell division cycle including DNA replication, mitotic entry and chromosome segregation. Cell fate: studies on how cells decide to differentiate, survive or migrate depending on their environment. Cell signalling: investigating the intracellular communication networks which link receptor signalling and gene expression.

Wellcome Trust Centre for Cell Matrix Research

The Wellcome Trust Centre for Cell-Matrix Research is a multidisciplinary research centre embedded within the Faculty of Life Sciences. The centre aims to elucidate the structure and function of extracellular matrices (ECM) and their cellular receptors, to define the role of ECM and cell-ECM interactions in normal development and human diseases. The molecular mechanisms that determine survival, differentiation and movement in multicellular organisms are dependent on interactions with the ECM. Cells in tissues are structurally and functionally integrated with their surrounding ECM via numerous dynamic connections. On the intracellular face of these linkages, adhesion receptors tether the contractile cytoskeleton to the plasma membrane and compartmentalise cytoplasmic signalling events, while at the extracellular face, the same receptors direct the deposition of the ECM itself. These membrane-proximal functions trigger distal processes, such as alterations in the direction of cell movement and the regulation of cell fate, and the construction of ECM networks and consequent shaping of higher order tissue structure. Understanding the molecular events that underpin ECM function would therefore help elucidate some of the key organising principles of multicellular life. ECM and cell-ECM interactions also contribute widely to disease. Many of the major human diseases are either caused by defects in cell-ECM coordination, are exacerbated by aberrant use of normal cell adhesive processes, or are potentially correctable by altering tissue structure or cell movement. The development of strategies to correct ECM dysfunction has enormous promise as a route for improving treatment of many important clinical conditions. Research within the Centre is packaged into four programmes: Matrix assembly, Adhesion Signalling, Cell Fate Determination & Tissue Regeneration.

Gene Expression

Manchester is a major centre for research in gene expression and its critical role in several biological processes including development, cancer, stress responses and biological clocks. A variety of model organisms/systems are used, from yeast to mammals, to study different aspects of gene expression including signal transduction, transcriptional regulation, RNA processing and translation. Research in this grouping focuses on three main programs: The regulation of transcription: studying the mechanisms by which both general and sequence-specific transcription factors function at the gene promoter. Cellular signalling mechanisms: understanding how signals are sensed by cells and transmitted to cytoplasmic and nuclear substrates. RNA biology and the control of translation: investigating the biological activities of RNA and RNA-protein complexes.

Developmental Biology

Research in this grouping focuses on Cell fate specification and differentiation, Patterning, morphogenesis and organogenesis, Developmental cell signalling, and Developmental genetics and Human disease. The research uses a variety of model organisms and systems including *Dictyostelium*, *Drosophila*, *Arabidopsis*, *Xenopus*, cockroach, zebrafish, chick, mouse, nervous system, mammary gland, epidermis, hairs and feathers, palate, germ cells and stem cells. A diversity of state-of the-art techniques are employed, including various types of genetic analysis, transgenesis, gene targeting, expression profiling, cell and organ culture, proteomics, microscopic imaging, electron microscopy, biophysics and X-ray crystallography

Neurobiology

Members of this research grouping study the central and peripheral nervous systems in health and disease.

Facing page: The Michael Smith Building, Manchester.

Above: Inside the Michael Smith Building.

Working with a variety of model systems from *Drosophila* through to man, the grouping maintains expertise in genetics, molecular and cell biology, as well as anatomy, electrophysiology and aspects of behaviour. Specific strengths lie in the areas of neuroinflammation and neuropathy, circadian rhythms, neuroendocrinology and development.

Channels and Transporters

The vast majority of chronic diseases can be related to abnormal structure or function of one or more membrane transport proteins. Research within the Channels and Transporters group spans the molecular structure, function and regulation of these important proteins, as well as their pharmacology and role in disease. Particular areas of interest include pancreatic stem cells and diabetes, fluid and electrolyte transport in epithelial cells, chloride channels in the choroid plexus, potassium channels in vascular smooth muscle and endothelial cells, cardiac and neuronal voltage-gated calcium channels, oxygen-sensing ion channels, urea transporters and calcium-sensing receptors in the kidney and vasculature. Research addresses the characterisation of ion channel and transporter genes, trafficking and localisation of ion channels in the membrane, spatio-temporal patterning of cytosolic calcium signals and other signalling pathways, such as the inositol lipids. Future work will continue to advance knowledge and understanding of transport mechanisms in these physiologically important areas.

Bioinformatics and Functional Genomics

This grouping seeks to study and understand biological function through 'wet' and 'dry' approaches that build up from molecular building blocks to genome and systems wide features. Bioinformatics groups use and develop sequence-based databases and tools to study motifs and collate genomic information for species such as chicken. Much can be learned from comparing genomes, such as molecular evolution and gene networks in yeasts and bacteria, or how genes are regulated in the fly. Molecular evolution and phylogeny are also common themes, studying species from wheat through pathogens (HIV) to humans. Functional genomics - transcriptomics, proteomics and metabolomics – is applied to areas such as yeast metabolic control and molecular parasitology, and is complemented by attendant statistical analysis and software development. Structural bioinformaticians are studying themes such as protein evolution and interactions, function prediction and electrostatics.

Evolutionary Biology

Research in this grouping focuses on understanding how complex characters evolve, and the translation of genotypes into complex phenotypes. Approaches used include population and quantitative genetics as well as phylogenetics, and we apply molecular, statistical and modelling techniques. The main research foci are behaviour, development and ecological genetics. Research is conducted on model and non-model organisms, with an emphasis on mammalian, insect and plant systems. In addition to the development of mathematical models to complement our empirical work, statistical techniques and new applications of statistics are being developed.

Immunology

The response to damage arising from infection, wounding or autoimmune/hypersensitive episodes involves the complex interplay between the molecules and cells of the inflammatory, innate and adaptive immune response and, in the case if infection, virulence and evasion strategies of the pathogen. This research grouping applies an integrated in vitro and in vivo approach to defining the mechanisms underlying disease processes, from induction through to resolution.

The future

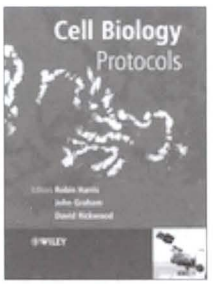
The final phase of building, Wing D, has recently been completed. Construction on the Smith Extension is now well underway. This facility is located between the Michael Smith Building and the Core Technology Facility, which houses researchers from the Faculties of Life Sciences and Medical and Human Sciences as well as biotech companies. The building, due for completion in 2008, will have a neuroscience focus and includes dedicated electrophysiology and histology suites. Aerial bridges will connect the three buildings, providing a physical link between the molecular cell biology in the Michael Smith Building and the neurobiology and physiology in the in the Smith Extension and Core Technology Facility. This work is part of a larger University-wide program of capital building worth around £600M, the biggest in UK higher education to date. In terms of research, the Faculty recently undertook an ambitious recruitment exercise, which has already resulted in the appointment of more than 10 new scientists in areas spanning the Faculty's research portfolio. Many of the new appointees have a background in physical science, and a key area for future development is the interface between physical science and life science. As bioscience becomes more quantitative, the use of physical and mathematical methods will enable a more detailed understanding of the biomolecular and cellular components of living systems. To this end, three Physical/Life Science Interface Fellowships have just been awarded, and it is anticipated that this scheme will continue in the future. Together, these developments will help to ensure that cell biological research at Manchester continues to expand in the next decade.

Lisa Swanton, University of Manchester.

Book Reviews

Cell Biology Protocols

EDITED BY ROBIN HARRIS, JOHN GRAHAM AND DAVID RICKWOOD



Cell Biology Protocols
John Wiley & Sons
ISBN: 0-470-84758-1
January 2006

Cell biology is one of the fundamental areas of research that incorporates a broad range of laboratory methods and techniques. The present book is a collection of a wide variety of such protocols that could be applied by anyone who works on cell biology, from the student to the experienced scientist. Overall, the format of this book is very helpful, with tables, figures and graphs cleverly supplied where more information is needed. Reagents and materials are described in detail and the steps of the protocols are presented in bullet-points in short but clear sentences that would be easy to follow in praxis. Also, each chapter has a short introduction that orients the reader to the content of the protocols that are to follow.

In specific terms, the book covers six areas of cell biology methods. The first two chapters give a thorough insight into light and electron microscopy. The basic principles of the function of the light and electron microscope are well reviewed and accompanied by detailed yet clear to understand schematic figures. This theoretical explanation is followed by a number of protocols on the processing of biological specimens for microscopy, whether they are tissue sections, cells or organelles.

The next chapter describes methods for primary as well as secondary culture. Starting with the extraction of the cells of interest from fresh tissue, the procedure is explained step-by-step to the method of cell isolation from a heterogeneous culture to cell sub-culture as well as thawing and freezing of cell lines. Cell counting, quantification of cell viability and purification methods are included. Admittedly, there is a degree of variation in cell culturing methods depending on the cell type, however the authors present a very good baseline of protocols with great efficacy regarding any cell type.

The subsequent chapter deals with the purification of subcellular membranes, organelles and organelle components. For example, protocols describe isolation or preparation of nuclei, nucleolei and nuclear membranes, chromosomes, mitochondria and lysosomes as well as separation of the smooth and the rough ER. Some protocols cover the preparation of organelle components of plant tissues. Also a number of assays for quantification of enzymatic activity can be found.

The next chapter is dedicated to the separation of subcellular domains to be used in studies related to membrane trafficking and cell signalling, using different gradient media. Sucrose, Nycodenz® and iodixanol gradients are mainly used in the protocols provided in order to fractionate subcellular compartments such as lipid rafts, the apical and basolateral domains of polarised mammalian cells and the ER and Golgi systems.

The last chapter presents a wide variety of in vitro assays of reconstitution in cell biology dealing with nuclear components, cell membrane systems as well as cytoskeletal and fibrillar systems. A few examples of the protocols presented are DNA labelling techniques for cell functional studies, techniques for the study of nuclear–matrix interactions and the uncovering of nuclear matrix for microscopical observation as well as nuclear assembly techniques. The concept of the use of nanocapsules to improve drug delivery intracellularly is well interpreted, as are apoptosis detection assays and a microarray-based protocol for studies on membrane transport processes. In addition, protocols relating to fibrillar systems, amyloid-β-enzyme interaction and amyloid-β phosphorylation can be found. The final additional chapter contains useful information on chemical safety and the procedure of centrifugation.

Numerous references are provided at the end of each protocol for further reading as well as notes, explanatory comments and tips which often prove to be crucial for the success of a laboratory technique. A valuable possession for every cell biologist's library.

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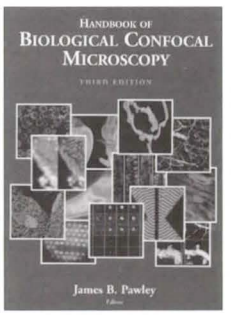
Handbook of Biological Confocal Microscopy, 3rd Edn

EDITED BY JAMES B. PAWLEY



Handbook of Biological Confocal Microscopy, Third Edition
Edited by James B. Pawley
985 pages; Springer Science+Business Media, 2006
ISBN 0-387-25921-X

The long awaited 3rd edition of the '*Handbook of Biological Confocal Microscopy*' is now out. It is a hefty offering with 52 newly written chapters plus 3 appendices by 123 authors, tallying in at 985 pages in A4 format. In its previous incarnation, the 1995 2nd edition had been referred to reverently by many as the 'Bible of Biological Microscopy'. Thus, expectations for the new edition of this book were high and it does not disappoint. It succeeds in covering authoritatively, with chapters written by world-class experts in a comprehensible style throughout, all important aspects of modern microscopy techniques from the basics to the latest advances in instrumentation, microscopy techniques and software.

The range of topics is impressive indeed. To name but a few, optical elements, digitizing of image data, lasers, contrast formation in microscopy, disk spinning microscopy, measurements of point spread functions, photon detection and CCD cameras, problems with signal-to-noise ratio, deconvolution techniques used in microscopy, data storage, and display and image presentation software are covered expertly. Other chapters deal with fluorophores and selection of fluorescent probes for live cell imaging, and some of biologists' favourite techniques/ approaches such as Fluorescence Recovery after Photobleaching (FRAP), Fluorescence Resonance Energy Transfer (FRET), Fluorescence Lifetime Imaging (FLIM), ion imaging, and automated high-content screening by confocal microscopy. Thick, large specimens pose special problems in live cell imaging. Three chapters in the book therefore deal with the theory and practical aspects of live multi-photon imaging of cells, brain slices and embryos, and a chapter by Ernst Stelzer (EMBL Heidelberg) describes selective plane illumination microscopy (SPIM) as an alternative to multi-photon imaging of thick live specimen like embryos. At the other end of the size spectrum, Stefan Hell (MPI Göttingen) discusses 4-Pi microscopy, a technique that manages to break the "diffraction barrier", Abbe's predicted limit for the resolution of light microscopes (~250 nm). With special instrumentation, this allows resolution of small structures in the size range of 30–40 nm in living cells. Correlative light and electron microscopy is described in another chapter as a technique that allows live cell observations followed by high resolution electron microscopic analysis of the same structures previously highlighted by GFP-chimeras in cells of interest, after fixation.

The quality of the figures and illustrations is excellent throughout, all in best Springer tradition, which adds to the book's impact and appeal and helps to keep track of the many complicated topics discussed. In summary this book, which is a 'must have' for imaging centres and university libraries as a reference book, is also highly recommended for individual scientists and for any laboratory seriously interested in the complex practical and theoretical aspects of modern live cell imaging.

Rainer Duden, Royal Holloway University of London.

Principles of Gene Manipulation and Genomics

S. B. PRIMROSE AND R. M. TWYMAN, EDITORS

When the first viral genome (ϕX174) was completed[1] in 1977, about 1000 base pairs could be sequenced a year. Completing the *E. coli* K-12 sequence at that pace would have taken over a millennium, while the sequencing of the human genome would have required over a million years[2].

Yet the first genome of free-living microorganism was sequenced[3] in 1995, that of *E. coli* K-12 strain MG-1655 was completed[4] in 1997, and the first draft of the human genome was published[5,6] in 2001. Biomedical sciences have witnessed amazing advances over the past few years, and these advances represent a powerful testimony to the plethora of new techniques that have been developed and implemented. *Principles of Gene Manipulation and Genomics* insightfully describes these advances. At the same time, the book accomplishes much more: it discusses principles and concepts, ranging from simple to more intricate ones, in several disciplines that converge on the increasingly interdisciplinary and multidisciplinary fields of gene manipulation and genomics.

Published for the first time in 2006, *Principles of Gene Manipulation and Genomics* forges a wonderful link between genetics and genomics; between the study of genetics in the pre-and post-genomic era; between the past, the present and the future. The volume combines material previously covered in two separate books. One is *Principles of Gene Manipulation*, a text dedicated to recombinant DNA technology and genetic engineering in various organisms; the second is *Principles of Genome Analysis of Genomics*, the last edition of which addressed several fascinating topics about the newly emerging field of genomics.

The four parts of *Principles of Gene Manipulation and Genomics* are organized into 26 chapters, and cover topics related to cloning, mutagenesis, DNA sequencing, genetic manipulation in various organisms, genome organization, genomics, proteomics, transcriptomics and bioinformatics. The book includes two new chapters on the applications of genomics.

The abundance of figures and tables throughout the volume, together with boxes illustrating key ideas, immensely facilitate the comprehension of difficult concepts. Every chapter is followed by a list of suggested readings and many of the references are accompanied by succinct explanations of the merits of the specific paper. The list of references often concludes with a review of useful websites relevant to the topic(s) discussed.

Although *Principles of Gene Manipulation and Genomics* targets an advanced undergraduate audience, the text will greatly benefit a much broader readership. For some readers, it will provide the basics and guide their first steps through the field; for others, it will be an opportunity to reminisce about long-forgotten, yet important principles, ideas and techniques. The book will provide a formidable text for anyone interested in the field, from students to professionals.

Richard A. Stein, Michael Heidelberger Division of Immunology, Department of Pathology, New York University Medical Center, New York, NY 10016, steinr01@med.nyu.edu

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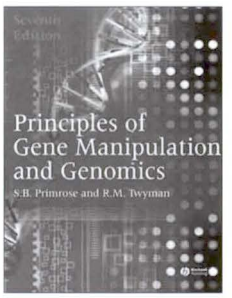


Principles of Gene Manipulation and Genomics
Blackwell Publishing,
ISBN 1-4051-3544-1
2006, xxii + 644 pages

Books for review

DNA Topology Bates & Maxwell, OUP

Essential Developmental Biology Slack, Blackwell

Cell Imaging: Methods Express Stephens, Scion Publishing

Whole Genome Amplification: Methods Express Hughes & Lasken, Scion Publishing

Immunohistochemistry: Methods Express Renshaw, Scion Publishing

Bioinformatics: Methods Express Dear, Scion Publishing

Proteomics: Methods Express O'Connor, Scion Publishing

A Textbook of Neuroanatomy Patestas and Gartner, Blackwell

Skeletal Development and Remodelling in Health, Disease and Aging Zaidi, Annals of the New York Academy of Sciences

Epithelial Anion Transport in Health and Disease Novartis Foundation symposium 273 Wiley

Heart Failure: molecules, mechanisms and therapeutic targets Novartis Foundation symposium 274 Wiley

Purinergic Signalling in Neuron-Glia Interactions Novartis Foundation symposium 276 Wiley

Protein Degradation: Cell biology of the ubiquitin-proteasome system Mayer, Ciechanover and Rechsteiner, Wiley VCH

Bioinformatics: genomics and post-genomics Dardel and Kepes, Wiley

Cancer Bioinformatics Nagl, Wiley

Meeting Reports

Neurospora 2006

30 March – 2 April 2006, Asilomar Conference Center, Pacific Grove, California, USA.

This meeting takes place every 2 years, alternating annually with the broader and larger Fungal Genetics Conference at the same venue. Concentrating solely on the model filamentous fungal genera, *Neurospora*, the meeting is a great opportunity for the younger scientists to interact with key figures in this lively field of research.

The conference grounds are not far from Monterey, situated at the northern edge of the famous 17 mile drive and California's Big Sur coastline. The onsite accommodation and catering meant that the delegation could spend time with one another out of the formal talks. And with a beach just a few hundred metres away, relaxing after a day of seminars was very easy. Talks took place in the Chapel, and as there were no concurrent sessions, we could attend all the talks and not miss out on anything.

In this 4-day meeting, we had a great opportunity to get to know recent research in *Neurospora* field. On the first day, the conference opened with a welcome 'mixer' where we were encouraged to get to know the other delegates.

The morning session of the second day was '**From Genes to Populations**'. Most of the talks were very interesting, in particular 'Control of DNA methylation in *Neurospora*' by **Eric Selker** (University of Oregon, OR). He presented their current understanding about how A:T-rich DNA triggers methylation. The DIM-2 DNA methytransferase is directed by heterochormatin protein (HP1), which in turn recognizes trimethyl-lysine 9 on histone H3, placed by DIM-5 histone H3 methyltransferase. DNA methylation can lead to deacetylation of histones, which may aid in propagation of DNA methylation and the associated silenced chromatin state. Another talk was 'So, a protein involved in hyphal fusion localizes to septal plugs' by **Andre Flessner** (University of California, CA). In this talk, they labelled the SO protein with GFP and to show this protein will accumulate at the septal plugs.

This was followed in the afternoon with a workshop on how to utilize the latest online tools and resources that have emerged from the *Neurospora* genome project, given by the creators and curators of these websites. This really opened my eyes to what was already available. The speakers also encouraged the audience to suggest ways of improving the resources to offer a better service.

In the evening, speakers on the session '**Genomics and program project report**' presented research on the *Neurospora* genome.

The next day contained the sessions of '**Cell morphogenesis and Assembly**' and '**Cell signalling and Gene Regulation**'. In the session of 'Cell signalling and gene regulation', my supervisor, Prof. **Nick Read** (University of Edinburgh), presented Hsiao-Che Kuo's recent study on sexual reproduction in *Neurospora crassa* and raised some interesting discussions with the audience.

Other talks, especially that of **Gregory Jedd** (National University of Singapore, Singapore) on Woronin body formation was directly related to Graham Wright's research. Woronin bodies are peroxisome-derived organelles that are centered on a crystalline core of the HEX-1 protein and function as emergency patches of the septal pore. Their results define the genetic dfferention of apical hyphal compartments and show that polarized gene expression is a key determinant of apically localized Woronin body-genesis (Tey et al., 2005).

In the evening poster session, both of us presented two posters and interacted with other researchers interested in topics. Graham Wright's poster, showed the application of the optical tweezers for micromanipulation in *Neurospora crassa*. We have built a simple, safe and user-friendly optical tweezers system that can be mounted on a commercial microscope and is importantly, easy to use for biologists who lack optics experience. In Hsiao-Che Kuo's poster, the early stage of sexual reproduction in *Neurospora crassa* was presented. Because *N. crassa* is a model filamentous fungus, it is very easy to handle and answer basic biological questions. This poster showed female and male nuclei behaviour in the very early stage of the sexual reproduction.

The later session in the last day, '**Clocks, light, and Oxygen**', discussed how the fungus senses the environment. **Yi Liu** (University of Texas Southwestern Medical Centre, TX) talked about the molecular mechanism of light responses in *Neurospora*. **Carlo Cogoni** (Universita' degli Studi di Roma 'La Sapienza, Italy) presented the quelling machinery in *N. crassa*. The identification of genes required in the silencing process together with findings from other organisms has led to a current model for quelling. In *Neurospora*, it would seem that quelling is severing to limit the expansion of transposons since an introduced Tad element, a LINE-1-like retrotransposon, has an elevated expansion in the absent of the quelling components QDE2 and DICER.

The whole experience of attending and presenting such an important international conference of *Neurospora* was fantastic. I learned from and enjoyed the conference very much. Also, it was good to get to know people in the same field of research from around the world. In addition to the BSCB, we would like to thank James Rennie Bequest travel award for the financial support to attend the *Neurospora* 2006 conference.

Hsiao-Che Kuo and Graham D. Wright, University of Edinburgh.

British Yeast Group Meeting 2006

Fisher's Hotel, Pitlochry, 10–12 April 2006

BYG 2006 was the latest in a series of annual meetings that bring the UK yeast research community together, providing a forum for 28 offered talks and 43 posters punctuated by 8 longer talks by invited speakers. As in previous years, younger scientists in particular were encouraged to be active participants, benefiting from being able to present their work in front of a critical but supportive audience.

The invited presentations at BYG 2006 covered a range of topics including cell-cycle control, protein targeting, transcriptional regulation, *Candida* pathogenesis and chromatin remodelling. The meeting got off to a great start with a presentation by **Tomo Tanaka**, who used an elegant genetic trick to enable the capture by a microtubule and subsequent biorientation on the mitotic spindle of a single chromosome to be visualised directly in budding yeast. Using this system, he showed how in most cases the side rather than the plus end of a microtubule captures kinetochores initially, with subsequent transport to the spindle involving the kinesin Kar3.

Frank Uhlmann continued the cell cycle theme with striking new data demonstrating a role for protein phosphatase 2A (PP2A) in controlling mitotic exit. In *S. cerevisiae*, Cdc14 phosphatase is required for mitotic exit and is released from the grip of Net1 in the nucleolus as cells go into anaphase, achieved through Net1 phosphorylation. Frank showed that PP2A antagonises phosphorylation of Net1 in metaphase until Separase (Esp1), in a process distinct from its role in triggering chromosome segregation through proteolysis of cohesin (the sister chromatid 'glue' destroyed to trigger anaphase), binds to and inhibits PP2A so that Net1 phosphorylation can occur. **Tony Carr** discussed the role of the COP9/Signalosome complex in fission yeast, where it controls the nuclear export of the small subunit of ribonucleotide reductase that normally occurs in response to slowed DNA replication or DNA damage. **Neil Gow** went on to describe how different cell wall components in Candida albicans are involved in its virulence, showing how these components act through different pathways to trigger cytokine production in cells of the immune system.

For most of us, the budding yeast GAL promoter is simply a way to switch the expression of our favourite gene on and off at will, but **Richard Reece** explained how this transcriptional switch works at the molecular level, with Gal3 working as the galactose sensor. Gal3 is really an inactive galactokinase, and the same mutations that convert the real galactokinase (Gal1) into an enzyme that phosphorylates glucose can be introduced into Gal3 to make the system respond to glucose instead of galactose.

For those of us under the illusion that the yeast post-Golgi secretary pathway in yeast is a unitary affair, **Anne Spang** showed that specialised cargos such as chitin synthase 3 are exported from the Golgi in vesicles with a novel Arf-dependent coat consisting of Chs5 and the ChAPs (a family of four related proteins), a route distinct from that by which most proteins reach the yeast cell surface. While many of us are familiar with the concept of nuclear 'factories' for replication, transcription and ribosome biogenesis, it turns out that yeast also has discrete nuclear centres for removing defective or unwanted pre-ribosomes: **David Tollervey** described the identification of the 'No-body', a sub-nuclear structure in which the exosome and the TRAMP complexes that degrade unwanted pre-ribosomes are enriched.

As might be expected for a meeting organised around a group of highly related experimental organisms rather than a specific subject, the offered presentations covered a diverse range of topics. Despite this, the talks were of a high standard and managed by and large to sustain the interest of the audience. The poster session, though equally varied, also demonstrated the wealth of high quality research that is currently being carried out by the UK yeast research community. Holding BYG 2006 in a hotel rather than the customary University campus location was originally triggered by the unavailability of accommodation at the organiser's own institution, but had the unexpected benefit of catalysing many interesting discussions and promoting a high level of scientific interaction.

Mike Stark, Division of Gene Regulation and Expression, School of Life Sciences, University of Dundee.

BYG 2007 will be held in Sheffield from 26-28 March 2007. Contact Alastair Goldman (a.goldman@sheffield.ac.uk) for further details.

World Congress meeting of the Tissue Engineering and Regenerative Medicine International Society (TERMIS)

Pittsburgh, USA 24–27 April 2006

With nearly 1000 delegates, 150 oral presentations and over 360 poster presentations this was the premium tissue engineering meeting of 2006 which proved an informative and highly enjoyable conference.

The Tissue Engineering and Regenerative Medicine International Society (TERMIS) meeting, organised and hosted by the Pittsburgh Tissue Engineering Initiative (PTEI) and the McGowan Institute for Regenerative Medicine in Pittsburgh, was created from the merged former Tissue Engineering Society International (TESi) and the European Tissue Engineering Society (ETES). The venue for this exciting meeting was the Westin Convention Centre in the heart of Pittsburgh's vibrant metropolitan complex.

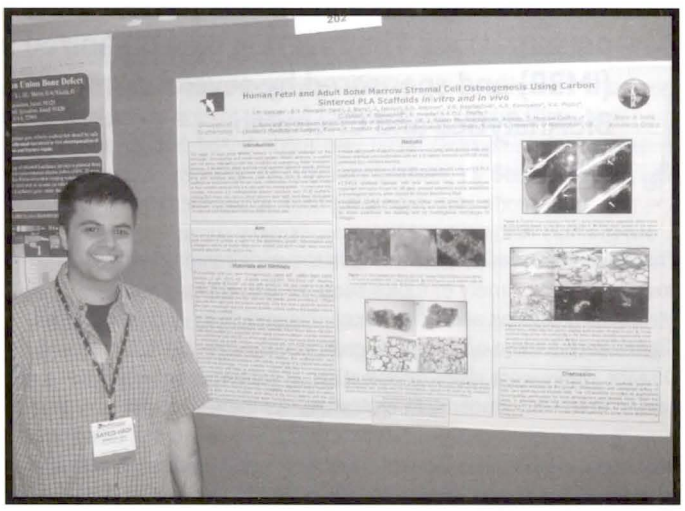
A number of activities were organised prior to the conference for the members of the TERMIS Students and Young Investigators Section (SYIS). The Pittsburgh meeting saw the launch of TERMIS-SYIS, a society of which I have been a part of since my involvement in the creation of the section at the previous TESi meeting in Shanghai in 2005.

The SYIS activities included a tour of the impressive McGowan Institute and an opportunity to meet student and post-docs currently working at the McGowan to discuss their research. This was followed by a 'Meet and Greet' gathering of students who had just arrived. This was a great opportunity to meet other young researchers and discuss the current research landscape with other like-minded students. The evening also included an open discussion of the recent book '**Redesigning Humans: Choosing our Genes, Changing our Future**', with the author **Dr Gregory Stock**, the director of the Program on Medicine, Technology, and Society at UCLA. This was an excellent session, both informative and stimulating. Dr Stock eloquently described the promise that the fields of stem cell research and regenerative medicine can offer to ageing and disease, and also the perils of progressing without the adequate controls or responsibility. Other issues discussed included the genetic and bionic development of society, and the postulated spilt between the genetically enhanced human and those without, although as Dr Stock argued, we have already begun our technological evolution as personal hi-fi and mobile phones can be viewed as bionic extensions of ourselves!

The congress was opened by **Dr Alan Russell**, the director of the McGowan Institute for Regenerative Medicine and president of TERMIS, and welcomed Dr Stock who gave an opening plenary on issues discussed within his book. This was followed by parallel sessions on topics such as stem cell and biomaterials, and of particular interest to me, talks given on the current developments in orthopaedics. **Dr Dietmar Hutmacher** of the National University of Singapore spoke on the *in vitro* engineering of tubular bone grafts using the electro-spinning technique to fabricate scaffold matrices. Bone marrow stromal cells in monolayer were cultured in osteogenic conditions before these cell sheets were wrapped around scaffolds and implanted into mice. Viability of cells was demonstrated after four weeks with fluorescent cell tracking and molecular analysis showed the expression osterix, osteopontin and osteocalcin, proteins found within bone tissue. **Dr Ivan Martin** of the University Hospital Basel, Switzerland, showed expansion of human bone marrow stromal cells within porous three-dimensional scaffolds seeded via a dynamic perfusion system, allowing cells in suspension to wash through the scaffold until they adhered.

The poster sessions allowed discussion of my work entitled 'Human Foetal and Adult Bone Marrow Stromal Cell Osteogenesis Using Carbon Sintered PLA Scaffolds *in vitro* and *in vivo*', work performed in collaboration with Dr Janos Kanczler within our Bone & Joint Research Group at the University of Southampton. Our work demonstrated a proof-of-principle for the use of rapid prototyped biomaterials based on the resorbable poly-lactic acid (PLA) polymeric scaffolds, and attracted attention from other bone tissue engineers as well as other groups working on human foetal tissue, currently limited within the United States. Therefore this session gave our work a platform and importantly, allowed me to present research currently carried out in the UK within an international setting.

Personally, the highlight of the conference for me was a talk given by **Dr Pamela Robey** of the National Institutes of Health, Maryland, USA, who discussed her work on post-natal skeletal stem cells for maxillofacial regeneration. Dr Robey's excellent presentation detailed the difference between mesenchymal stem cells and the heterogeneous bone marrow stromal cell population they are derived



from, and the importance of clonal studies when investigating multilineage potential of isolated mesenchymal populations. Dr Robey also described the requirement now for functional and robust new tissue, not just the expression of specific markers, and the current Food and Drug Administration (FDA) rulings on the removal of animal-derived serum for 4 days prior to clinical use, which allows for the survival of *in vitro* cultured cells. In addition, her latest work into maxillofacial regeneration demonstrated the efficacy of hydroxyapatite/tri-calcium phosphate scaffolds for the development of new bone marrow stroma. In addition, Dr Robey showed that removal of the surrounding periosteum prior to surgery provided better integration of implanted scaffolds within bone.

The TERMIS-SYIS activities continued with the 'Student meet Mentor' session in which students and young investigators could have their questions answered by leaders within the field. I spoke with **Dr Anthony Atala** of the Wake Forest Institute of Regenerative Medicine who had just published data that week on the successful clinical follow-up of tissue engineered bladders in patients after six years, which had made the UK national press. His insights into the future developments of tissue engineering were highly valuable, and he encouraged the group to maintain the multi-disciplinary effort required for successful application of regenerative medicine, through continued collaboration of clinicians, surgeons and research scientists.

Another highlight of the conference was the morning plenary session of the second day, chaired by **Michael Lysaught** of Brown University, USA, and **Robert Nerem** of Georgia Institute of Technology, USA, who also contributed to the session entitled '**Tissue Engineering Centres from Around the World: An Overview**'. Included in the plenary session were Dr Russell, Dr Atala and **Dr David Williams**, head of the UK Centre for Tissue Engineering at the University of Liverpool. Each speaker described their research institution with their current aims, faculty and facilities and discussed the challenges faced in regenerative medicine, not only with regard to science but securing future funding as well.

Other interesting presentations included an excellent overview of the current use of mesenchymal stem and progenitor cells for bone repair given by **Prof Ranieri Cancedda** of the National Institute for Cancer Research, University of Genoa, Italy. Prof Cancedda detailed the *in vivo* resorption of silicon/tri-calcium phosphate scaffolds in comparison to hydroxyapatite, and described the importance of scaffold resorption in new bone formation. Prof Cancedda also described the lack of immunological response to allogeneic mesenchymal stem cells, which were shown to suppress the proliferation of T-lymphocyte immune cells *in vitro*. This will prove important for the use of these cells in future cell-based tissue regeneration strategies.

Miss Doreen Hamann, a PhD student from University of Twente, the Netherlands, described the use of tri-phasic (three-layered) scaffolds to engineer an osteochondral construct, with osteogenic differentiation conditions at one end of the scaffold and chondrogenic conditions at the other. Bone marrow stromal cells could not attach to the materials within the chondrogenic conditions, so cell aggregates were formed prior to seeding which then allowed cellular attachment. This work is important as our current understanding of these tissues allows us to start to manipulate these systems, so that we may engineer an interface between new tissue types for improved integration and enhanced development.

As the conference progressed many of the sessions turned to clinical translation and commercialisation. An interesting presentation by **Dr Anthony Ratcliffe**, president of Synthasome, Inc., San Diego, USA, discussed the cost of bringing innovative ideas and medicines to the marketplace. He also encouraged any business model to be both realistic and honest and promoted working with pharmaceutical companies to develop new tissue engineering strategies.

Before returning home I was able to visit to the Andy Warhol museum and John Heinz History centre, and to the main campus of the University of Pittsburgh which included the impressive Cathedral of Learning, one of the tallest educational centres in the world; not forgetting my visit to the PNC Baseball Park, home of the Pittsburgh Pirates, overlooking the beautiful Pittsburgh skyline. Go Pirates!

Altogether, this proved a memorable and enjoyable trip that allowed an opportunity to develop new friendships and foster new collaborative links. Therefore I would once again like to offer thanks to the BSCB who made my attendance at this excellent conference possible.

Sayed-Hadi Mirmalek-Sani, Bone & Joint Research Group, University of Southampton

European Worm Meeting

29 April – 3 May 2006, Hersonissos, Crete, Greece

This five day conference was packed with 93 talks and 162 poster presentations. The meeting was organized by Dr. Tavernarakis (IMBB) and sponsored by several companies including Roche, BioAnalytica and Chemilab.

The conference was divided into thirteen wide-ranging sessions covering a wide variety of topics such as: Cell Biology and Metabolism, Ageing and Senescence, Neurobiology and Behaviour, Signal Transduction and Gene Expression, Development, Stress and Disease. Talks were given by scientists at varying stages of their career, from PhD students to professors. As there were no simultaneous presentation, it was possible to attend each talk and thus concentrate better without missing out any of them. I was very keen on attending this conference because I have been working for almost two years on *C. elegans* and it was my first opportunity to see a comprehensive series of talks and presentations about the worm.

Following the welcome by **Nektarios Tavernarakis**, the local conference organizer, the meeting opened with a talk about a novel exosome-mediated apical secretion mechanism in epidermal cells. The presenter showed that multivesicular bodies can release hedgehog-related peptides (HRPs)-containing exosomes through VHA-5. They infected worms with fungi and observed that secretion defective VHA-5 mutants appear hypersensitive to infection. Therefore, there is a

possibility that VHA-5 is necessary for efficient innate immunity.

In the session covering issues in **Gene Expression**, **Thomas Burglin** challenged us with the dynamic expression of Homeobox genes during embryogenesis. I was so impressed when he showed each cell separately with a different colour by two-channel 4D microscopy using live GFP time-lapse recording. He recorded GFP expression of eleven *ceh* genes during early embryogenesis. Interestingly his lab is developing several software tools to convert GFP expression data into digital data.

I am particularly interested in transcription factor regulation, and **John Reece-Hydes** talk was about my particular interest. His work is a part of Localization of Expression Mapping Project (LEMP) with the aims to use cloned *C. elegans* promoter fragments to generate a genome-wide set of expression patterns termed Localizome. His group identified 934 transcription factors in the genome, which are accessible to the scientific community online and he discussed the implications of having access to that valuable data.

Another interesting talk was identification of evolutionary conserved DNA damage response. **Gijs van haafften** used RNAi to screen for genes that protect cells against ionizing radiation, and identified a total 45 genes. These include orthologs of well-known human cancer predisposition genes. He described that almost all identified genes in his screen are conserved across animal phylogeny, indicating that this set of genes is important for future cancer treatment.

In addition to the interesting and informative talks, the conference provided me with an opportunity to present my poster, entitled "Identification of upstream factors regulating the expression of the T-box gene mab-9". The posters were on display throughout the entire meeting, as well as during scheduled poster viewing sessions, for delegates to view. I received some good feedback through discussion about my research work.

The social committee was equally enthusiastic and successful at organising our meals and extracurricular events. We were well-fed and entertained throughout the conference, particularly on the last evening when the conference ended with a Gala diner and traditional Greek dancing presentation.

There was a time set aside during the meeting for sight-seeing in Crete. We visited Knossos palace and a museum in Heraklion. Crete's fertile soil and towering peaks witnessed the development of the Minoan civilization (2800–1150 B.C.), one of the most important in the history of mankind. The Minoans built the famous palatial centres of that palace. In a visit to the Archaeological Museum of Herakleion we had a chance to see one of the most magnificent collections of Minoan art and culture in the world which covers a period of 5,000 years.

I found the meeting very exciting, educational, and valuable to my PhD studies. I would like to thank the British Society for Cell Biology (BSCB) for their generous provision of Honor Fell Travel Award which allowed me to attend this exciting conference.

Gholamali Jafari, Genetics Unit, University of Oxford

Society for Research on Biological Rhythms 10th Meeting

Destin, Florida, May 2006

The Society for Research on Biological Rhythms conferences are held biennially and provide an excellent forum for the exchange of current ideas in the fields of circadian rhythms and sleep.

The Society for Research on Biological Rhythms 10th meeting was held at the Sandestin Beach and Golf Resort on the Gulf coast of Florida and we were very happy to find that our hotel was close to both the conference centre and the beach. After a day spent exploring the resort and unwinding on the beach the conference began with an evening reception on the grand lawn in front of the conference center, facing the Choctawhatchee Bay.

The meeting began in earnest at 8.30am the next morning with parallel sessions examining the **Molecular Regulation of Circadian Rhythms** and **Neuronal Pacemaker Organisation and Disorganisation**. The first session of talks were followed each day by shorter slide presentations, usually by more junior researchers. These parallel sessions covered topics as varied as the role of clocks in disease, metabolism and sleep or focused on new findings about clock genes in *Drosophila*, mammals, plants and *Neurospora*. We were fortunate enough to have a few free hours after lunch each day to enjoy the resort before returning for the afternoon sessions.

The **Circadian clock mechanism session** held on the first afternoon of the conference was of special interest to us. **Justin Blau**, New York University, NY, talked about how larval *Drosophila* pacemaker neurons process light signals. He was followed by **Steve Reppert**, University of Massachusetts, who presented data about the organization and transcriptional control of the circadian clock in the Monarch butterfly, and **Charles Weitz**, Harvard University, focused on circadian clock control in the mouse retina.

The final speaker of the session was **Paolo Sassone-Corsi**, University of California, Irvine, talking about his group's data on the role of chromatin remodeling in clock function which suggests that CLOCK is actually a histone acetyltransferase.

The Tuesday morning session on **Transcriptional/Translational Feedback Loops** and **Neuronal Electrochemical Signaling** was extremely relevant to our work. The first talk was given by our

supervisor **Michael Nitabach**, Yale University, who presented my work looking at the involvement of Calcium signals in generating a feedback loop linking electrochemical events at the plasma membrane with the transcriptional oscillator in *Drosophila*. He also talked about Ying's work generating a series of tethered toxin ion channel inhibitors derived from spider venom which she will use to probe the role of specific ion channels in the *Drosophila* circadian oscillator. The second talk was given by **Chris Colwell**, University of California, LA, on the changes in conductance of fast delayed rectifier K^+ currents in SCN neurons over circadian time. He was followed by **Andrea Meredith**, University of Maryland, discussing how the absence of BK K^+ current in *slo*$^{-/-}$ mice alters their free running rhythms in locomoter behaviour. BK channels are expressed in the SCN and channel expression cycles with a circadian rhythm, peaking at night. The session closed with talk by **Bruce Bean**, Harvard University, on the mechanism of spontaneous electrical activity of SCN neurons in dissociated and slice cultures.

A series of short presentations on *Drosophila clock* genes followed after the tea break with an interesting talk by **Sheeba Vasu** (Todd Holmes' group), New York University, NY. She presented work continuing the examination of the effects of rendering the LNv subset of clock neurons hyper-excitable by expression of NaChBac. This included whole cell patch clamping data confirming the hyper-excitability of the LNvs and the molecular basis for the emergence of a stable bimodal oscillation in locomotor rhythm in NaChBac expressing flies in constant darkness. **Jeff Price**, University of Missouri-Kansas City, discussed work examining the effects of mutating Doubletime (DBT) on behavioural rhythms in flies and PER phosphorylation in cultured cells. He showed that DBT- dependent PER degradation required DBT kinase activity and that mutated forms of the mammalian DBT homologue Casein kinase1 functioned in an equivalent manner in *Drosophila*, confirming a high level of evolutionary conservation.

Juliana Benito (Paul Hardin's lab), University of Houston, presented work suggesting that the PAR domain protein 1E (PDP1E) functions to control oscillator output rather than as a component of the core oscillator itself. **Dan Stoleru** (Michael Rosbash's group) at Brandeis University, Massachusetts, presented his work examining the molecular and neural basis of photoadaptation in the circadian clock, focusing on the effects of disrupting cryptochrome (CRY) mediated photoreception in cells that control either evening or morning bouts of activity in *Drosophila*.

That evening the SRBR student and trainee social event was well attended, no doubt in part due to the free pizza and drinks.

One of the Wednesday afternoon sessions was dedicated to a workshop on the **Comparative Anatomy of Circadian pacemaker Networks in Arthropod brains**. This began with a brief but detailed overview of the best characterized of the insect circadian systems, that of *Drosophila*, presented by **Orie Shafer**, Washington University. In comparison much less is known of the molecular and neuroanatomical organization of the clock in Honey bees (*Apis mellifera*), as presented by **Guy Bloch**, The Hebrew University of Jerusalem. The *Apis mellifera* circadian clock consists of oscillating PER, CRY and CYC proteins, whilst CLK and TIM do not oscillate, suggesting that the clock in this insect species is more similar in organization to the mouse system than to *Drosophila*. This may be due to the divergence of *Drosophila* from the insect ancestral organisation, followed by the convergence of bees with mammals. The talk by **Makio Takeda**, Kobe University, on the neuronal organisation of the clock in the silkworm moth *Bombyx mori* was followed by an overview by the session chair **Charlotte Helfrich-Forster**, University of Regensburg. She concluded that although clock proteins are well conserved across insect species the functions and localisation of these proteins may vary. In addition, the insect circadian clock is probably comprised of a hierarchical network of neurons, similar to that seen in *Drosophila*.

Thursday morning was dedicated to talks by New Investigators and on Non-image forming photo-receptors, with other smaller sessions on Mammalian clock genes, plant and Neurospora clock genes and Human circadian rhythms. Later that afternoon, prior to the plenary lecture Charlotte Helfrich-Forster gave a moving tribute to Erwin Bünning (1906–1990) on the date of his 100th birthday. Professor Bünning, author of '*The Physiological Clock*', was a pioneer in the field of circadian rhythms.

The Pittendrigh/Aschoff Lecture was given by **Michael Young**, Rockefeller University, NY, on work carried out in his lab by Pablo Meyer and Lino Saez on the real-time movement and FRET visualised interaction of clock proteins between the cytoplasm and nucleus in cultured insect S2 cells. He reviewed their published findings showing that PER and TIM associate rapidly in the cytoplasm following induction of expression and that a lag period is observed before the proteins disassociate and enter the nucleus separately. This was in contrast to the previously held view that the delay in nuclear entry was due to a delay in PER and TIM association. These findings lead to the obvious questions of which protein(s) regulates the delay in the dissociation of PER and TIM and how do these protein(s) know when to initiate PER/TIM dissociation and nuclear entry?

In trying to address this problem the Young lab has begun looking at the sub-cellular localizations of other known PER/TIM interacting proteins over real time. New preliminary data was presented showing that DBT is relocalized from the nucleus to the cytoplasm following induction of PER expression. Co-expression of DBT leads to PER degradation over time, but this is inhibited by the additional expression of TIM.

That evening we attended the conference dinner and, after an enjoyable meal, a group of us took advantage of the resorts facilities to socialise some more.

Overall this was a very stimulating and enjoyable conference. I would like to thank the BSCB for their generosity in giving me an Honor Fell Travel award to enable me to attend this meeting, and the Biochemical Society for additional funding.

Marie Harrisingh, Cellular and Molecular Physiology, Yale School of Medicine marie.harrisingh@yale.edu

Erling Seeberg Symposium on DNA Repair

Bodø and Henningsvær, Lofoten, Norway, 28 May-2 June 2006

I was very excited to go to my first international conference in Norway at time of the year when sun was continuously visible. The meeting was organized to honour Erling Seeberg, pioneer in the field of DNA repair, who passed away in December 2004. The venue of meeting was Lofoten Islands, a spectacular place, ideal for lively and fruitful discussions.

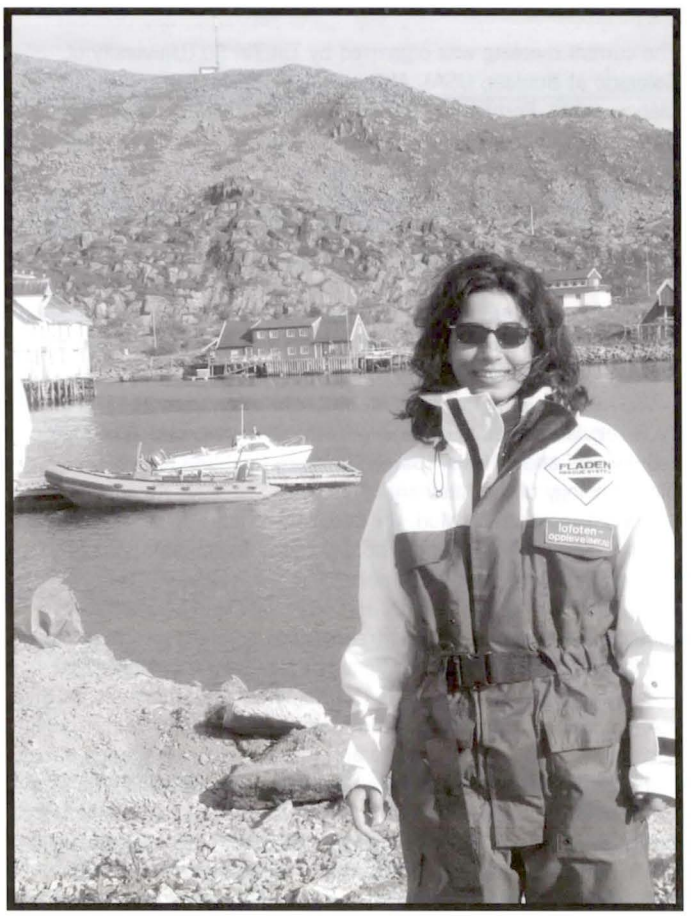
The conference had 150 attendees and was divided into 12 sessions covering 36 plenary talks, 10 short talks and 26 posters. It began at Rica Hotel, Bodø with two keynote addresses by **E.C. Friedberg** (University of Texas) and **Tomas Lindahl** (Clare Hall Laboratories). I particularly enjoyed T. Lindahl's talk in which he described that incorporation of large quantities of anticancer drug 5-fluorouracil (FU) in DNA is cytotoxic and SMUG1 enzyme repairs these mis-incorporated FU by initiating base excision repair. He also demonstrated that mutation in mammalian DNA exonuclease TREX1/Dnase III is involved in human Aicardi-Goutieres Syndrome. The enzyme shares homology with E. coli polymerase III proofreading subunit MutD, a protein that I have worked too in my PhD's research.

The second day session in the morning covered recognition and repair of spontaneous DNA damage. **Bruce Demple** (Harvard School of Public Health) discused the processing of abasic sites by base excision repair. He showed that Ape1 protein is essential and readily incises the lactone lesions but their excision by polymerase leads to a stable DNA–protein cross-link that may necessitate alternative repair pathway.

In the afternoon, we boarded on Nordlys of Hurtigruten, the Norwegian Coastal Voyage, to travel to Svolvær. It was an enjoyable journey of six hours with dinner on board. Throughout the journey, many of us were out on the deck, enjoying the beautiful Norwegian scenery and taking pictures. From Svolvær, buses took us to Heningsvær where the rest of the meeting took place. For most of us the accommodation had been arranged in rorbus, which are refurbished fishermen houses. Very unusual but pleasant place to stay!

On the third day, the morning sessions covered mutagenic DNA processing and DNA repair and aging. The talk of my interest was the one given by **Cynthia McMurray** (Mayo Clinic, Rochester). She described her work on CAG expansions in somatic cells where she discussed that age dependent somatic mutations associated with Huntington's disease occur in the process of removing oxidized base lesions, and is dependent on base excision repair enzyme, OGG1. The afternoon sessions covered DNA damage as therapeutic targets and nucleotide excision repair. **Leona Samson** (MIT, Cambridge) talked about the global genomic responses to alkylation damage. DNA glycosylase (AAG) mutants have increased mutator phenotype and AAG expression induces frameshift mutation in *S. cerevisiae*. In the evening, 26 posters were displayed which were covering work done in organisms like meningococci, *S. pombe* and mice. I enjoyed many of them and had stimulating discussions with the presenting authors. The poster of **Lev Kleppa** (University of Oslo) was very interesting. She showed the work for investigating the role of FEN1. FEN1 knock-out mouse was lethal at blastocyte stage. A FEN1 knock-in variant was developed, having an intact FEN1 gene with amino acid change in conserved endonuclease domain. This mutant is planned to be crossed with Huntington transgenic mice to study CAG repeat stability. **Yilun Liu** (Clare Hall Laboratories) presented the work on the function of Rad51C and XRCC3 in late homologous recombination. Rad51C and XRCC3 deficient cell lines have decreased Holliday junction resolution. The complex interacts with Holliday junctions and influences RecQ helicase.

This was a long day (actually it had been day all the times) as we were taken for midnight sun excursion. The organizers chose a good spot to enjoy the beauty of midnight sun. The sun could be seen going down the horizon on the ocean. At exact midnight, champagne bottles were opened. I don't drink myself but I enjoyed everyone enjoying and drinking. Some clouds did the trick of hiding sun from us but we could see the lights and it was spectacular along the ocean. Everyone had cameras ready to catch the spectacular views. My best shot can be seen below.



Next morning sessions had talks about repair of DNA double strand breaks and repair associated with transcription and chromatin remodelling. In the afternoon, the short talks were delivered and I was privileged to be one of them. I felt pleased with my presentation, received a good feedback and interesting questions were asked. I also had a chance to have fruitful discussions about my work with other scientists during meals and coffee breaks.

Last day's sessions covered base excision repair and translesion synthesis. **Robert Fuchs** (Genome Instability and Carcinogenesis, Marseille) discussed the processing of 3'-end of a blocked primer by specialized DNA polymerases II and V. He showed in the *in vivo* data that both Pol II and Pol V use distinct 3'-ends as their preferred substrates so the two bypass pathways act independent rather than competitive.

In the afternoon, the organizers had planned a number of excursions including deep sea rafting, fishing trip, hiking or a cruise trip. I joined a group of 14 to go for deep sea rafting. It was a great experience and good fun. Sea was quite calm and we sailed through narrow straits between rocks and islets, enjoying the beauty of Lofoten's splendid mountains. Since it was last day, a banquette dinner was arranged. Then everyone enjoyed a late night party with dancing. Next day, we left Heningsvær with fond memories of good scientific discussions and friendships developed during the meeting.

Once again, I am thankful to British Society of Cell Biology for granting me an Honor Fell travel award to attend such a good conference.

Rabaab Zahra, Institute of Cell Biology, University of Edinburgh

Drosophila Cell Division Cycle, 3rd International Workshop

Porto, Portugal. 14–18 June 2006

This series of workshops was initiated in Scotland, in 1995 and has provided an arena to discuss research on cell cycle regulation and cell division in *Drosophila*.

The current meeting was organised by Tin Tin Su (University of Colorado at Boulder, USA), Alvaro Tavares (Instituto Gulbenkian de Ciência (IGC), Portugal) and Claudio Sunkel (Instituto de Biologia Molecular e Celular (IBMC), Portugal) and was attended by 180 participants. The meeting was composed of a large number of talks (78), complemented by 80 posters.

The opening session started with a series of talks on cell cycle regulation and was followed by the Keynote lecture. This talk, entitled "Getting into and out of mitosis", was sponsored by EMBO and given by the Nobel Laureate, **Tim Hunt**. This was a fascinating account on the history of research on the cell cycle. The evening ended with a poster session running till midnight.

We were up early the next day, eager to listen to more talks on cell cycle regulation, cell and tissue growth, stem cells and asymmetric cell division and mitosis. **Sarah Bowman** (Institute for Molecular Biotechnology of the Austrian Academy of Sciences, Austria) presented research on Mud, the *Drosophila* homolog of NuMA and its role in asymmetric cell division. *Drosophila* neuroblasts divide asymmetrically to form a neuroblast and a ganglion mother cell. These divisions have a polarity axis whereby the mitotic spindle contacts and responds to polarized cortical domains ensuring that cell fate determinants are positioned correctly for the subsequent divisions. Mud is a microtubule (MT) binding protein present on the apical cortex in neuroblasts. Mud mutants fail to co-ordinate spindle position with the axis of polarity and result in mis-segregation of cell fate determinants. This causes an excess production of neuroblasts and results in tumour-like over-proliferation in the larval brain. **Terry Orr-Weaver** (MIT Whitehead Institute, USA) described the PAN GU (PNG) complex (png, plu, gnu) which is required to limit DNA replication and promote mitosis during the zygotic cycles in the early embryo. These cycles are rapid, utilizing the maternal stockpile of mRNA and early embryonic cell cycles are regulated post-transcriptionally. If any genes of the PNG complex are removed, DNA replication occurs in the absence of nuclear division resulting in the formation of giant, polyploid nuclei. Her research has shown that the PNG complex is required for the translation of Cyclins A and B and that this regulation may be regulated via their poly(A) tail length.

The third day of the conference started with a session on tumorogenesis followed by chromosome structure and replication. The afternoon sessions included talks on spindle organisation and function, and the spindle checkpoint. **Hiro Ohkura** (University of Edinburgh) gave an impressive talk on acentrosomal spindle formation in female meiosis in which he showed time-lapse movies of the 1st female meiotic spindle forming. He also described a screen to identify spindle mutants in female meiosis and is currently characterising seven that affect spindle morphology and 11 that are defective in chromosome alignment.

That night we had a fantastic meal at Comissao de Viticultura da Regiao dos Vinhos Verdes overlooking the River. The meal afforded an opportunity for PhD students, Post Docs and PIs to casually interact and talk about current and future research plans.

The final day began with a session on centrosomes. **Bodo Lange** (Max Planck Institute for Molecular Genetics, Germany) reported on a screen to identify all of *Drosophila* centrosomal components and to characterise their function. They isolated centrosomes from syncytial embyros and identified their composition by mass spectrometry. More than 200 candidate centrosomal proteins were identified and their cellular functions subsequently assayed by RNAi. Many of these are indeed required for centrosome function and cell cycle progression. In another screen, which aimed to characterise the maturation of mitotic

centrosomes, **Jeroen Dobbelaere** (Gurdon Institute, Cambridge) described the consequences of depleting kinases and phosphatases by RNAi. Thus, they were able to identify genes known to be involved in centrosome replication and maturation as well as new regulators. **Tim Megraw** (University of Texas Southwestern Medical Center, USA) gave a fascinating talk on the domain functions of centrosomin (CNN) in which he showed that the two conserved domains, CNN 1 and CNN 2 have different functions. Mutations in the N-terminal domain, CNN 1, affected the microtubule-organizing centre (MTOC) activity of the centrosome whereas, mutations in the CNN 2 domain resulted in the formation of 'chains' of linked spindles. This was shown to be due to defective actin organisation, which affected the formation of the pseudo-cleavage furrows. Interestingly, CNN still localised to centrosomes in this mutant. A yeast 2-hybrid study showed that the CNN 2 domain interacted with motif 2 interacting protein (MTIP). CNN and MTIP were shown to co-immunoprecipitate and MTIP was localised to pseudo-cleavage furrow and to centrosomes. Thus, the role of CNN in syncytial embryos appears to be determined by two domains: one involved with gamma-tubulin activity and a second which is required to organise the actin cytoskeleton.

This session was followed by talks on kinetochores and chromosome segregation. **Helder Maiato** (IBMC, Portugal) gave an interesting talk on the ultrastructure analysis of the kinetochore. He showed that the number of microtubules that bind to kinetochores in S2R+ cells is 11±2 and that 80% of MT were embedded in the outer layer of the kinetochore and do not penetrate further than 100 nm into the kinetochore. He also described distinct plus end profiles of MTs representing their dynamic state and mentioned that 2/3 of MTs were in a deploymerisation configuration. **Mar Carmena** (University of Edinburgh) spoke about the chromosomal passenger protein, INCENP. This work focused on the function of INCENP in male meiosis and revealed both chromosome segregation and cytokinesis defects. In particular, non-disjunction was increased, under-condensed chromosomes were present and there was a loss of sister chromatid cohesion in meiosis I. This work further showed that INCENP and MEI-S332 co-localise and that MEI-S332 was mislocalised following INCENP RNAi. MEI-S332 was shown to be a substrate of Aurora B, a kinase that is part of the chromosome passenger complex. Overall, these results implicate the chromosome passenger complex in the control of sister-chromatid cohesion in male meiosis.

The afternoon session had talks on cytokinesis and on DNA and checkpoint. **Sebastein Carreno** (Centre de Biologie du Developpement – CNRS, France) spoke about moesin, the only member of the ezrin/radixin/moesin (ERM) family in *Drosophila*. ERM proteins provide a signal-dependent link between cortical actin and membrane proteins and are enriched at the cleavage furrow thus suggesting a role of ERM protein in the connection of cortical actin to the plasma membrane during cytokinesis. He found that following RNAi, binucleate cells formed, indicating cytokinetic failure. Interestingly, the cortex was also affected and abnormal blebbing was observed in 80% of cells. A cytokinesis defect was recapitulated in a mutant of moesin. They also showed that dPlkk was able to phosphorylate moesin, that moesin and Plkk co-localise and that RNAi of Plkk caused the spindle to rotate in the cell. **James Wakefield** (University of Oxford) further spoke on chromosomal passenger proteins, which consist of aurora B, INCENP, survivin and borealin. Borealin is present in embryos, testes, ovaries, and in the mitotic divisions of male meiosis but it is not present during male meiotic divisions. He identified and showed that australin is a testes specific homologue of borealin in *Drosophila*. australin localises to the central spindle in male meiosis but is not found in other tissues. A mutant of australin fails both Meiosis I and II and shows chromosome alignment and spindle defects including the absence of a central spindle. He proposed that the function of australin was to localise the chromosome passenger complex proteins, Aurora B and INCENP, to kinetochores in male meiosis.

Overall, I thoroughly enjoyed this workshop. It was a great environment to be in and I found it extremely stimulating. I have returned home motivated and full of ideas. I would like to express my thanks to the Honor Fell Travel Award from the BSCB for providing the opportunity to attend such a fantastic meeting.

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The 65th Annual Meeting for the Society of Developmental Biology

University of Michigan, 17–21 June

The Society for Developmental Biology was founded in 1939 to promote the field of developmental biology, and has evolved to provide an international forum for research, education and career development in the field.

The 65th Meeting of the Society of Developmental Biology was opened by **Enrico Coen** (John Innes Centre, Norwich, UK). I must start by confessing that this was the only plant development talk that I attended, although plant development was well represented at this meeting. Coen's work provides insights into the developmental processes behind the evolution of flower shape and showed how the evolution of the two types of flower shape, racemes and cymes, is not as separate as it seems. **Gayle Martin** (University of California) then took me back to familiar territory with her work on FGF signaling in vertebrate organogenesis. Her talk focused on how the

levels of FGF signaling are controlled during development, in particular the role of Sprouty genes in tooth morphogenesis.

The meeting proper started on a sunny Sunday morning with two concurrent symposia covering "**Development and Senescence**" and "**Novel genetic mechanisms in development**." The development and senescence symposium, chaired by **Mahendra Rao** (National Institute of Aging, Baltimore) looked at the use of embryonic stem cells and model organisms to determine the molecular mechanisms behind senescence and programmed cell death. Rao's own work demonstrated that embryonic stem cells do age and can be used as a model for cell aging and they have started to identify factors affecting senescence. **Farida Sohrabji** (Texas A and M) went on to show that model organisms can also be used to investigate senescence by presenting her interesting work on reproductive senescence and its effects on the menopausal brain. Her lab has shown that the reproductive senescent rat is a better model for reproductive senescence than the usual surgical menopausal model.

The novel genetic mechanisms in development symposium included talks from **J Yuan** (Cornell) and **HG Simon** (Northwestern). Yuan presented work that demonstrated that Mls-2, an intrinsic factor in cell-fate specification in *C. elegans*, can be regulated at the transcriptional and post-transcriptional level to regulate many aspects of mesodermal development, including cleavage orientation, cell proliferation and cell fate specification. Simon presented work that showed how Tbx5 is differentially regulated during heart development. The data demonstrated that Tbx5 is expressed in the cytoplasm and the nucleus in developing heart cells in the zebrafish. LMP4 acts as a repressor of Tbx5 by binding to it and targeting it to the actin cytoskeleton. In the absence of LMP4, Tbx5 can enter the nucleus and bind to its target gene.

The lunchtime workshop "Cool career choices in Life Sciences" was very useful to me as I am in the second year of my PhD. The definition of cool, by the way is anything outside of academia! This workshop included talks from Scott Gilbert, Pam Hines and Rob Williams, who told us about working in a Liberal Arts college, for Science magazine and in industry respectively.

The closing session was titled "**Genomics and gene networks**" and included a talk by **E Davidson** (Caltech). Davidson presented his network of regulatory pathways involved in embryogenesis in the sea urchin, the genome of which has just been published.

Monday started with seminars on "**Stem cells in animals and plants**" and "**Cell–cell signaling**". In the cell–cell signaling session, **M Levin** (Harvard) talked about his work on bioelectrical controls of morphogenesis. His lab investigates the role of ion flux and endogenous voltage gradients in the tail region of *Xenopus*, and how membrane voltages play a role in tail regeneration. They have identified an ATPase H^+ pump that is induced in existing cells to increase proliferation in the tail region and induce regeneration. Mis-expression of this ATPase has even rescued regeneration in a non-regenerative system. Also in this session, **Robert Ho** (University of Chicago) described his work on the role of caudal transcription factors during segmentation of the nervous system and paraxial mesoderm. His main focus is to answer the question as to why the vertebrate spinal cord is not segmented. His work has shown that caudal transcription factors in the parahox cluster maintain the unsegmented character of the spinal cord. Ho presented his work on the Cdx1a/Cdx4 double knock out mouse, and described how this mouse has lost spinal cord characteristics and has an expansion of the hindbrain region. Further analyses of this mouse have shown that Cdx interacts with FGF and RA, the gradients of which control neuronal differentiation and mesodermal segmentation.

In the stem cell session **Alan Spradling** (Carnegie Institute) presented his work on multiple types of niche control stem cells in *Drosophila* adults. He described the differences between the male and female germline stem cells and their niches, and then described how these two niches aren't as different as previously thought. For instance, he demonstrates that there is another type of stem cell present in the female ovary, namely escort stem cells, and Jak-STAT signaling is required to maintain the niche. He then went on to describe a stem cell population present in the *Drosophila* adult gut. His work demonstrates that this population of mulitpotent stem cells is more reminiscent of neural and epithelial stem cells in that there is no non-dividing partner cell, their differentiation is regulated by Notch signaling and there is a high degree of contact with the basal membrane and muscle layer.

After lunch post-docs were given an opportunity to present their work in the Post-doc symposium. **B Davidson** from the Levine lab (University of California) presented his work on heart development in *Ciona intestinalis*. *C. intestinalis* is closely related to vertebrates and is a useful organism in which to study development due to the low number of cells in the embryo and it has less genetic redundancy. The heart of *C. intestinalis* is single chambered and originates from two cells which have two lineages, either they become heart cells or anterior tail muscle cells. His work shows that Mesp bHLH transcription factors play an important role in the decision between these two fates. He demonstrated that certain cells express dpERK and FoxF and respond to FGFs, and these cells migrate and form heart cells. Ectopic expression of FGF causes all the cells to become heart cells. Sometimes in these embryos two independent heart compartments were formed that initially acted independently of each other but would then synchronise and work together as a functioning heart. This has lead Davidson to build a possible model for the evolution of the heart from a single to a double chambered organ.

The day closed with the second plenary session that focused on **Evolution of development**. **A Burke** (Wesleyan University) talked about patterning domains in the vertebrate mesoderm and the role of *hox* genes.

The last day of the conference started with symposia entitled "**Pathfinding**" and "**Beyond Model Organisms**". The latter symposium introduced us to *Oncopeltus fasciatus*, the Milkweed bug. **K. Panfilio** (Cambridge University, UK) introduced her work on the derived *Hox* gene *Zen*. She showed that *Zen* is required for function of the extra-embryonic serosal membrane of *O. fasciatus*. Loss of Zen results in re-specification of cells from an extra-embryonic fate to an embryonic fate. Her work has shown that by blocking Zen, katatrepsis is blocked. Katatrepsis is the process of repositioning the embryo half way through embryogenesis. When katatrepsis is blocked the embryo is left 'inside out'. Also in this symposium, **R. Freitas** (University of Florida) introduced us to her model organism, the cat shark. Freitas uses the cat shark to study the origin of vertebrate fin development. She is asking whether the mechanisms of fin development evolve from the midline, and she is using the cat shark because it possesses median fins dorsal to the neural tube. She has shown that each fin bud has differential (spatially and temporally) patterning of *Hoxd* genes, and paired and median fins develop using common mechanisms. Freitas has also used the Lamprey, which has no paired fins, to propose that the genetic programme for fin development evolved in the midline.

One of the last speakers of the conference was **Robb Krumlauf** (Stowers Institute, Kansas City) who presented another talk about *Hox* genes. Krumlauf presented work on hindbrain segmentation, and how retinoic acid and repressors of its downstream targets regulate formation of the different rhombomeres. He helped explain how the expression of *Hox* genes is restricted to specific rhombomeres. For instance, in rhombomere 4, a feedback loop enables Hoxb1 to regulate its own expression when RA is no longer present, whereas, expression of Krox20 in rhombomere 3 and 5 represses Hoxb1 expression in spite of the presence of RA. This ensures Hoxb1 expression is restricted to rhombomere 4.

I would like to take this opportunity to thank the BSCB for providing me with funding and therefore enabling me to attend this informative and interesting conference.

Caroline A Pearson, MRC Centre for Development and Biomedical Genetics, University of Sheffield, Sheffield, UK.

Translation UK 2006

Newcastle University, 3–5th July 2006.

Translation UK is an annual meeting, bringing together researchers whose interests range from understanding of ribosome assembly and function, through the multiple mechanisms of regulating translation of both viral and cellular mRNAs, to RNA turnover.

This meeting drew in cell, developmental and structural biologists as well as virologists and geneticists – a potent mix that was reflected in a varied and interesting programme. Many, but not all, of the 115 delegates represented Universities and Research Institutes in the UK. A small number of laboratories from other European countries were represented, including a small Italian contingent that was slightly distracted by proceedings at the Football World Cup.

The meeting was very successful, with excellent platform presentations and a well attended, if somewhat hot and crowded, poster session (perhaps a measure of success for any meeting!). A highlight was the plenary talk delivered by **Virginia (Jenny) Pain**. Jenny has recently retired from the University of Sussex, and her talk, entitled "Understanding the physiological role of the cap binding complex eIF4F – where are we now?" included both a brief introduction highlighting the people and research questions which had influenced her career through to up-to-the-minute results on determining the range of iso-forms of the cap-binding protein eIF4G that are expressed in mammalian cells. Following her talk, Jenny was presented with a print of Inuit art as a small token of appreciation from the research community to which she has contributed so much, and within which she has been an inspirational figure for many years.

The meeting featured two further plenary talks. **Beverley Osborne** (Rennes, France) got proceedings off to a great start with a presentation largely focussed on the key role that regulated adenylation/deadenylation of mRNA plays in gene expression during development. **David Tollervey** (Edinburgh) spoke at the end of the meeting on the critical process of quality control during eukaryotic ribosome biogenesis. Work in David's group has recently revealed that various activities involved in this process are concentrated in a sub-nucleolar region that may represent a 'junk-yard' where faulty pre-ribosomes are disassembled (Dez et al. (2006), EMBO J. 25:1534-46).

It is not possible to mention all of the excellent presentations here. My favourites included a cryo-EM structure of a ribosome stalled at an RNA pseudoknot that causes –1 frame-shifting. The structure, which also revealed the presence of eEF2, suggests that the pseudoknot imposes a physical block to completion of ribosomal translocation on the mRNA, and that the mechanical forces that result from this might be sufficient to drive the frame-shift event (Namy et al. (2006) Nature 441:44-7). This presentation, by **Stephen Moran** (Oxford), won a Biochemical Journal Young Investigator Award. A second award was presented to **Rachel Allison** (Cambridge) whose excellent poster detailed biochemical characterisation of a complex of factors critical for localisation of Vg1 mRNA to the vegetal cortex during *Xenopus* oogenesis. Further highlights included a dissection by **Ian Goodfellow** (London) of atypical translation initiation on calicivirus and norovirus mRNAs that requires interaction between initiation factors and the VPg protein that is covalently coupled to the 5' end of the RNA (Chaudhry et al. (2006) J. Biol. Chem. 281:25315-25) and a presentation by **Ilaria Napoli** (Rome, Italy) of data indicating that the Fragile X Mental Retardation Protein (FMRP), a key regulator of translation in neurons, functions by forming a translation-inhibiting complex with a specialised neuronal eIF4E binding protein and the BC1 RNA. **Mike Clemens** (London) presented data implicating ubiquitination and proteasome-mediated degradation as a major regulatory mechanism for 4E-BP1, processes that lead to more rapid turnover of the protein and consequently increased availability of eIF4E for translation.

An emerging theme over the last few years is that not all factors involved in translation and RNA turnover are distributed randomly in the cytoplasm of the cell. Examples of localisation are the cytoplasmic 'P-bodies', in which untranslated mRNA and many factors involved in RNA degradation are concentrated, and foci of eIF2/eIF2B. P-bodies are closely-related to neuronal RNA granules, and this link was explored in **Jens Hillebrand**'s (Dublin, Ireland) talk. Jens presented data showing that *Drosophila* neuronal RNA granules contain components of the microRNA, nonsense mediated decay and general translational repression pathways as well as mRNA degrading enzymes. Amongst the translational repressors visualised in the granules was the *Drosophila* homologue of FMRP, and co-localisation of FMRP with P-body markers in mammalian neuronal cells was also described by **Ilaria Napoli**. eIF2 is a key factor in translation initiation which binds the small ribosomal subunit in complex with GTP and initiator methionyl-tRNA (the ternary complex), and eIF2B is the nucleotide exchange factor for eIF2. **Susan Campbell** (Manchester) presented a poster revealing that these, but not other initiation factors, localise to cytoplasmic foci in both yeast and human (HeLa) cells. Further, she found that the exchange factor was stably associated with the foci in yeast, but eIF2 rapidly cycled through the foci, suggesting that the latter represent stable sites of nucleotide exchange for eIF2.

A conference dinner at the Centre for Life was squeezed into the packed schedule – and included an extremely energetic and enjoyable Ceilidh, with music provided by local band "The 11th Hour". Finally, many thanks to the companies and organisations (including the BSCB) that supported the meeting through sponsorship, providing prizes, and/or attending the excellent product showcase that ran alongside it. The sponsorship raised provided bursaries for PhD student attendees, significantly reducing the cost of attending the meeting for these delegates.

Jeremy Brown, Institute for Cell and Molecular Biosciences, Newcastle University, Newcastle upon Tyne, NE2 4HH (on behalf of the organising committee: Nicola Grey, Ian Stansfield, Sarah Newbury and Jeremy Brown)

American Society for Cell Biology: Stem Cell Niches

15–18 July 2006, Boston

A balmy Boston played host to the first ASCB summer meeting specifically focused on stem cell niches - the microenvironment essential for the maintenance and proper differentiation of stem cells.

The cellular and chemical factors that make up the stem cell niche were discussed through examples from different model systems including mice, *Drosophila* and nematodes. In addition to talks by well established researchers there were a large number by those who had recently set up their own lab, which made for good discussions and novel angles on old ideas and models.

The hub cells at the tip of the *Drosophila* testis form a stem cell niche. The male germ line stem cells (GSC) cluster around the hub and are in direct contact with it. These hub cells and their interaction with the GSCs was analysed by many including **Yukiko Yamashita** (Stanford University, CA). Yukiko discussed elegant work which highlighted the importance of the orientation of the mitotic spindle of the GSCs relative to the hub. Only a spindle perpendicular to the hub results in the required asymmetrical division whereby one GSC division produces one GSC and one daughter cell, which goes onto form mature spermatids. The correct orientation of the mitotic spindle was achieved through the specific duplication of centrosomes and the movement of the daughter centrosome away from the hub cells. **Leanne Jones** (Salk Institute, CA) discussed how spermatogenesis declines with age. She showed that there was decreased cell-cell adhesion between the hub cells and reduced signalling from the hub cells to the GSCs. The alterations of the stem cell niche may be the cause of the loss of GSCs seen in the testis of older Drosophila.

Work by **Judith Kimble** (University of Wisconsin, WI) highlighted the role of mesenchymal distal tip cells (DTC) in the formation of the GSC niche in the nematode *C. elegans*. Increased Wnt signalling was shown to increase the number of niches while inhibition of Wnt/MAPK signalling results in the loss of the niche and subsequent differentiation of the GSCs.

The stem cell niche is not simply composed of cells but also by environmental factors such as oxygen levels. **Brian Keith** (University of Pennsylvania, PA) showed evidence that Hif2α, an oxygen regulated transcription factor, acts at the Oct-4 promoter in mice. Oct-4 is important for maintaining stem cell pluripotency and is reduced in Hif2α knock-out mice resulting in reduced numbers of primordial germ cells.

While the majority of the talks in the heamatopoetic stem cell (HSC) niche session focussed on different aspects of the microenvironment composing the adult HSC niche, **Hanna Mikkola** (UCLA, CA) discussed the HSC niche of the placenta. Hanna showed through the use of *runx1*$^{acZ/+}$ mice that the labyrinthine vessels of the placenta, where exchange of oxygen and nutrients occurs between mother and foetus, may function as the HSC niche prior to formation of HSC niches within the embryo.

Bringing together researchers with experience of different model systems was beneficial in establishing factors to consider and experimental techniques to use in analysis of novel stem cell niches. I would like to the BSCB for granting me a travel award to attend this meeting.

Karen Groot, Centre of Respiratory Research, University College London, London. k.groot@ucl.ac.uk

FASEB Summer Research Conference: Regulation and Function of Small GTPases

15–20 July 2006 Vermont Academy, Vermont

It was very exciting to go on my first international conference with the help of the British Society of Cell Biology. This was a very dynamic meeting with a lot of discussion taking place and a great atmosphere that encouraged everyone to chat about their work.

This conference was relatively small (about 150 delegates) so only one session ran at a time, which was good as we didn't have to miss out on anything being presented.

On the first night the conference kicked-off with a wine reception and dinner followed by the keynote presentation by **Channing J. Der** entitled "Fifteen years of Ras regulation, signalling and oncogenesis." This was a stimulating way to start the series of excellent talks that went on during the following four days; it was a brilliant overview of this field. He spoke about the diversity in the role played by the different small GTPases and linked some of them to current issues such as their effect in cancer.

On the first full day of the conference we had a '**Subcellular Localization**' session. In this session **Adrienne Cox** from the University of North Carolina at Chapel Hill, USA (who was also one of the organisers for this meeting) gave a great talk on unconventional posttranslational modifications. She described the contribution that regulation by mono-ubiquitination and phosphorylation can have on the localisation of Rho proteins Wrch-1 and RhoE.

Another very interesting talk was given on the second day by **William E. Balch** (The Scripps Research Institute, USA) in the session related to trafficking; he spoke about the ER export COPII vesicles. They use a variety of approaches to study how small GTPases that belong to the Sar1 and Rab families and their effectors can contribute to the self-assembly nanoparticles and cargo selection. At the end of his talk he showed how *in vitro*, it is possible to form larger structures using the COPII subunits in order to transport molecules such as chylomycron.

During the first two afternoons we had a couple of free hours on our timetable. During those hours I enjoyed going for a swim with some other PhD students on a local small lake. This was particularly refreshing as we were going through a heat wave that week; the swim was a great way to relax and get ready for the sessions that took place at the end of the day. The end of the first two afternoons was filled with poster sessions that ran for two hours with about 35 posters in each session. I found these sessions stimulating and I was able to learn a lot from them. Presenting my poster was a great learning experience as this was the first time I was doing such a thing; I had a variety of people interested in my work. I really enjoyed discussing my project with them and it was interesting to find out people's opinions about it. It was also interesting to learn about some other groups' research on the same small GTPase I work on, RhoB.

On the third day the highlight for me was the excellent talk given by **Patricia J. Keely** from the University of Wisconsin, USA. She spoke during the '**Cell Adhesion, Migration and Invasion**' session. Her work concentrated on altering the properties of 3D collagen gels in order to study the consequence the extra-cellular matrix can have on cell behaviour. She showed that in compliant, floating, contractile gels, tubulogenesis takes place in breast tissue; however in fixed dense matrices this is not the case. This is very interesting as collagen-rich dense breast tissue increases the risk of developing breast carcinoma by four to six-fold.

Patricia J. Kelly's talk was followed by **Erik Sahai** from Cancer research UK in London, who also gave an impressive talk. He used multiphoton confocal microscopy to look at migration of metastatic breast cancer cells in mice. The difference between migration of cells *in vitro* and *in vivo* was highlighted throughout the talk as well as the heterogeneity of the mobility of the cells within a tumour.

To finish what had been a quite long day, **Sabrice Guerrier** gave the last talk of this session. It was a very entertaining talk and he managed to keep everyone's attention all the way to the end.

On our fourth afternoon, we had the whole afternoon off as there were no more poster sessions. During this time canoeing on a river near by was organised for us. Many of the delegates (including myself – below) went canoeing, which made the afternoon fun and

gave everyone a chance to relax while enjoying the beautiful scenery that Vermont has to offer in the summer!

Another unique experience was the last dinner of the conference. Apparently it is a tradition that in the last dinner of these meetings lobster is always served! The dinner was great fun as there were many of us eating a lobster for the first time! What an experience!

As a whole the meeting covered many different aspects of this exciting field of research, from structure and localization to cancer and regulation of cell growth. I believe many of us left the conference excited about what is currently being researched and enthusiastic to carry on with our own individual research.

I really enjoyed attending this meeting. It was a real pleasure to hear some of the people who are currently making an impact on the field such as Henry Higgs, Helen Mott and Mohammed Reza Ahmadian.

I thank the BSCB for the Honor Fell Travel Award which gave me the opportunity to attend the conference and learn so much about my field of interest.

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FASEB Summer Research Conference: Ubiquitin & Cellular Regulation

22–27 July 2006 Vermont Academy, Vermont

The Ubiquitin & Cellular Regulation Meeting was organised by Linda Hicke (Northwestern University) and Allan Weissman (NIH/NCI). The meeting was held at the Vermont academy and brought together approximately 200 researchers working on the Ubiquitin-Proteasome System (UPS). It was more like a summer school, including all meals and lodging, this allowed plenty of time for meeting new people, exchanging views and the chance to the seek the insight of other researchers.

The meeting provided the perfect platform for me to learn about the latest research in UPS, and how it is used to control many of the fundamental processes in eukaryotic cells. The UPS is currently one of the hottest topics in biological research with the 2004 Nobel Prize in Chemistry being awarded to Aaron Ciechanover, Avram Hersko and Irwin Rose for the discovery of ubiquitin-proteasome mediated protein degradation. I am very grateful to the British Society of Cell Biology for funding my attendance at this great conference and would like to recommend this meeting, which is held every two years.

The programme of the meeting was designed that only one session ran at a time, ensuring the opportunity to hear every talk. In total during the five days, there were nine oral presentation sessions (five or six invited speakers per session) covering different topics, one workshop, and three poster sessions. I presented my poster entitled "Proteomic Approach to Study Inclusions Seeded by the Overexpression of α-synuclein" in session 1 – Proteasomes and Degradation. It gave me the opportunity to present my current research, discuss with researchers working on similar projects and look around related posters.

The opening night keynote lecture was entitled 'Physiological regulation by proteolysis: the N-end rule pathway and its functions'. It was given by **Alexander Varshavsky** (California Institute of Technology). His group was the first to discover that the N-terminal amino acid residue of a protein determines its *in vivo* half-life; a process now termed the N-end rule. He has since dedicated his scientific career to elucidate the mechanism underlying this process, and described new advances in the understanding of the N-end rule pathway in his talk. One topic he discussed was the N-end rule as a new kind of nitric oxide / oxygen sensor. The *in vivo* oxidation of N-terminal cysteine is essential for its arginylation and is shown to be controlled by nitric oxide and oxygen. UBR1 and UBR2 are the ubiquitin-protein ligases that are used for the N-end rule regulated degradation. Mutations of UBR1 in humans cause Johanson-Blizzard syndrome (JBS). Knockout mice indicate that the functions of UBR1 and UBR2 are significantly different. $UBR1^{-/-}$ mice are viable with pancreatic insufficiency, similarly to JBS. By contrast, $UBR2^{-/-}$ mice are inviable; defects in male meiosis are observed. $UBR1^{-/-}UBR2^{-/-}$ showed impaired neurogenesis and cardiovascular development in mice.

The main programme of the meeting began with a memorial session dedicated to Professor **Cecile Pickart** one of the pioneers of ubiquitin research who sadly died recently. The focus of her laboratory at John Hopkins University has been to investigate the assembly and recognition of polyubiquitin signals, focusing on proteasome proteolysis and DNA-damage tolerance. Six speakers in this session were either from the Pickart laboratory or her collaborators. **Eric Cooper** (Johns Hopkins) described a complex with deubiquitinating activity that is highly specific for K63-linked ubiquitin chains. It includes the lid of the proteasome, the COP9 signalosome, and a novel complex, C6.1A, which includes a poorly characterized JAMM/MPN domain. **Yien Che Tsai** (NCI) showed that the RING finger protein gp78, autocrine motility factor receptor, is critical for ER-associated degradation, and promotes tumour cell invasion in vitro by its pro-metastatic ubiquitin-protein ligase (E3) activity. **Zhijian 'James' Chen** (University of Texas) discussed ubiquitin-mediated

activation of protein kinases in the NF-κB pathway before leaving to also present the following day at the 'Ubiquitin and signalling' session of Bioscience 2006 in Glasgow, UK.

Then, there was a workshop about **Drug Discovery in the Ubiquitin-Proteasome Pathway**. **David Glass** (Novartis Institute for Biomedical Research) described signalling pathways that regulate protein ubiquitylation in skeletal muscle atrophy. Two E3s, MuRF1 and MAFbx, are transcriptionally upregulated. The pathways of regulating these E3s and their potential substrate were discussed. **Teresa Soucy** (Millennium Pharmaceuticals) described their work to inhibit the activity of NAE (Nedd8 activating enzyme) as an oncology target. The workshop was followed by a panel discussion '**Scientific and Career Opportunities in targeting the Ubiquitin System**'. This brainstorming session gave me a few ideas regarding the relative merits of potential careers in academia or industry.

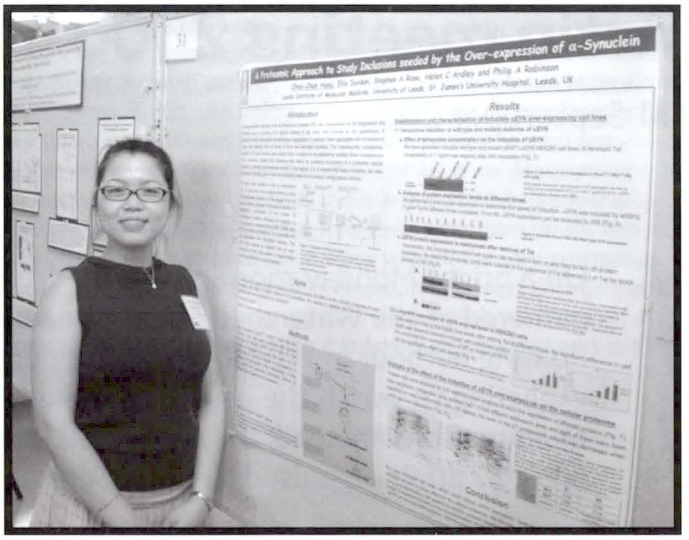
The focus of the second session was ubiquitin and ubiquitin-like (Ubl) binding proteins and their receptors. Ub-binding domains (UBDs) have been identified in many proteins that interact with mono-Ub and/or poly-Ub chains. **Ivan Dikic** (University of Frankfurt) initially described two UBDs referred to UBM and UBZ. However, during the session it became clear that there were additional UBDs discovered only recently. One of these was the reversed UIM domain (called MIU) described by **Simona Polo** (IFOM). Colin Gordon (MRC, Edinburgh) described a UBD in Mud1, which recognizes K48-linked multi-Ub chains.

In the third session, the SUMO and cullin neddylation modification pathways were described by **Chris Lima** (Sloan-Kettering Institute), and Ning Wei (Yale University) and **Brenda Schulman** (St Jude Children's Research Hospital), respectively. **Jon Huibregtse** (University of Texas) employed a proteomic approach to identify target proteins for ISG15 (interferon-inducible ubiquitin-like protein) modification. These included ISG15 E1 (Ube1L), ISG15 E2 (UbcH8) and HECT E3 (Herc5). As our group has previously looked at UbcH8 with respect to UPS mediated protein degradation, I found it particularly interesting that this protein has been found to have a role in ubiquitin-like protein modifications too. Also another thought for identification the targets of ubiquitin-like protein, it can be carried out by a comprehensive UPS microarray as presented in the poster session by Hartmut Scheel (Miltenyi Biotec).

The role of Ub in endocytosis and autophagy was discussed in the fourth session. Mono-Ub is required for the internalisation step of endocytosis, and Ub conjugation is required for autophagy. **Annie Angers** (University of Montreal) described how the E3 Itch was required to ubquitynate its substrate, the endocytic protein, Endophilin. **Yoshinori Ohsumi** (National Institute for Basic Biology) described two ubiquitin-like conjugation systems, Apg12 and Apg8, which regulate autophagosome formation in yeast.

The role of UPS in transcriptional regulation was discussed in session five, beginning with an overview by **William Tansey** (Cold Spring Harbor). Subsequently, the role of histone ubiquitylation and sumoylation in yeast transcriptional regulation was presented by **Shelley Berger** (Wistar institute). **Helle Ulrich** (CRUK) and **Stefan Jentsch** (Max Planck Institute of Biochemistry) discussed how the control of DNA damage tolerance mediated by PCNA (proliferating cell nuclear antigen) was dependent on ubiquitylation and SUMOylation.

The focus of the meeting then switched to the proteasome and deubiquitylating enzymes (DUBs). **Tingting Yao** (Stowers institute) began by describing his work, which indicates that the DUB, Uch37, is a component of the 19S regulatory complex in the cytoplasm, and an ATP-dependent chromatin remodelling complex in the nucleus. Together with Ubp6 (yeast) / Usp14 (human) and Rpn11 (yeast) there are three DUBs now known to associate with the proteasome. **Daniel Finley** (Harvard Medical School) discussed the regulation of proteasome activity by the E3, Hul5, and the DUB, Ubp6. Ubp6 has a conserved capacity to inhibit the proteasome while Hul5 serves to extend the chains of proteasome-bound ubiquitin conjugates. Therefore, the balance of Hul5 and Ubp6 activity might regulate substrate commitment to degradation.



In session 7, the ways in which substrate ubiquitylation can also be used as a signal to regulate other diverse cellular processes were described. **Donald Kirkpatrick** (Harvard Medical School) has developed a novel mass spectrometry based method, termed Ubiquitin-AQUA (Absolute QUAntification) to quantify the different linkage types of a given substrate (cyclin B1) ubiquitylated by APC (anaphase promoting complex). It was interesting to hear that the primary Ub linkage type mediated by the APC is the K63-type. In addition, **Raymond Deshaies** (California Institute of Technology) described another quantitative mass spectrometry method, MudPIT (multidimensional protein identification technology) to identify Ub substrates under different conditions, which permits differential ubiquitylation screens.

Because Parkinson's disease is the focus of my research, the eighth session was of real interest to me and was entitled 'Ubiquitin/Ubl physiology and Disease'. Problems with the ubiquitin system have been associated with many diseases including cancer, autoimmune disease, and neurodegenerative disorders. **Kwon-Yul Ryu** (Standford University) created UbB (ubiquitin B) knockout mice to show polyubiquitin B gene is required for energy homeostasis. Homozygous disruption of the UbB gene in mice has no effect on embryonic development, but the mice are obese. UbB mutants have increased adiposity, hyperleptinemia, reflex hyperphagia defective and reduced caloric efficiency. Therefore, ubiquitin functions not only cell signalling but also energy homeostasis. **Carole LaBonne** (Northwestern University) showed that the control of neural crest regulatory factor function requires both ubiquitynation and sumoylation. The neural crest is a population of precursor cells found only in vertebrate embryos, which share phenotypic and molecular characteristic with invasive tumour cells. She discussed how ubiquitin and SUMO plays a role in the processes of neural crest development.

In the final session (session 9), the way in which protein ubiquitylation is used by the cell to eliminate aberrant proteins, 'protein quality control (PQC) degradation', was described. **Rich Gardner** (Fred Hutchinson Cancer Research Center) recently identified the first PQC degradation pathway in the nucleus, where little if any protein synthesis occurs. Nuclear function requires protein action, but any aberrant proteins could cause damage to cell, therefore, the cell should be able to remove aberrant proteins from the nucleus. Key to PQC degradation in the nucleus is San1 (yeast), a RING-finger E3. ER-quality control system 'ERAD' was discussed by **Christian Hirsch** (Max-Belbruck Center) and Tom Rapoport (Harvard Medical School).

Overall, I felt that the conference was a brilliant experience. During the conference, my discussions with many of the participants helped me to understand more about the broader aspects of the UPS and provided me with practical hints for my own work.

Chao-Chun Hung, University of Leeds.

Actin meeting 2006

11 September 2006

On a bright Monday morning, people from all over the UK gathered at the Watershed, the cinema complex overlooking the floating harbour in Bristol. These 100 punters or so weren't expecting to watch the usual Watershed-fare of independent films or animations. Instead, they attended the first 'Actin Meeting', a one-day affair organised by Giles Cory and Harry Mellor.

The aim of this meeting was to bring together the UK actin community for a day of informal talks and discussion. It was wonderful to see that distance was no obstacle to many 'actin people' and registrants made the effort to come from as far afield as Sheffield, Durham, Dundee or Glasgow.

The main protagonists of this show were the twelve PhD students and postdocs chosen to give 15-minute talks, and the plot was further enriched by the high quality questions and discussion initiated by the numerous cameos sitting in the audience or chairing the sessions. **Batiste Boeda**, from **Michael Way**'s lab (CR-UK, London), kick-started the day in style with his talk on the Ena/Vasp protein Mena and the LIM-domain containing protein, Tes. The data was very clear and convincing, and Batiste's stunning images, enlarged to cover the entire screen, made his presentation all the more compelling. **Ana Cvejic**, from **Paul Martin**'s lab in Bristol, presented her work on the use of zebrafish to study leukocyte recruitment to wounds. Thanks to the transparency of the *Danio rerio* fin, it is possible to visualise in real time leukocytes exiting the bloodstream and migrating to the wound and back. Her presentation, mixing beautiful live imaging, EM, careful quantification and genetics, was a real showcase for this powerful system. Later in the day, **Alice Pollitt**, from **Robert Insall**'s lab (University of Birmingham) introduced another model organism, namely the slime mould *Dictyostelium discoideum*, and described the multiple levels of Scar/Wave regulation in this amoeba.

A couple of talks centred around the use of siRNAs to study gene-function and were sponsored by the company Perbio Dharmacon who has generated a library of siRNAs against proteins known to regulate the actin cytoskeleton. **Francisco Vega**, from **Anne Ridley**'s lab (Ludwig institute, London), used siRNAs against the closely related Rho proteins, RhoA, RhoB and/or RhoC, to unravel their specific roles in tumour cell invasion, whereas **Patricia Kunda**, from **Buzz Baum**'s lab (Ludwig institute, London), screened for regulators of the mitotic actin-based structures and in so doing, uncovered an intriguing role for the ERM protein, moesin.

Actin is involved in a range of cellular processes, from cell migration to gene expression, including trafficking, and presentations were given on all these topics. **Magdalene Michael**, from **Matthias Krause**'s lab (King's College, London) talked about lamellipodin, the Ena/Vasp binding protein, and its role in directional cell migration. **Sebastian Guetller** (CR-UK, London) gave an extremely clear overview of the recent work carried out in **Richard Treisman**'s lab. The Serum Response Factor (SRF) co-activator Mal binds G-actin and links the Rho GTPase/actin polymerisation signalling pathway to SRF activity by redistributing from the cytoplasm to the nucleus in response to Rho-induced actin polymerisation. Sebastian described all the different experimental approaches he took to dissect the complex interplay between actin, Mal and nuclear shuttling. He concluded that interaction with actin in the nucleus regulates Mal activity. Glasgow was represented by **Emma Sandilands** from **Margaret Frame**'s group. Emma addressed the issue of intracellular targeting and activation of the tyrosine kinases c-Src, c-Yes and c-Fyn, highlighting the differences between the three ubiquitously expressed Src kinases.

The program also included studies highlighting actin-dependent processes that are tissue-specific. **Giordano Pula**, from **Alistair Poole**'s lab (University of Bristol), works on human platelets and studies the mechanisms underlying the dramatic morphological changes these undergo when adhering to collagen fibres. Giordano showed that PKCδ affects filopodia formation through its regulation of VASP. **Jaime Millan**, from **Anne Ridley**'s group, talked about the different structures that endothelial adherens junctions can adopt, whereas **Dan Rocca**, from **Jonathan Hanley**'s lab (University of Bristol), presented his work on AMPA receptor internalisation at the postsynaptic membrane. As the meeting's token neurobiologist, Dan put his work in context and set the scene by giving a brief overview

of the cellular processes thought to underlie learning and memory. He then showed that Pick1, a protein known to bind the AMPA receptor GluR2 subunit, was able to regulate the actin nucleation machinery and help drive AMPA receptor internalisation.

The day ended with drinks and the prize giving ceremony. The panel decided to present their 'Actin Oscar' to **Michael Deeks**, who works with **Patrick Hussey** at the University of Durham. Michael gave a masterful overview of the generally overlooked, but none the less fascinating world of the plant actin cytoskeleton. The audience was taken from the unusual lands of *Arabidopsis* trichomes (or branched, single-celled epidermal hairs for the uninitiated) to the wonders of root hair tip growth. As if we hadn't already been spellbound, we were treated to a grand finale of charming movies showing wild-type and mutant *Arabidopsis* plants growing and swirling towards the light – it raised as many laughs as any good Aardman animation would!

This day was a great success. Personally, it gave me the invaluable opportunity to catch up with many "actin people" I'd met at previous conferences. There was unfortunately no time for a poster session but this did not prevent Dana George from putting hers up and discussing her work with an avid group of 10 or so! Many young people want to stay in this field and it is important for such small, focussed meetings to take place in order to help them do so. This meeting would never have taken place without the very generous support of the BSCB, Perbio-Dharmacon, Improvision, Olympus and Zeiss – so thank you ever so much! If there wasn't a UK actin community until now, let's hope it is taking shape. Can't wait until the next actin meeting...

Stéphanie Pellegrin, University of Bristol.

BSCB/RMS Conference: Imaging Membrane Dynamics

14–17 September, Royal Holloway College, UK

This Joint BSCB/RMS conference on imaging membrane dynamics was organised by Rainer Duden (Royal Holloway University of London) and David Stephens (University of Bristol) and held over 4 days at Royal Holloway University of London.

The conference began with a session on **ER export**. In this session **Ben Glick** (Cummings Life Science Centre, Chicago) presented data in which he used GFP labelled cis-golgi and RFP labelled trans-Golgi markers and tracked the progression of cisternae from green to red obtained in *Pichia pastoris*, lending support to a cisternal maturation model of Golgi function.

Then came a horrible shock – tea and coffee were missing! This disappointment was almost too much to bear, however we struggled on and were rewarded by the 2006 Hooke Medal Lecture as delivered by **David Owen** (Cambridge, UK). This lecture was given on structure and function studies on clathrin coat and we learnt that AP2 is more reminiscent of Mr. Tickle than of Mickey Mouse.

There was then a plenary lecture by **Jennifer Lippincott-Schwartz** (Bethesda, USA). This lecture introduced Photo activated Localisation (PALM) microscopy. This technique activates each fluorophore individually, plots its location by finding its centre of fluorescence by considering Gaussian distribution and the bleaches it. This process is repeated for all of the fluorophores in the sample, and the resultant image is close to the quality of an EM image. This lecture also considered the rapid partitioning model of Golgi maturation. A defining feature of the conference for me was the controversy surrounding different models of Golgi formation! The lecture was followed by heated debate on the validity of the rapid partitioning model with several different models being championed within the audience: debate which proved a popular topic of discussion in the evening!

Friday morning began with a **Golgi dynamics** session where **Chris Hawes** (Oxford Brookes) discussed Golgi dynamics in plant cells and focused particularly on the Golgi–ER interaction. He explained the two established paradigms of ER–Golgi interaction. These paradigms suggest that the Golgi either interacts with the ER and moves off again or forms stable interactions with ER exit sites. He then suggested that there was in fact actin based transport across the surface of the ER and that the ER:Golgi exit site may be moving with the surface of the ER.

This morning there was coffee, so all was well with the world! This was followed by a session focussing on "**Endosomal and Lysosomal dynamics**". **Pete Cullen** (Bristol) spoke about sorting nexins (SNX). He demonstrated that the SNX/BAR proteins are coincidence sensors that define microdomains of the endosome which contain PtdIns and are highly curved. He then suggested that the definition of microdomains assists in the assembly of protein complexes that regulate endocytic sorting.

The plenary lecture on Friday once again focused on **Golgi biogenesis**. In this lecture **Graham Warren** (Yale, USA) demonstrated using FRAP that a new Golgi was formed in a sequential fashion from seeds of old Golgi. Moreover he demonstrated that the Golgi body is formed from cis to trans and it is functional when it is only 10% of its normal size.

In the evening we decided to see what the local area had to offer (after all we are from the north so we felt like tourists) so two trains later we were sampling the delights of Windsor. We had a look at the

castle had a little wander round Eton (we probably weren't supposed to do this), toyed with the idea of starting a proletariat uprising and then made our way back.

The following day began with a session on **Cytoskeleton-membrane interactions**. In this session **Viki Allan** (Manchester) showed that cytoplasmic dynein (CD) is required for EGF trafficking. She demonstrated that movement of endosomes from the cell periphery is CD dependent and that CD inhibition also impairs endosomal sorting efficiency and rate of endosomal maturation.

The fifth session was entitled **"Events at the plasma membrane"**. In this session **Ben Nichols** (Cambridge) demonstrated that the relatively slow rate of diffusion in "real" rather than reconstituted membranes was not as had been previously suggested as a result of constraints caused by cortical actin or spectrin but rather that the density of protein in the membrane is the limiting factor on diffusion.

This session was followed by the conference banquet where there was an opportunity to get to know everyone without talking about science. This was good news as my brain had basically melted by this point in proceedings from all of the thinking I'd been doing.

Sunday saw two further sessions, the first of which was entitled **"Organisation and function in the ER"**. Here, **Tim Levine** (UCL) described how the positioning of the cortical ER (cER) regulates cell cycle in budding yeast. Using knockout strains they identified SCS2 (an integral ER membrane protein) as having a role in polarised growth and that the targeting of this protein to the bud tip is mediated by the polarisome. He also showed that SCS2, SHE3 and ICE2 are involved in progression through the G2 checkpoint. He then suggested that the cells monitor the inheritance of cER and that this is a critical regulator of the morphogenesis checkpoint.

The next session was on **"Novel trafficking pathways and machinery"**. In this session **Michael Way** (CRUK, London) discussed how virus particles were trafficked. He demonstrated the role of Src phosphorylation in regulating kinesin-1 loading onto *Vaccinia* virus particles. At the cell periphery, Src is phosphorylated which then causes loss of kinesin from the viral particle. The phosphorylated Src then triggers actin polymerisation, this and actin polymerisation is then used to drive the viral particle away from the cell.

Overall I felt that the conference was a great success with an astonishing diversity and quality of talks. I also enjoyed the opportunity to hear about topics not necessarily related to my subject area which provided several ideas for future experiments. I'd like to thank the BSCB for the Honor Fell award that made it possible for me to attend.

Becky Hughes, Faculty of Life Sciences, University of Manchester.

The BSCB awarded me an Honor Fell Travel Award with which I was able to attend my first conference and present a poster (a prize winning poster!) outlining my research. This represented an excellent opportunity to keep up to date with the developments in my field. The conference was held at The Royal Holloway University of London in the amazing architectural backdrop of the Founders building.

The opening session on **ER export** began with **Catherine Rabouille** (Utrecht, NL) discussing the function of the early secretory pathway and included **Ben Glick**'s (Chicago, USA) account of the Dynamics of the Early Secretory Pathway with his proposal for maturation of the Golgi complex. At this point the meeting took a slight adverse turn as the temperature rose, not due to the presentations, but rather the projector, which overheated and promptly turned itself off!

After a short coffee break and the arrival of a new projector we were all ready to continue with the afternoon's presentations. **David Owen** (Cambridge) presented the 2006 Hooke Medal Lecture after his original attempt had to be aborted due to an untimely and in his own words "messy" illness. His presentation was based on the structural analysis of clathrin coat formation and his model for the adaptor complex, which has recently changed from Mickey Mouse to Mr. Tickle, after it was identified that the α and β ear domains of AP-2 are connected by linker domains. He solved the crystal structure of all four of the subunits of AP-2 and identified residues important for protein function. He also showed that the binding of the adaptor protein, AP-2, to pIP2 and cargo is much stronger when AP-2 is phosphorylated rather than non-phosphorylated.

The final lecture of the day was give by **Jennifer Lippincott-Schwartz** (Bethesda, USA) where she described a technological advance in microscopy. This enabled the precise location of an over-expressed protein to be mapped. The technology, known as Photo Activated Light Microscopy (PALM), works by exciting small numbers of fluorophores followed by bleaching of these before further fluorophores are excited. She then presented her model for Golgi maturation, which lead to a slightly heated discussion as the model differed from that presented by Ben Glick (Chicago, USA).

Day two started with presentations on **Golgi dynamics** and included a talk by **Chris Hawes** (Oxford). COPII-mediated ER export leads to the formation of ER export sites (ERES). These regions of ER are dedicated to sort proteins for export. He showed that, in plant cells, Golgi bodies co-exist with the export site complex as a motile secretory unit associated with the ER and that the Golgi/ER exit site complex may be moving with the surface with the ER.

The final lecture of the day was the plenary lecture given by **Graham Warren** (Yale, USA) and the debate once again became heated as he presented another model for Golgi maturation.

The presentations that were of particular interest to me were on the third day. The first lecture was given by **Tom Kirchhausen** (Boston, USA). His talk began with a movie illustrating how individual clathrin triskelia interact to form a clathrin-coated vesicle. Using live cell imaging he showed recruitment of clathrin and adaptor proteins to the plasma is gradual followed by a catastrophic loss following internalisation which represented uncoating. The presentation was based on uncoating, but before this can occur the clathrin coated pit must undergo scission in order to become a clathrin-coated vesicle. It is known that the process of scission involves the large GTPase dynamin, although the precise mechanism is currently unknown. Kirchhausen screened a large number of compounds and has identified a reversible inhibitor of dynamin, termed dynasore. His preliminary experiments have shown that in the presence of dynasore clathrin coated pits are unable to undergo scission away from the plasma membrane.

The molecular chaperone, Hsc 70, is known to be involved in the uncoating of clathrin-coated vesicles and is guided into position by auxillin. There are 2 isoforms of auxillin, auxilin 1 and auxilin 2/GAK (cyclin G-associated protein kinase). Auxilin 2 differs from auxlin 1 in three ways: firstly it is ubiquitously expressed, whereas auxlin 1 is specific to neuronal cells, secondly it recognises AP-1 as well as AP-2, and thirdly it contains an amino-terminal kinase domain. It was

originally thought that auxillin was able to bind to the clathrin heavy chain at three sites on the same triskelion, however, Kirchhausen showed that although this was true the interpretation was incorrect. Using electron cryomicoscopy he showed that due to the location of auxilin within a clathrin coated vesicle, it binds to three separate heavy chains simultaneously. Hsc 70 is then recruited to drive uncoating. The presentation was concluded using a reverse of his opening video to illustrate uncoating accompanied by Beethoven's Fifth Symphony!

Stuart Mudell (Bristol) then described how different G protein-coupled (GPCR) receptors are sorted into distinct clathrin coated pits. Two purinergic GPCRs, $P2Y_1$ and $P2Y_{12}$ are internalised by clathrin mediated endocytosis, but this internalisation is mutually exclusive. Using live cell imaging he showed that $P2Y_1$ and $P2Y_{12}$ are both internalised by clathrin mediated endocytosis but that this internalisation is distinct until they reach the early endosome. The reason for this is currently unknown but it is thought that it may be due to a modification such as phosphorylation.

One of the major problems in live cell imaging of clathrin-mediated endocytosis has been the inability to pin point the moment of scission. **Christien Merrifield** (Cambridge) described an assay to visualise scission using a pH sensitive fluorophore, super ecliptic phlurin. The fluorophore has been conjugated to transferrin and is used to report the accessibility of clathrin-coated pits to changes in pH of the extracellular fluid. He has been able to detect scission with a resolution of 4 seconds. This system has enabled the recruitment and departure profiles of several endocytic proteins, such as dynamin, coractin, AP-2, and actin to be visualised. This experimental procedure will help to build a more accurate time scaled model for the recruitment and withdrawal of endocytic proteins.

It has long been known that proteins containing internalisation motifs, such as Yxxϕ, in their cytoplasmic tails are concentrated in clathrin-coated pits, due to the recognition of this sorting signal by the μ subunit of AP-2 at the plasma membrane. **Valentina Mercanti** (Geneva, Switzerland) has identified an alternative sorting mechanism. In the absence of a C-terminal endocytic internalisation motif it is the length of the transmembrane domain which can determine if the protein is internalised.

The high quality of the research talks made for a very interesting meeting and led to stimulating discussions both formal and informal. I really enjoyed all the talks, can't wait until the next one!

Matt Foley, University of Sheffield.

The European Society of Cardiology Working Group on Cardiac Cellular Electrophysiology.

22–24 September 2006 in Florence, Italy.

The European Society of Cardiology held its 30th Annual Working Group Meeting on Cardiac Cellular Electrophysiology in September in Florence, Italy.

The meeting was held inside the historic walls of Educatorio Del Fuligno which was originally established as a nunnery in 1416 and then went on to become a college for underprivileged young girls in 1829. Many of the walls were decorated in frescoes dating from as early as 1419 which have been faithfully restored and maintained and served as a wonderful backdrop for the congress venue.

This was a relatively small meeting attended by approximately 200 scientists from many different countries. The aims of the workshop were to present and discuss recent findings on cardiac electrogenesis and arrhythmias, excitation-contraction coupling, cardiac development and growth and to stimulate discussion between renowned scientists and young researchers in the field. The programme featured key review lectures relating to these topics accompanied by a selection of relevant shorter oral communications. I particularly enjoyed the sessions focused on intracellular calcium and excitation-contraction coupling, and arrhythmias and anti-arrhythmic interventions in which a variety of interesting topics were discussed. These included 'Individualised Whole-Heart Models, with Para- and Sub- Cellular Resolution', presented by Dr **Peter Kohl** (University of Oxford), in addition to a discussion of 'Increased Susceptibility to Atrial Tachyarrhythmia in Hearts from Spontaneously Hypertensive Rats' presented by Dr **Andrew James** (University of Bristol).

In addition there were many poster presentations. I presented a poster relating to potassium channels involved in repolarisation of the cardiac action potential and how these become altered in response to voluntary exercise induced cardiac hypertrophy in rats. The poster sessions provided the opportunity for interesting discussions and networking with those researchers working in a similar field or using similar methods to investigate their research area. I am very grateful to the British Society for Cell Biology for providing me with the opportunity to attend and present my work at this conference as a result of their generous award from the Honor Fell Travel Fund.

Rachel Stones, Institute of Membrane and Systems Biology, University of Leeds.

BSCB Spring meeting

29 March – 1 April 2007. Heriot Watt University, Edinburgh

The annual Spring meeting is the main event in the BSCB calendar. Following the focus of the 2006 meeting on Stem Cells, the next Spring meeting will cover a broader range of topics and provides a great opportunity for BSCB members to meet and hear about the latest advances in many areas of cell biology.

The meeting will have a special emphasis on protein modifications and dynamics in health and disease and features an outstanding list of speakers. Several sessions will examine the mechanism and biological roles of post-translational protein modifications. This includes the role of ubiquitin in regulating protein trafficking and cell signalling and the contribution of protein modifiers in disease mechanisms. Two sessions will concentrate on chromosomes and nuclear events, examining protein dynamics, regulation of chromosome structure and gene expression and genetic disorders. Finally, the impact of important new methods for high throughput analyses of proteins, gene expression and cell regulatory mechanisms, using "omics" strategies, will be presented. As usual, poster prizes will be available for PhD students and a special lecture will be presented by the winner of the 2006 Hooke Medal, Tomo Tanaka.

The Spring meeting also hosts the society **Annual General Meeting** which all members are encouraged to attend. This provides the membership with the opportunity to directly impact on the society activities and is also the meeting at which new committee members are voted in.

The 2007 Spring meeting is organized jointly by the **British Society for Cell Biology**, the **British Society for Developmental Biology** and the **Genetics Society** and will be held from March 29th to April 1st at Heriot Watt University in Edinburgh, with en suite accommodation available on campus for participants. The meeting will feature plenary lectures by Barbara Meyer, Elliott Meyerowitz and Matthias Mann with session topics on: "Protein Modification", Ubiquitin, Trafficking and Signalling", "Nuclear Dynamics", Genomes, Chromosomes & Disease"; parallel sessions on "Cell Growth", "Biological Clocks", "Genetics of Behaviour" & "Cell Polarity and Migration" and a concluding joint session on, "Systems Biology, Omics and High Throughput Screens".

BSCB Organizers: Sylvie Urbe and Angus Lamond

For more details and registration information see: http://www.bscb.org

Thursday 29th

Delegate registration	1400-1930	
Committee Meetings		
British Society for Developmental Biology	1300-1600	
British Society for Cell Biology	1500-1800	
Genetics Socety	1500-1800	
Dinner: All delegates	1800-2000	
Plenary Lecture		
Sex and Repression: Barbara Meyer (University of California Berkeley, USA)	2000-2050	James Watt Centre I
Drinks Reception & Trade Exhibition	2100-2230	James Watt Centre II
Students Social Event	2130-2230	Carnegie Room

Friday 30th

Breakfast: All delegates	0700-0830	
Delegate registration	0800-1700	
Plenary Lecture		
Dynamics of cell-cell communication and growth in plant shoot apical meristems: local interactions lead to global patterns Elliot Meyerowitz (California Institute of Technology, USA)	0830-0920	James Watt Centre I

Friday 30th (cont)

Session IA: Protein modification

Chair: Ron Hay
Lecture Theatre IV

	0930-1230
Role of SUMO specific proteases in transcriptional regulation Ron Hay (University of Dundee, UK)	0930-1000
Short Talk selected from Abstracts	1000-1015
Coffee Break	1015-1045
Ubiquitylation and sumoylation in DNA damage bypass Helle Ulrich (Cancer Research UK)	1045-1115
Short Talk selected from Abstracts	1115-1130
Secretion and spreading of Hedgehog family proteins Pascal Therond (CNRS, France)	1130-1200
Histone modifications, nucleosome dynamics and gene expression Jane Mellor (University of Oxford, UK)	1200-1230
Buffet Lunch: Trade Exhibition & Poster set up	1230-1400
'Setting up your Lab' Workshop	1230-1400

Session IB: Cell growth

Chair: Laura Johnston
James Watt Centre I

Pleonastic phosphorylation, substrate recognition and higher order structure of the SCF ubiquitin ligase Mike Tyers (Samuel Lunenfeld Research Institute, Canada)
Short Talk chosen from Abstracts
Coffee Break
Winners and losers during growth of the *Drosophila* wing Laura Johnston (Columbia University, USA)
Short Talk chosen from Abstracts
Growth and Cell Cycle control during *Drosophila* development Bruce Edgar (Fred Hutchinson Cancer Research Centre, USA)
TOR signalling and control of cell growth in yeast and mammals Michael Hall (University of Basel, Switzerland)
James Watt Centre II
Carnegie Room

Session IIA: Ubiquitin, Trafficking and Signalling

Chair: Sylvie Urbé
James Watt Centre I

	1400-1700
De-ubiquitination enzymes in traffic and signalling Sylvie Urbé (University of Liverpool, UK)	1400-1430
Ubiquitin signaling pathways Ivan Dikic (Goethe University Frankfurt, Germany)	1430-1500
Short Talk selected from Abstracts	1500-1515
Short Talk selected from Abstracts	1515-1530
Tea Break	1500-1530
Control of cell surface receptors by ubiquitin: lessons from viruses Paul Lehner (CIMR, UK)	1530-1600
Ubiquitin signaling in the NF-kappaB pathway Zhijian 'James' Chen (University of Texas, USA)	1630-1700
Genetics Society Medal Lecture: The need for winter in the switch to flowering Caroline Dean (John Innes Centre)	1715-1815
Annual General Meetings (BSCB, BSDB, GenSoc)	1815-1845
Dinner	1800-2000
Drinks Reception:	1930-2200
Trade Exhibition & Posters Odd numbers	1930-2045
Even Numbers	2045-2200

Session IIB: Biological Clocks

Chair: Olivier Pourquie
Lecture Theatre IV

Patterning the vertebrate axis by the segmentation clock Olivier Pourquié (HHMI & Stowers Institute for Medical Research, USA)
Ultradian clocks that regulate somite segmentation and other events Ryoichiro Kageyama (Kyoto University, Japan)
Systems biology of circadian rhythms in plants Andrew Millar (University of Edinburgh, UK)
Tea Break
Circadian rythms in mammals Ueli Schibler (University of Geneva, Switzerland
Beddington Medal Lecture (TBA)
James Watt Centre I
In parallel
James Watt Centre II

Saturday 31st

Breakfast: All Delegates 0700-0830

Session IIIA: Nuclear Dynamics

Chair: Bill Earnshaw
James Watt Centre I

	0830-1130
Inactivation of a conditional human kinetochore by targeted epigenetic alteration of centrochromatin Bill Earnshaw (University of Edinburgh, UK)	0830-0900

Session IIIB: Genetics of behaviour

Chair: Michael Bate
Lecture Theatre IV

Specifying the machinery for movement in *Drosophila* Michael Bate (University of Cambridge, UK)

Spatial organization of gene expression Wouter de Laat (Erasmus Medical School, The Netherlands)	0900-0930	**Brain asymmetry: from genes to circuitry and behaviour** Steve Wilson (University College London, UK)
Genome-wide microscopy-based RNAi screening to identify mitotic genes in human cells Jan Ellenberg (EMBL, Germany)	0930-1000	**Genetic dissection of zebrafish visual perception and behavior** Herwig Baier (University of California San Francisco, USA)
Coffee break	1000-1030	Coffee Break
Protein Dynamics in the Cell Nucleus Angus Lamond (Univeristy of Dundee, UK)	1030-1100	**Genetic and neuroimaging studies of sensory circuits in *C. elegans*** Bill Schafer (LMB, UK)
Short Talk selected from Abstracts	1100-1115	Promega Lecture
Short Talk selected from Abstracts	1115-1130	
Hooke Medal Lecture **Kinetochore capture and bi-orientation of the mitotic spindle** Tomo Tanaka (University of Dundee, UK)	1130-1230	James Watt Centre I
Buffet Lunch: Posters & Trade Exhibition	1230-1400	James Watt Centre II
Session IVA: Genomes, Chr and disease Chair: Bob Goldman Lecture Theatre IV	1400-1700	**Session IVB: Cell polarity and migration** Chair: Daniel St Johnston James Watt Centre I
Altered nuclear architecture in laminopathies Bob Goldman (Northwestern University, USA)	1400-1430	**Polarising the Anterior-Posterior axis in *Drosophila*** Daniel St Johnston (Wellcome Trust/Cancer Research UK Gurdon Institute)
Short Talk selected from Abstracts	1430-1445	Short Talk selected from Abstracts
New mechanisms of human genetic disease Doug Higgs (MRC Molecular Haematology Unit, UK)	1445-1515	**Regulation of cell surface mechanics underlying tissue morphogenesis** Thomas Lecuit (IBDML, France)
Short Talk selected from Abstracts	1515-1530	Short Talk selected from Abstracts
Tea Break	1530-1600	Tea Break
The Epigenetic Disease Mechanism of FSHD Silvère van der Maarel (Leiden University Medical Centrer, The Netherlands)	1600-1630	**Genetic regulation of gastrulation movements in zebrafish** Liliana Solnica-Krezel (Vanderbilt University, USA)
Dynamic Nuclear Reorganisation in Neurodegenerative Disease Stephen Davies (University College London, UK)	1630-1700	**Making a polar cell with a conserved tool box** Liam Dolan (John Innes Centre Norwich, UK)
Waddington Medal Lecture	1715-1815	James Watt Centre I
Conference Dinner:	1930-Late	Venue TBC

Sunday 1st

Breakfast:All Delegates	0800-0900	
Plenary Lecture **Quantitative proteomics and phosphoproteomics for systems biology** Matthias Mann (Max-Planck Institute for Biochemistry, Germany)	0900-0950	James Watt Centre I
Session VA: Systems Biology, "Omics" And High Throughput Screens: The Future? Chair: Matthias Mann	1000-1300	James Watt Centre I
Retinal growth and regeneration: balance between proliferation and differentiation Jochen Wittbrodt (EMBL, Germany)	1000-1030	
Short Talk selected from Abstracts	1030-1045	
Coffee Break	1045-1115	
A human protein atlas – profiling the human proteome Matthias Uhlen (Royal Institute of Technology, Sweden)	1115-1145	
Short Talk selected from Abstracts	1145-1200	
How to deal with the complexity of a "simple" eukaryotic cell Steve Oliver (University of Manchester, UK)	1200-1230	
A systems approach to virus entry and endocytosis in mammalian cells Lucas Pelkmans (ETH Institute of Molecular Systems Biology, Switzerland)	1230-1300	
Buffet Lunch:	1300-1430	James Watt Centre II

Other forthcoming meetings

2007

Calcium signals and developmental patterning
19– 20 February 2007, The Royal Society, London, UK
www.royalsoc.ac.uk/event.asp?id=4159&month=2,2007

Imaging Life and Death
March 2007, Cambridge, UK
www.rms.org.uk/event_lifedeath.shtml

30th Annual Meeting of the German Society for Cell Biology (DGZ)
14–17 March 2007, Frankfurt am Main, Germany
www.zellbiologie.de

BSCB/BSDB/Genetic Society Joint Spring Meeting
29 March – 1 April 2007, Heriot Watt University
Organisers: Sylvie Urbe and Angus Lamond
(see page 32)
www.bscb.org

7th International ELMI meeting on Advanced Light Microscopy
17 April 2007, National Science Learning Centre, York, UK
www.rms.org.uk/event_elmi07.shtml

16th International Congress of Cytology
13–17 May 2007, Vancouver, BC, CANADA
www.venuewest.com/2007/icc/

Pan-American Society of Developmental Biologists Congress
A joint meeting between the Latin American Society for Developmental Biology and the International
Society for Cell Biology.
June 2007. Cancun, Mexico
www.niob.knaw.nl/isdb/meetings.htm

American Society for Cell Biology and European Cytoskeletal Forum joint meeting
27–30 June 2007 Dijon, France
www.cytoskeletonforum.org/ or
www.ascb.org

32nd FEBS Congress "Molecular Machines"
7–12 July 2007
Vienna, Austria
www.univie.ac.at/febs2007/

Regulation of protein function by SUMO modification
25–27 June 2007, Manchester, UK
www.biochemistry.org/meetings/programme.cfm?Meeting_No=SA073

LifeSciences 2007
A joint meeting of the Biochemical Society the British Pharmacological Society, & the Physiological Society
8–12 July 2007, Glasgow
www.lifesciences2007.org

European Life Scientist Organization Annual Meeting.
1–4 September 2007, Dresden, Germany
www.elso.org

BSCB Autumn Meeting, Abercrombie Meeting on Cell Motility
9–12 September 2007, St Catherine's College, Oxford
Organisers: Anne Ridley, Michelle Peckham and Peter Clark
www.bscb.org

64th Harden Conference – Mitochondrial biochemistry, physiology and disease
14–18 September 2007
St Martin's College, Ambleside
www.biochemistry.org/meetings/programme.cfm?Meeting_No=64HDN

ASCB Annual Meeting
1–5 December 2007, Washington DC, USA
www.ascb.org

2008

33rd Congress of the Federation of European Biochemical Societies and 11th Conference of the International Union of Biochemistry and Molecular Biology.
Biochemistry of Cell Regulation.
28 June – 3 July 2008. Athens, Greece
www.febs-iubmb-2008.org

ASCB Annual Meeting
13–17 December 2008, San Francisco, USA
www.ascb.org

Application to join the BSCB

Please complete and return along with a signed Direct Debit mandate to:
Margaret Clements, c/o The Company of Biologists Ltd., 140 Cowley Road, Cambridge CB4 0DL, UK

Name: _____ ☐ Mr ☐ Ms ☐ Mrs ☐ Dr ☐ Prof

Position: _____ ☐ Male ☐ Female

Academic qualifications: _____

Email: _____

Telephone: _____

Fax: _____

Address: _____

_____

_____ Postcode: _____

Research interests: _____

_____

Membership of other societies: _____

_____

BSCB Member	Proposer	Seconder
Name:	_____	_____
Membership Number:	_____	_____
Signature:	_____	_____

Applicants without proposers should enclose a brief CV

The society has a searchable database of its members on the BSCB web page. This list is not sold or distributed in any other way.

Your details will be included only if you tick this box ☐

Applicant's signature: _____ Date: _____

British Society for Cell Biology

DIRECT Debit

Please complete parts 1, 2, 3, 4 and 6 to instruct your branch to make payments directly from your account. Then return the form to: British Society for Cell Biology, c/o Margaret Clements, The Company of Biologists Ltd., 140 Cowley Road, Cambridge CB4 0DL, UK

To The Manager,

Originator's identification number | 9 | 4 | 1 | 4 | 5 | 1 |

Bank/Building Society

Address

Postcode

FOR BSCB USE ONLY
This is not part of the instruction to your bank/building society
5. Originator's reference number (for office use only)

BRITSO | | | | |

1. Please write the full postal address of your branch in the box above.

2. Name of account holder

3. Account number | | | | | | | | |

4. Sort code | | | — | | | — | | |

Banks/Building Societies may refuse to accept instructions to pay direct debits from some types of account.

6. Instructions to the Bank or Building Society

Please pay the British Society for Cell Biology Direct Debits from the account detailed on this Instruction subject to the safeguards assured by the Direct Debit Guarantee.

Signature

Date

This guarantee should be detached and retained by the payee

THE DIRECT DEBIT GUARANTEE

- This guarantee is offered by all Banks and Building Societies that take part in the Direct Debit scheme. The efficiency and security of the scheme is monitored and protected by your own Bank or Building Society.
- If the amounts to be paid or the payment dates change, the BSCB will notify at least 14 days in advance of your account being debited or as otherwise agreed.
- If an error is made by the BSCB or by your Bank/Building Society, you are guaranteed a full and immediate refund from your branch of the amount paid.
- You can cancel a Direct Debit at any time, by writing to your Bank or Building Society. Please also send a copy of the letter to the BSCB.

Honor Fell Travel Awards

Jointly funded by the BSCB and the Company of Biologists

Honor Fell Travel awards are made to provide financial support for BSCB members, usually at the beginning of their research careers, to attend meetings. Applications are considered for any meeting relevant to cell biology. The amount of the award depends on the location of the meeting. Awards will be up to £250 for UK meetings (except for BSCB Spring or Autumn Meetings for which the registration and accommodation costs will be made, even in excess of £250), up to £300 for European meetings and up to £400 for meetings in the rest of the world. Awards are made throughout the year.
The following rules apply:

- Awards are not normally made to applicants over 35 years of age.
- Applicants must have been a BSCB member for at least a year or be in the first year of their PhD.
- No applicant will receive more than one award per year or three in toto.
- The applicant must contribute a poster or a talk on/at which they should acknowledge BSCB support.

No single lab will receive more than £1000 per year.

Applications should be sent to:
Jordan Raff, The Wellcome Trust/CR UK Gurdon Institute, Tennis Court Road, Cambridge CB2 1QN

All applications must contain the following:

- **the completed and signed application form (below)**
- **a copy of the abstract being presented**
- **proof of registration and travel costs**
- **a copy of the completed meeting registration form.**

First-year PhD students should send a copy of their BSCB membership application.

Application for an Honor Fell travel award

Full name and Mailing address:

_____

_____

_____

_____

Email address: _____

Age: _____

BSCB Membership number: _____

☐ I have been a BSCB member for more than one year

The years of previous Honor Fell Travel Awards:

_____

Degrees with dates: _____

_____

Present Position: _____

Number of Meetings attended last year:

_____

Meeting for which application is made (title, place and date):

_____

_____

_____

Expenses

Travel: _____

Registration: _____

☐ I have included proof of registration and travel costs

Have you submitted any other applications for financial support?

☐ YES ☐ NO

If YES give details including, source and whether these monies are known to be forthcoming.

_____

_____

Supporting statement by Head of Laboratory

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.

☐ My laboratory has not received more than £1000 in Honor Fell Travel Awards this calendar year.

Signature: _____

Name: _____

Applicant

Signature: _____

Name: _____

Undergraduate bursaries to attend the Spring Meeting

Administered through the Honor Fell Travel Award Scheme

Jointly funded by the BSCB and the Company of Biologists

Undergraduate Bursaries are made to provide financial support for undergraduates currently studying cell biology or a related degree subject to attend the BSCB Spring Meeting. The award will cover the registration and accommodation costs of attendance.

Travel costs are expected to be met by the University that the undergraduate attends.

The following rules apply:

- Awards are made to undergraduates in their final year of study.
- Applicants must be studying for a Cell Biology or related degree.
- Applications must be accompanied by a half page justification from the student and by a supporting statement from the supervisor of studies or course organiser.

Applications should be sent to: Jordan Raff, Wellcome/Cancer Research UK Institute, University of Cambridge, Tennis Court Road, Cambridge CB2 1QR.

All applications must contain:

- the completed and signed application form (below)
- statements from both the student and course organiser.

- The statement from the student should include details on why they wish to attend,what they hope to gain and also aspects of cell biology that to date they have found interesting.
- The statement from the course co-ordinator should indicate the course being undertaken by the student and reflect the calibre of the student, their enthusiasm for the subject and why they believe the student will benefit from the experience of attending the meeting.

Application for an undergraduate Honor Fell travel award

Full name and Work address

(write clearly – this will be used as a return label)

_____

_____

_____

_____

_____

E-mail address: _____

Age: _____

Institution attended: _____

Degree course: _____

Main cell biological interests: _____

_____

_____

Supporting statement by Head of Department or Course Co-ordinator: This applicant requires these funds and is worthy of support.The University/Department also agrees to pay the travel costs for the named undergraduate to attend the meeting.

Signature: _____

Name: _____

Applicant's signature: _____

Name: _____

DEADLINE FOR APPLICATIONS: 31 January 2007

The British Society For Cell Biology Financial Statements for the year ended 31 December 2005

Registered Charity number: 265816

Officers And Advisers

Executive Committee Members:
Prof. C.M. Isacke (President)
Prof. L. Smythe (Secretary)
Prof. M. Marsh (Treasurer)
Dr. K. Hodivala-Dilke (Meetings Secretary)
Dr. J. Pines (Membership Secretary)
Dr. D. Stephens (Newsletter Editor)
Dr. A. Ng (Website Co-ordinator)
Dr. V. Braga
Prof. A.J. Harwood
Dr. M. Heck
Prof. I.M. Hagan
Dr. S. Nurrish
Dr. R. Quinlan
Dr. J. Raff
Dr M. Way
Dr. S. Urbe
Dr. S. Munro

Bankers:
HSBC Bank plc
Arlington Business Centre
Millshaw Park Lane
Leeds LS11 0PA

National Savings
Glasgow G58 1SB

Independent Examiner:
C M Thompson FCA
C/o Jacob Cavenagh & Skeet
Acorn House, 2 Greenhill Crescent
Watford, Herts WD18 8AH

Contact Address:
c/o Prof. M. Marsh
MRC LMCB
University College London
Gower Street, London WC1E 6BT

Executive Committee's Report for the year ended 31 December 2005

The Executive Committee (who are the trustees of the Society for the purposes of charity law) have pleasure in presenting their report and the independently examined accounts of the Society for the year ended 31 December 2005. These accounts have been prepared in accordance with the Charities Act 1993, the Statement of Recommended Practice 'Accounting & Reporting by Charities' (SORP 2000); and the constitution of the Society.

Officers and Executive Committee
Under the constitution of the Society the Officers of the Society are a President, a Secretary, a Treasurer, a Meetings Convenor, a Membership Secretary, a Newsletter Editor and a Website Co-ordinator. There is also an Executive Committee of the Society consisting of the Officers and twelve other elected members.

The Executive Committee is elected at the Annual General Meeting, with the Officers being elected by the Executive Committee and the President being nominated by the Executive Committee.

The Executive Committee prepares the Agenda for meetings of the Society, and between meetings acts as necessary on behalf of the Society, reporting on any such actions to the next meeting of the Society.

The individuals who served as Officers and Executive Committee members during the year, and since the year-end, were as follows:
Dr V. Braga
Prof. G. Griffiths (resigned 31/03/06)
Dr M. Heck
Dr. K. Hodivala-Dilke
Prof. A. Lamond (resigned 31/03/06)
Prof. P. Luzio (resigned 31/03/06)
Dr. J. Marsh (resigned 31/03/06)
Prof. M. Marsh
Dr. A. Ng
Dr. S. Nurrish
Dr. J. Pines
Dr. R. Quinlan
Dr. J. Raff
Prof. E. Smythe
Dr D. Stephens
Dr. M. Way
Dr. F. Watt (resigned 31/03/06)
Prof. M. J. Whitaker (resigned 31/03/06)
Dr. S. Urbe
Dr. S. Munro
Prof. C.M. Isacke (appointed 01/04/06)
Prof. A.J. Harwood (appointed 01/04/06)
Prof. I.M. Hagan (appointed 01/04/06)
The Executive Committee members in office at the date of this report are detailed above

Status and constitution
The Society was established under a constitution executed in 1965, and amended in 2002. The Society is a registered charity, number 265816.

Objects
The objects of the Society are to promote the advance of research in relation to all branches of cell biology and to encourage the interchange of information. The Society generally aims to fulfil these objects by organising and sponsoring two meetings each year on topics relevant to cell biology; issuing a twice yearly newsletter; and maintaining a website (www.bscb.org).

Review of Activities
At the beginning of April the Society held its Annual Spring Meeting at the University of Warwick together with the British Society for Developmental Biology (BSDB). The BSCB section of the meeting was organised by Dr. Kairbaan Hodivala-Dilke and Dr. Jordan Raff and featured Professor Cori Barmann as the plenary Borden lecturer. Altogether the meeting attracted over 400 delegates.

In September Michael Way organised the Societies' annual 'Autumn Workshop on Signalling and Cytoskeletal Dynamics during Infection' at Herriot Watt University, Edinburgh. Despite an excellent scientific programme the meeting was not well supported and the Society incurred some loss.

The Society continued to provide travel funds through The Honor Fell Travel Award scheme. In total 85 grants were made in 2005, which included bursaries for two scientists from Eastern Europe and two undergraduate students to attend the Spring Meeting. The other awards, which in total cost £27,000, allowed graduate student and postdoctoral members of the Society to attend national and international meetings. The meetings and travel grants were supported by a major award from the Company of Biologists.

At the end of the year the Society initiated a major overhaul of its web site. This should be finished by mid-2006 and, in addition to easier access to information, the new site will enable Society membership to be managed on-line.

Further details of the Society's activities throughout the year and meeting reports can be found in the half yearly magazine, available on the Society's website (www.bscb.org).

The financial results of the Society are set out below.

Reserves
The Executive Committee regularly reviews

the reserves of the charity to ensure that sufficient liquid funds are available for the Society to meet its ongoing obligations. The reserves throughout the period have been adequate to fulfil this objective.

Investment Policy
The Executive Committee's policy at present is to invest in low-risk and reasonably liquid assets, so that funds are available to meet any unforeseen needs that arise as a consequence of meeting activities.

Risk assessment
The major risks to which the Society is exposed, as identified by the Executive Committee, have been reviewed in the year and systems are in place to mitigate these.

Executive Committee's Responsibilities
The Executive Committee is responsible for preparing the Annual Report and the financial statements in accordance with applicable law and United Kingdom Generally Accepted Accounting Practice.

Charity law requires the Executive Committee to prepare financial statements for each financial year which give a true and fair view of the state of affairs of the Society and of the surplus or deficit for that period. In preparing those financial statements, the Executive Committee are required to:
- select suitable accounting policies and then apply them consistently;
- make judgements and estimates that are reasonable and prudent;
- prepare the financial statements on the going concern basis unless it is inappropriate to assume that the Charity will continue in existence.

The Executive Committee has overall responsibility for ensuring that the Society has an appropriate system of controls, financial and otherwise. It is also responsible for keeping proper accounting records which disclose with reasonable accuracy at any time the financial position of the Society, and for safeguarding the assets of the Society and hence for taking reasonable steps for the prevention and detection of fraud and other irregularities.

Governance and internal control
The Executive Committee is also responsible for implementing systems of internal control which provide reasonable assurance that:
- the Society is operating efficiently and effectively;
- its assets are safeguarded against unauthorised use or disposition;
- proper records are maintained and financial information used within the charity or for publication is reliable;
- the Society complies with relevant laws and regulations.

The systems of internal control are designed to provide reasonable, but not absolute, assurance against material misstatement or loss. They include:
- delegation of authority and segregation of duties;
- identification and management of risks.

Trustee: M.C.P Marsh
Date: 17th Nov. 2006

Independent Examiner's Report to the Trustees of the British Society For Cell Biology

I report on the accounts of the Trust for the year ended 31 December 2005, which comprise the Statement of Financial Activities, the Balance Sheet and the related notes.

Respective responsibilities of trustees and examiner
The charity's trustees are responsible for the preparation of the accounts. The charity's trustees consider that an audit is not required for the year (under section 43(2) of the Charities Act 1993 (the 1993 Act)) and that an independent examination is needed.

It is my responsibility to state, on the basis of procedures specified in the General Directions given by the Charities Commissioners under section 43(7)(b) of the 1993 Act, whether particular matters have come to my attention.

Basis of independent examiner's report
My examination was carried out in accordance with the General Directions given by the Charity Commissioners. An examination includes a review of the accounting records kept by the charity and a comparison of the accounts presented with those records. It also includes consideration of any unusual items or disclosures in the accounts, and the seeking of explanations from you as trustees concerning any such matters. The procedures undertaken do not provide all the evidence that would be required in an audit and, consequently, I do not express an audit opinion on the view given by the accounts.

Independent examiner's statement
In connection with my examination, no matter has come to my attention:

1. which gives me reasonable cause to believe that, in any material respect, the requirements:
- to keep accounting records in accordance with section 41 of the Act; and
- to prepare accounts which accord with the accounting records and to comply with the accounting requirements of the Act

have not been met; or

2 . to which, in my opinion, attention should be drawn in order to enable a proper understanding of the accounts to be reached.

C M Thompson FCA
Chartered Accountant
Acorn House, 2 Greenhill Crescent
Watford, Hertfordshire WD18 8AH
Date: 20th Nov. 2006

Notes to the accounts (see page 42)

1. Accounting Policies
a) Basis of accounting
The financial statements are prepared under the historical cost convention and in accordance with applicable Accounting Standards, the Statement of Recommended Practice 'Accounting and Reporting by Charities' (issued October 2000) and the Charities Act 1993.

The Society has taken advantage of the exemption in Financial Reporting Standard 1 from producing a cash flow statement, on the grounds that it would have been a small company had it been a company incorporated under companies' legislation.

b) Funds
General unrestricted funds represent the funds of the Society that are not subject to any restrictions regarding their use and are available for application on the general purposes of the Society.

Restricted funds are those subject to specific trusts, which may be declared by the donor or with their authority. The restricted funds of the Society are restricted income funds given for specific purposes which are expendable at the discretion of the Executive Committee in accordance with particular activities of the Society.

c) Incoming Resources
Donations and similar incoming resources are accounted for when receivable. Subscriptions and mailing list sales represent amounts receivable during the year. Meetings income is recognised in the period when the meeting takes place and investment income (which represents interest on National Savings deposits and bank accounts) are the amounts receivable for the year.

d) Resources Expended
Expenditure represents purchases and expenses incurred during the year including irrecoverable VAT. All expenditure is recognised on an accruals basis, with advance expenditure for meetings being deferred until the period when the meeting takes place.

Transactions in foreign currency are translated at the rate ruling on the date of the transaction. Balances denominated in foreign currencies are retranslated at the year-end, with the gain or loss on retranslation going through the Statement of Financial Activities for the year.

Management and administration comprises all the expenditure incurred in connection with the management and administration of the charity.

Grants and awards are accounted for when paid over, or when awarded, if that award creates a binding obligation on the charity.

e) Joint arrangement
Where applicable the share of income and expenditure from joint arrangements to put on meetings in the year is accounted for under the appropriate heading in the Statement of Financial Activities (see note 4).

2. Grants received and made
A grant of £45,000 was received from The Company of Biologists during the year. £20,000 of this was restricted for funding the Honor Fell Awards and £25,000 was unrestricted. Honor Fell travel awards represent grants made to members to enable them to travel to meetings of the Society. During the year grants totalling £28,566 were made to 85 individuals (2004: £37,459 to 93 individuals). No individual grants or travel awards exceeded £1,000 in the year.

3. Executive Committee members and employees
No Executive Committee member or any person connected with them received, or is due to receive, any remuneration for the year directly or indirectly from the Society's funds.

Eleven (2004: twelve) Executive Committee members received a total of £2,418 (2004: £2,673) in respect of reimbursed travel expenses during the year. The Society has no employees.

Charity funds were used to purchase trustee indemnity insurance during the year (£919).

4. Joint arrangements

During the year the Society entered into a joint arrangement with the British Society for Developmental Biology in respect of the meeting in Warwick.

Statement of Financial Activities for the year to 31 December 2005

Income and Expenditure		**2005**		**2004**
	Unrestricted	*Restricted*	*Total*	*Total*
	£	£	£	£
Incoming Resources				
Donations, legacies and similar incoming resources[2]	25,000	20,000	45,000	45,000
Activities in furtherance of the charity's objects:				
Meetings	92,040	–	92,040	135,291
Subscriptions	20,697	–	20,697	25,348
Investment income	6,102	–	6,102	4,925
Other income	–	–	–	31
Total incoming resources	**143,839**	**20,000**	**163,839**	**210,595**
Resources Expended				
Charitable Expenditure				
Grants payable in furtherance of the charity's objects:				
Honor Fell travel awards[2]	8,566	20,000	28,566	37,459
Costs of activities in furtherance of the charity's objects:				
Costs of meetings 154,507		141,407	–	141,407
Newsletter costs	6,236	–	6,236	12,262
Website expenses 1,945		7,175	–	7,175
Management and administration[5]	8,395	–	8,395	11,308
Total resources expended	**171,779**	**20,000**	**191,779**	**217,481**
Net movement in funds for the year	(27,940)	–	(27,940)	(6,886)
Funds brought forward at 1 January	176,058	–	176,058	182,944
Funds carried forward at 31 December	**148,118**	**–**	**148,118**	**176,058**

Balance Sheet as at 31 December 2005

	£	**2005** £	£	**2004** £
Current Assets				
Debtors:				
Prepayments and accrued income		22,029		2,916
Cash at bank and in hand:				
National Savings Investment Account		63,125		60,668
HSBC Bank Accounts		82,541		124,382
		167,695		**187,966**
Less: Creditors falling due within one year				
Income received in advance	8,745		1,000	
Creditors and accruals	10,832		10,908	
		19,577		**11,908**
Net Assets		**148,118**		**176,058**
Funds				
Unrestricted funds		148,118		176,058
		148,118		**176,058**

5. Management and Administration expenses

		2005		**2004**
	Unrestricted	*Restricted*	*Total*	*Total*
	£	£	£	£
Secretarial	15	–	15	706
Executive Committee expenses	3,449	–	3,449	3,316
Subscriptions	1,201	–	1,201	2,292
Bank charges	559	–	559	678
Exchange (gains)/losses	(539)	–	(539)	922
New Hooke Medals	–	–	–	838
Insurance	919	–	919	–
Auditors' remuneration: Audit	–	–	–	1,275
Examiners' Examiner's fee:				
Accountancy	2,791	–	2,791	1,281
	8,395	**–**	**8,395**	**11,308**

6. Restricted funds

	Balance b/f	*Incoming resources*	*Resources expended*	*Balance c/f*
	£	£	£	£
Honor Fell Fund	–	20,000	20,000	–
	–	**20,000**	**20,000**	**–**

Approved by the Trustees and signed on their behalf by:

M.C.P Marsh 17th Nov. 2006
S.J. Nurrish 17th Nov. 2006

Comittee Members 2006

President
Professor Clare Isacke
Breakthrough Breast Cancer Research Centre
Institute of Cancer Research
237 Fulham Road
London SW3 6JB
Tel: +44 (0) 20 7153 5510
Fax: +44 (0) 20 7153 5340
E mail: clare.isacke@icr.ac.uk
Appointed 2006; retires 2010

Secretary
Professor Elizabeth Smythe
Centre for Biomedical and Developmental Genetics,
Department of Biomedical Sciences, University of Sheffield,
Western Bank, Sheffield S10 2TN
Tel: 0114 2224635
e-mail: e.smythe@sheffield.ac.uk
Appointed 2006; retires 2010

Treasurer
Professor Mark Marsh
Cell Biology Unit,
MRC Laboratory for Molecular Cell Biology,
University College London,
Gower Street, London, WC1E 6BT
Tel: 020 7679 7807
Fax: 020 7679 7805
e-mail: m.marsh@ucl.ac.uk
Appointed 2001; retires 2007

Meetings Secretary
Dr Kairbaan Hodivala-Dilke
The Cell Adhesion and Disease Laboratory
Tumour Biology Laboratory
Cancer Research UK Clinical Centre
Bart's & The London
Queen Mary's School Of Medicine & Dentistry,
John Vane Science Center,
Charterhouse Square,
London, EC1M 6BQ
Tel: 020 7014 0406
FAX: 020 7 014 0401
email: kairbaan.hodivala-dilke@cancer.org.uk
Appointed 2003; retires 2009

Membership Secretary
Dr Jonathon Pines
Wellcome/CRC Institute of Cancer and Developmental Biology,
Tennis Court Road,
Cambridge, CB2 1QR
Tel: 01223 334088
Fax: 01223 334089
e-mail: j.pines@gurdon.cam.ac.uk
Appointed 2003; retires 2009

Newsletter editor
Dr David Stephens
Department of Biochemistry,
University of Bristol,
School of Medical Sciences,
University Walk,
Bristol BS8 1TD
Tel: 0117 928 7432
e-mail:
david.stephens@bristol.ac.uk
(to whom material should be sent – see guidelines for contributors)
Appointed 2004; retires 2010

Website Coordinator
Dr Tony Ng
Randall Centre, 3rd Floor,
New Hunt's House,
Guy's Medical School Campus,
King's College London,
London SE1 1UL
Tel: 020 7848 8056
Fax: 020 7848 6435
e-mail: tony.ng@kcl.ac.uk
Appointed 2003; retires 2009

Committee members

Dr Vania Braga
Molecular and Cellular Medicine Section,
Faculty of Natural Sciences,
Imperial College London,
Sir Alexander Fleming Building,
London SW7 2AZ
Tel: 020 7594-3233
e-mail: v.braga@imperial.ac.uk
Appointed 2004; re-election due 2007

Professor Iain Hagan
Cell Division Group
Paterson Institute for Cancer Research
University of Manchester
Wilmslow Road
Withington, Manchester M20 4BX
e.mail: ihagan@picr.man.ac.uk
Appointed 2006, re-election due 2009

Professor Adrian Harwood
Cardiff School of Biosciences
Biomedical Building
Museum Avenue
Cardiff CF10 3US
Tel: +44 (0)29 879358
Fax: +44 (0)29 2087 4116
Email: HarwoodAJ@cf.ac.uk
Appointed 2006, re-election due 2009

Dr Margarete Heck
The Wellcome Trust Centre for Cell Biology
Institute of Cell and Molecular Biology
University of Edinburgh
Michael Swann Building, Mayfield Road
Edinburgh EH9 3JR
Tel: 0131 650 7114
Margarete.Heck@ed.ac.uk
Appointed 2004; re-election due 2007

Dr Sean Munro
MRC Laboratory of Molecular Biology, Hills Road
Cambridge CB2 2QH
Telephone: (01223) 402236
Fax: (01223) 412142
E-mail: sean@mrc-lmb.cam.ac.uk
Appointed: 2005; re-election due 2008

Dr Stephen Nurrish
MRC Laboratory for Molecular Cell Biology,
University College London, Gower St, London,
WC1E 6BT
Tel: 020 7679 7267
e-mail: s.nurrish@ucl.ac.uk
Appointed 2003; retires 2009

Professor Roy Quinlan
School of Biological and Biomedical Sciences
South Road Science Site
The University
Durham DH1 3LE
Tel: 0191 334 1331
Fax: 0191 334 1201
e-mail r.a.quinlan@dur.ac.uk
Appointed 2001; retires 2007

Dr Jordan Raff (**Honor Fell Travel Awards**)
Wellcome Trust/Cancer Research UK Gurdon Institute
University of Cambridge
Tennis Court Road
Cambridge CB2 1QR
Tel: 01223 334114
e-mail: j.raff@gurdon.cam.ac.uk
Appointed 2002; retires 2010

Dr. Michael Way
Cell Motility Group
Cancer Research UK
Lincoln's Inn Fields laboratories,
44 Lincoln's Inn Fields
London WC2A 3PX
Tel: 44 (0) 207 269 3733
e-mail:
Michael.Way@cancer.org.uk
Appointed 2002; retires 2008

Dr Sylvie Urbé,
Department of Physiology,
University of Liverpool,
Liverpool
Tel: 0151 794 5432
e.mail: urbe@liv.ac.uk
Appointed 2004, re-election due 2008

Non-elected members

BSCB assistant
Margaret Clements
Department of Zoology,
Downing Street, Cambridge, CB2 3EJ
Tel: 01223 336655
Fax: 01223 353980
e-mail: bscb@bscb.org

Schools Liaison Officer
David Archer
43 Lindsay Gardens,
St.Andrews,
Fife, KY16 8XD
email: d.archer@talktalk.net

The BSCB newsletter is published twice a year in June and December.

Submission

If you have an idea for an article please e-mail the editor a brief outline first. Appropriate colour images are welcomed for consideration for the front cover.

It is preferable to send all articles, reports and images by e-mail (though alternatives can be arranged after contacting the editor).

Attachments for text can be in txt, rift or doc format. Please send images as 300dpi JPEG, TIFF or PSD files.
If images are for the front cover, please send as CMYK.

Submission of articles and images should be made to

Dr David Stephens
Department of Biochemistry,
University of Bristol,
School of Medical Sciences,
University Walk, Bristol BS8 1TD
Tel: 0117 928 7432
e-mail: david.stephens@bristol.ac.uk

Meetings

Please note there is no charge to advertise a scientific or educational meeting. Please contact the editor with details of any meeting you wish to advertise.

Subscription information

Paying by direct debit:
Regular member £25
Student, school teacher, retired member £10
UK resident members NOT paying by direct debit:
Regular member £35
Student, school teacher, retired member £15
Overseas members paying by bankers draft:
Regular member £25
Student, school teacher, retired member £10

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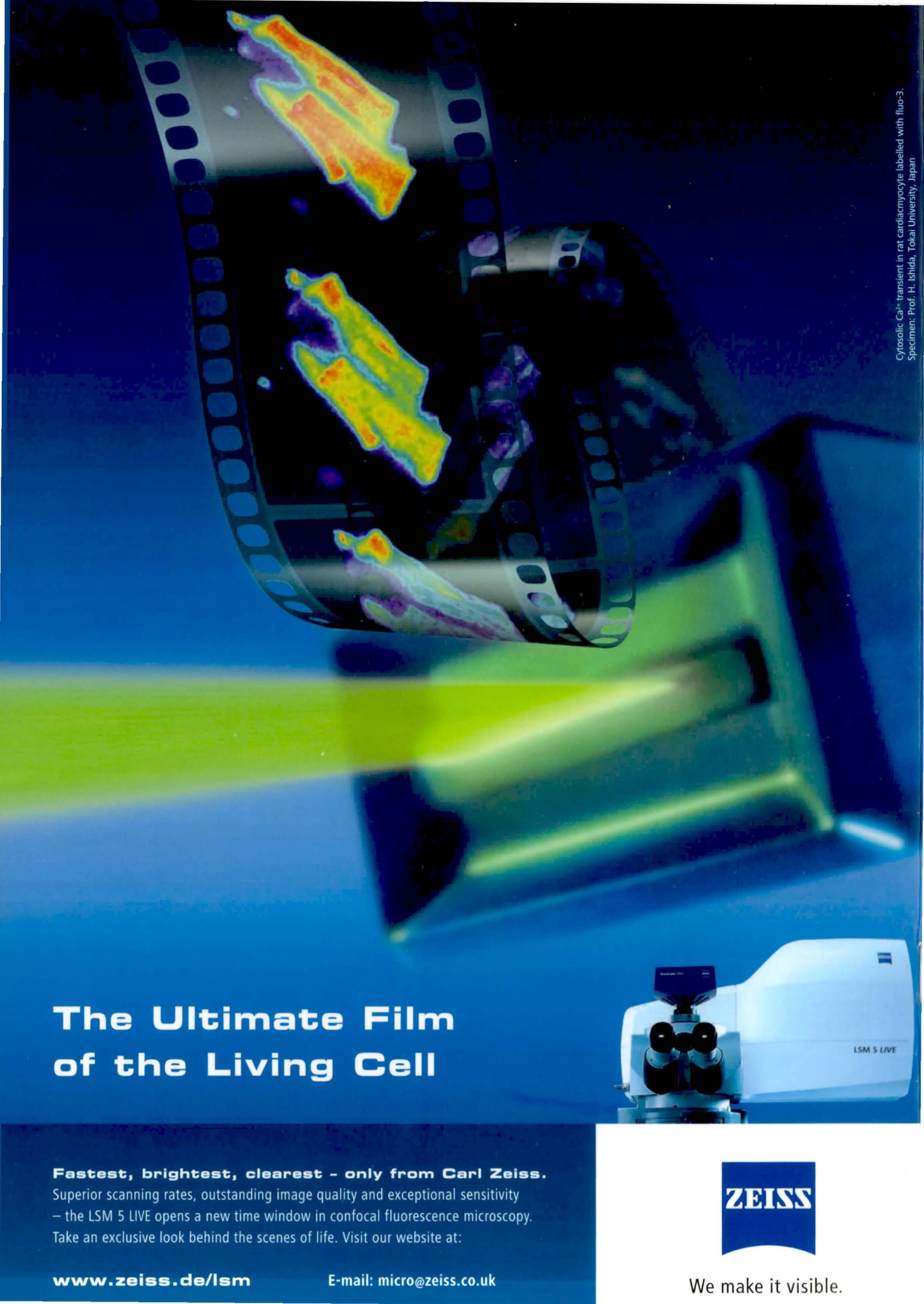

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