

BSCB Newsletter Summer 2005



Clearly special



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BSCB Newsletter

Summer 2005

Editorial

Once again, we returned to Warwick for a successful Spring meeting. A full report appears in this newsletter: my thanks as always to those who (were) volunteered to cover sessions. Everyone provided a lively report within a few days of the conference, most of them with very little prior warning. We organized two more themed lunches and these are described separately. They proved popular and will be a regular feature of the Spring meeting.

The major news this year is that the Society is now 40 years old. I visited one of the Founder members and heard some stories about the early days, the reasons for founding the society and its initial activities. I will follow this up over the summer and hope to report some more in the Winter Newsletter. Anyone who has any ideas or suggestions relevant to the anniversary should contact me or Fiona Watt, our President. Any budding journalists who would like to interview past officers would be especially welcome.

We have an excellent collection of meeting reports from those given an Honor Fell Travel Award. The scheme is proving more popular than ever and the Society invests a considerable sum; however, the enthusiasm in the reports suggests that it is money well spent. There is also another good collection of book reviews and my thanks to the contributors of these — some of whom are becoming 'regulars'. Finally, Anna Nasmyth has sent an interesting article about the Mendel Museum in Brno.

The Editor

Newsletter editor: Joan Marsh Design/layout: Giles Newton Printer: Hobbs Website: www.bscb.org

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News

Anne Ridley wins Liliane Bettencourt Life Sciences Award

The 2004 Liliane Bettencourt Life Sciences Award has been awarded to Anne Ridley for her outstanding research into the adhesion and migration of inlammatory and cancer cells and the important contribution she has made to the understanding of cell behaviour.

Professor Pierre Chardin said: "I do not know of any other living female biologist who has accomplished more by the age of 40 or conducted research of such importance". Anne was the

40th birthday

The BSCB celebrates its 40th anniversary this year. If anyone has any suggestions for articles or events to mark this occasion, please contact Joan Marsh or Fiona Watt, respectively. Any photographs or other material from the early days of the Society would be especially welcome.

BSCB committee

We have two new committee members, Sylvia Urbe (University of Liverpool) and Sean Munro (MRC Laboratory of Molecular Biology, Cambridge). Their contact details may be found on page 40.





inaugural winner of the BSCB's Hooke Medal in 2000.

The Liliane Bettencourt Life Sciences Award of Euro 250,000 is part of the Bettencourt Schueller Foundation's commitment to medical research. It aims to encourage a top-level European researcher under the age of 45, along with his or her team, to pursue their work in the field of biology or medicine. www.fondationbs.org

Poster Prize

This year's poster prize at the Spring Meeting was awarded to Eliana Lucas from the Gurdon Institute in Cambridge for her poster on the 'Role of Drosophila Pericentrin-like Protein: The strength of the centrosome'. Eliana wins a trip to the ASCB meeting in San Francisco this December. The runners up were JL Lathia from the University of Cambridge with his poster entitled 'Laminins are involved in neural stem cell maintenance' and C Panblanco, also from the Gurdon Institute, with a poster describing 'A casein kinase 1 required for the stability of the mitotic spindle in the C. elegans 1-cell embryo'. They win subscriptions to Nature Cell Biology and Developmental Cell, kindly donated by the publishers.

Be a cover star

Do you have a stunning photo of your cells that is just begging for greater recognition? Would you like it to grace the cover of a future BSCB Newsletter? If so, please send a TIFF or JPEG file (CMYK and high resolution) to Joan Marsh (jmarsh@wiley.co.uk).

In brief...

Cheaper journal subs for members

Did you know that BSCB members are entitled to discount subscriptions for several journals? The money saved more than compensates for your membership fee, so encourage your friends to join the Society. Details are on the inside back cover.

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. Requests for funds should be sent to the Treasurer, Mark Marsh (see page 39), accompanied by a report of a previous meeting. If a meeting receives such support, a report of the 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 (margaret@biologists.com).

BSCB Ambassadors

Don't forget the Society's Ambassador scheme. Representatives at the institutions listed below, which cover 80% of our membership, have agreed to promote Society activities and membership within their university or institute. They disseminate e-mail advertisements concerning future BSCB meetings, promote the advantages of BSCB membership, particularly to new PhD students, and are available to sign application forms and answer any BSCB-related queries. If your institution is not represented and you would be willing to become an Ambassador, please contact Jonathan Pines (see age 39).

City/Institution	Representative	E-mail
Aberdeen Bath Birmingham Bristol Cambridge Dundee Durham	Denys Wheatley Geoff Holman Rob Insall Harry Mellor Jon Pines/Paul Luzio Angus Lamond Roy Quinlan	wheatley@abdn.ac.uk g.d.holman@bath.ac.uk R.H.Insall@bham.ac.uk H.Mellor@bristol.ac.uk jp103@cam.ac.uk a.i.lamond@dundee.ac.uk r.a.quinlan@durham.ac.uk
Edinburgh	Bill Earnshaw	Bill.Earnshaw@ed.ac.uk
Glasgow	Steve Winder	s.winder@bio.gla.ac.uk
Guildford	Tom Wileman (Pirbright and all BBSRC)	thomas.wileman@bbsrc.ac.uk
Imperial	Vania Braga	v.braga@ic.ac.uk
Cancer Research UK (LIF)	Fiona Watt	f.watt@cancer.org.uk
Kings/Guys	Simon Hughes	s.hughes@kcl.ac.uk
Leeds	Michelle Peckham	m.peckham@leeds.ac.uk
Manchester NHLI etc Norwich	Charles Streuli Clare Isacke Peter Shaw/Grant Wheeler	charles.streuli@man.ac.uk c.isacke@icr.ac.uk grant.wheeler@uea.ac.uk
Newcastle Queen Mary	Michael Whitaker Mark Turner	michael.whitaker@newcastle.ac.uk m.d.turner@qmul.ac.uk
Sheffield	Liz Smythe	e.smythe@sheffield.ac.uk
UCL	Mark Marsh	m.marsh@ucl.ac.uk
York	John Sparrow	jcs1@york.ac.uk

Schools News

"All biologies are equal but some are more equal than others" Adapted from George Orwell, 'Animal Farm', with apologies.

By David Archer

This article is not about the evolution or revolution that determines dominance or recessiveness in certain pigs. It is about the interesting figures regarding the uptake of biology at 'Highers' level in Scotland and 'A' level in the rest of the UK and whether biology at 'A' level as currently studied is an appropriate precursor subject for the study of biology at tertiary level and a career in biosciences.

We have almost become accustomed to hearing about the decline of interest in science as a school subject, with physics and chemistry taking most of

the knocks. At 'A' level, biology is still a popular subject and the most popular of the main three sciences, which rank in the order biology, chemistry, physics. It attracted 52 264 candidates in 2004 compared with 28 698 candidates for physics. In Scotland, however, at 'Higher' level (the nearest equivalent to 'A' level), biology was the least popular of the three main sciences with 8852 pupils electing for biology, 9271 for chemistry and 9286 for physics. The reason for the marked difference between Scotland and the rest of the UK is not known to the writer but could be influenced by,

amongst other things, the acceptability of subject groupings. In many schools in England, biology can be taken as a 'stand alone' subject for either one or two years, with no need to study chemistry or physics concurrently.

The present 'A' level courses are considered by many to demand the recall of too many facts and the use of too few skills. Be that as it may, the subject is popular with both pupils and schools and is considered a 'must have' for young people considering a career at any level in veterinary work, medicine and related occupations. For schools, it attracts a high number of entries at 'A' level and provides a harvest of good grades. These in turn are good for school performance tables and for attracting 'new customers'. In addition, teachers of biology are not too hard to find compared with those proficient in chemistry and physics. For pupils, the result is an 'AS' or complete 'A' level in a science subject, but without the perceived difficulty of chemistry or physics. Many pupils elect to study biology in the expectation that it is a 'softer' and less numerate subject. More than a few find it harder and more numerate than anticipated.

You could say that all this is positive and ensures that a percentage of the adult population will have some degree of scientific literacy. BUT, does the content and teaching of 'A' level biology provide a good introduction and foundation for later work in modern biology and does this matter anyway?

The answer is not simple. If biology is taken on its own, it probably does not provide a good introduction. If it is coupled with chemistry, it probably does.

Does it matter? Probably not, if the student does not intend to study the subject beyond the first year of 'A' level. It could be argued that it is better to have a science subject that remains attractive than to add so much quantitative biology and biological chemistry that it loses out in the popularity stakes. The syllabus is pretty up-to-date but is overloaded and there are moves to prune the amount of content and emphasize concepts and skills. An attempt to represent or accommodate the full range of subdisciplines of biological study is not an option. Special interest societies or groups will have to trust that a course based on main concepts and contemporary skills will be so fundamental as to be acceptable to all. More detailed information for

special projects will, as now, be available from specialist societies and publications. Obtaining this information will also give students a better insight into study and work opportunities in the different biosciences.

So how do we go forward? For biology at 'A' level or equivalent, we must make sure that the work is attractive to students. It must be understandable to pupils working to a tight time schedule with access to the Internet, but with perhaps less access to upto-date book library facilities than in the past.

Teachers must feel comfortable with the course content and skills. The Save British Science Newsletter for July 2003 (No37) included the statement that "...only half of biology teachers say that they have a "lot of confidence" in their ability to teach modern biological material." Teachers who are not confident about their own subject knowledge are unlikely to be enthusiastic when teaching it.

To accommodate all these factors, a syllabus of carefully thought out major concepts, interwoven with skills such as data handling and pattern recognition, could be suitable and attractive. We cannot let the popularity of 'A' level biology fall. Perhaps some of the detailed work can be left to tertiary level when a decision has been made to 'bond to biology' – or not! There have been some suggestions that a new biosciences curriculum for the 'new biology' should be written. Those wanting a more quantitative and chemical approach could elect for this, but I doubt whether it will become more than a talking point, at least in the foreseeable future.

I end therefore by saying "All biologies are equal", are valuable and have a place. However, at certain times and in certain places some biologies will be "more equal than others" only to be ousted by other newly developed, or newly named, disciplines. Have you seen the new label on the door: 'Office of Omics and Systems Biology'?

(Note: Figures for exam results are taken from secondary sources: the Internet and press).

David Archer d.archer9@ntlworld.com

Gregor Mendel's Legacy

New museum of genetics

The legacy of Gregor Mendel, the 19th century friar who discovered the laws of heredity, has been secured by the formation of the Mendel Museum, Museum of Genetics, in the Abbey of St Thomas in Brno, where Mendel lived and worked.

Fundraising for the Mendel Museum began in May 2002, when the Abbey opened its doors to the public with an exhibition entitled, 'The Genius of Genetics, a celebration of Gregor Mendel through science and art', curated by Marina Wallace and Martin Kemp. The exhibition has been seen by over 15,000 people in Brno and travelled to the Genoa Science Festival in October 2003. A second exhibition, 'Genes and Genius, the Inheritance of Gregor Mendel', is planned for September 2005 and both exhibitions will transfer to the Field Museum in Chicago in 2006, followed by an American tour.

This initiative laid a solid foundation to establish the Mendel Museum, Museum of Genetics, now a legal entity under Czech law. The Mendel Museum provides a permanent home in museum conditions for the archive of items and documents belonging to Gregor Mendel and the Abbey of St Thomas, most of which have never been on display before. The aim of the Museum is to establish a programme of genetic exhibitions that cover both the history of genetics and current research topics and to communicate these with imagination and insight to a wide general audience.

The restoration of Mendel's garden continues, as part of the Museum's work. A landscape design competition was held for students at the Mendel University of Agriculture and Forestry and the winner participated in the design process with the architect, Eva Jiricna, to complete the genetics demonstration garden and to redesign the Abbey garden along genetic themes. Mendel's bee house has been restored and working bees are now once more in the apiary.

The Mendel Center, which opened in the Abbey in May 2002 with the inaugural conference 'EMBO



Workshop, Genetics after the Genome', aims to provide a centre for scientific discovery, communication and education alongside the Museum. The Mendel Center hosts conferences in the elegant Abbey rooms and runs a lecture series in association with the Czech Academy of Sciences. Speakers in the lecture series have included: Walter Bodmer, François Gros, David Hopwood, Tim Hunt, Horace Freeland Judson, Anne McLaren, Robert Olby and Charles Weissmann. This year's speakers are: Barry Dickson, Ernst Hafen, Alec Jeffries, Marc-Andre Sirard, Jack Szostak and Edward Trifonov and confirmed speakers for 2006 are Adrian Bird, Susan Lindquist and Steve McKnight. Bookings for conferences can be made through Anna Nasmyth.

Professor Gustav Ammerer, director of the Mendel Museum, said: "We have reached a milestone in our plans to preserve Gregor Mendel's scientific and intellectual legacy. Scientists and public alike will be able to learn not only about the remarkable origins of the science of heredity but also about the enormous impact it has had on society."

Anna Nasmyth anna@imp.univie.ac.at www.mendel-museum.org

The Mendel Museum acknowledges the support of the City of Brno, the South Moravian Region and the Czech Commission for UNESCO.

Call for assistance

Please help us preserve Mendel's legacy by making it available to the public. Donations can be made to "The Gregor Mendel Trust", c/o Simon Weil, Bircham Dyson Bell, 50 Broadway, London, SW1H OBL (simonweil@bdb-law.co.uk).

Sponsor Mendel's Garden

You can help restore Mendel's Garden as a genetics demonstration garden by sponsoring a square metre for £100. We will send you a certificate and your name will be added to the list of sponsors on the web site. The garden is being restored under the supervision of John S. Parker, Director of the Cambridge University Botanic Garden, Prof Ladislav Havel of the Mendel University of Agriculture and Forestry in Brno and Eva Jiricna, a renowned Czech architect.

Back to the beginnings

Memories of 40 years ago from Sam Frank

This year, the British Society for Cell Biology celebrates its 40th anniversary and the committee felt it appropriate that we contacted some of the founder members to hear about the early days. This resulted in my enjoying a very pleasant lunch with Sam Frank and his wife, then listening to Sam talk about the events that led to the establishment of the Society.

By Joan Marsh

Sam Frank was a medical doctor who served in Italy during the Second World War and has some fascinating tales to tell of his time there. On leaving the Army, he took a hospital position and was then one of the first people to work in a new unit studying human cancer – this was to become the Imperial Cancer Research Fund Unit in Lincolns Inn Fields, only recently incorporated into Cancer Research UK. Sam worked on prostate cancer and, since this was a disease of old men, he also became interested in ageing.

Honor Fell was trying to establish organ cultures, so Sam focused on cell cultures. This may seem routine today but was a major challenge at the time. The first to succeed was George Guy, who cultured over 200 human tumours before he managed to establish the HeLa cell line. Cells were grown in serum, either horse serum or placental cord serum collected from maternity wards. No antibiotics were used as the scientists were worried that these would alter the properties of the cells. People were not routinely looking at the cells in their cultures: Sam examined the cells using electron microscopy and was able to demonstrate that the claimed 'surface antigens' were all bacteria. He wrote a paper for the International Review of Cytology in 1977 on the origin and ultrastructure of cells in culture that he says all students should read, even today, so that they know what they are looking at.

There was a European Tissue Culture Club dating from the Second World War or even earlier, but in about 1950, Sam Frank, together with Honor Fell, John Paul, Michael Abercrombie and Neville Willmer, set up the British Tissue Culture Society. As time progressed, there was some debate in the USA as to whether a society should be based around a technique. Those working on tissue cul-

ture decided that there was a need for such a society and continued, while others founded the American Society for Cell Biology. A similar discussion occurred in the UK in the early 1960s and finally the British Society for Cell Biology was established. Honor Fell was the President and Sam Frank later became the Secretary/Treasurer. Michael Abercrombie organized a very well attended first conference. This tradition persists and the Society holds an Abercrombie meeting every four years in Michael's honour.

The BSCB benefitted from another conference organized in St Louis, USA, in 1970 under the auspices of the International Federation for Cell Biology, which had four national societies as founder members (it now has over 50) and Sam Frank as its Secretary-General. Half of the profits of this conference were assigned to the BSCB and used to establish the Honor Fell Travel Fund. Each year, several students take advantage of this scheme to attend science meetings around the world; I doubt many of them know that the original requirement was that one should be beardless and not wear purple corduroy!

Unfortunately, during John Gurdon's tenure as President, there was a flood at his laboratory and the minutes of all the early activities of the Society were destroyed. If anyone has any information regarding the formative years of the Society, I would be pleased to hear from them.

My thanks to Sam for the lunch and for a most informative and entertaining afternoon. And the thanks of all current BSCB members to those who were far-sighted enough to establish the Society that we know today.

Joan Marsh, jmarsh@wiley.co.uk

Dennis Summerbell

1947-2005

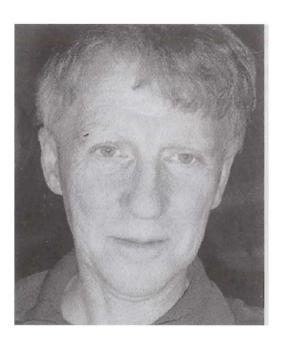
Dennis Summerbell, who performed pioneering experiments on the development of the chick limb, has died at the early age of 58. He had been suffering from pancreatic cancer for 19 months, and had borne his illness with characteristic courage, dignity and humour.

By Jim Smith

I first came across Dennis in 1976, when I started as a graduate student with Lewis Wolpert, just three years after Dennis had finished his own PhD in the same lab. Dennis had first gone on to do a postdoc in Grenoble with Philippe Sengel, and by now was working in the University of Otago, New Zealand. Dennis's location on the other side of the world enhanced his almost mythical status in Lewis's lab, for he had produced a PhD thesis that was so comprehensive, so magisterial, so brilliant, that my fellow students and I (including Nigel Holder, Geoff Shellswell and John McLachlan) despaired of finding any new experiment that he hadn't already done.

In my own PhD thesis, first author papers by Summerbell would have occupied a whole page in the references had I not, to tease him, squeezed in a Szabo reference before starting a new sheet of paper. I sometimes wonder how Dennis managed to achieve so much in such a short time; it must have had something to do with the way he combined his great technical expertise with a deep interest in theoretical models for limb development. A rare combination then, and even more so now.

The most significant parts of Dennis' PhD thesis helped us understand how positional information along the antero-posterior and proximo-distal axes of the developing limb is specified. The work was described in two articles in *Nature*, the first with Julian Lewis and Lewis Wolpert and the second with Cheryll Tickle and Lewis Wolpert, and they were of extraordinary importance. The papers are cited to this day, and their significance was illustrated recently by the intense excitement that surrounded the suggestion that the so-called 'progress



zone' model (in his thesis Dennis called it the 'magic zone') might be wrong. Any developmental biologist would have been delighted, and proud, if their model had managed to last for over 30 years before being questioned in a serious manner, but Dennis had cause to be even prouder, because, the recent results notwithstanding, the progress zone model remains the best way to understand how positional information is specified along the proximo-distal axis of the limb.

On returning from Otago, Dennis moved to Mike Gaze's Division in the National Institute for Medical Research in Mill Hill, where he teamed up with like-minded developmental biologists such as Jonathan Cooke, with whom he analysed cell divi-

sion during limb development, Vicky Stirling, with whom he carried out some beautiful experiments on innervation of the limb, and Malcolm Maden. The partnership with Malcolm was particularly important, providing, as it did, some of the first insights into the roles of retinoic acid signalling during development. As I got to know Dennis, I came to see how this work, like all his work, was careful, meticulous and beautifully controlled. Dennis had a strong regard for the truth and would never make any assertion unless he knew beyond doubt that it was true.

Not long after Malcolm left NIMR to go to King's College London, Dennis decided that he wanted to learn how to apply molecular biological techniques to development and he joined Peter Rigby, also at Mill Hill, to study the regulation of muscle gene expression in the mouse embryo. He worked on the *Hox* genes and on the skeletal muscle determination gene *Myf5*. His characteristically painstaking and beautiful analysis of the extraordinarily complex regulation of *Myf5* has been highly influential in the field.

He published regularly with Peter in journals such as Development and Genes and Development and

moved with him to The Institute of Cancer Research in 2000, where he continued to work at a high level even after he was diagnosed with pancreatic cancer. He did it, in spite of the pain, because he just loved doing science.

Dennis married Amata Hornbruch in 1971. Their house, close to NIMR, became a haven for PhD students and postdocs in developmental biology: for times of celebration, for when things were getting a bit stressful, and for when they (the younger scientists) fancied a good glass of wine; Dennis had an excellent cellar and he and Amata were superb hosts.

Dennis was also a tremendous teacher and mentor of young scientists and was an important influence on many careers. He will be greatly missed by his colleagues at The Institute of Cancer Research, as well as by his many friends and admirers at Mill Hill and around the world. But no one will miss Dennis more than Amata, to whom we send our deepest sympathy.

Jim Smith

Books for review

Fancy reviewing a book? If so, choose one from the selection listed below. Alternatively, if there is a book you would like to review that is not included here, contact me (jmarsh@wiley.co.uk) and I will request a review copy from the publisher.

Reversible Protein Acetylation Novartis Foundation symposium Wiley

Stem cells: nuclear reprogramming and therapeutic applications Novartis Foundation symposium 265 Wiley

Database Curation Lesk, Wiley

Ecological Genetics Lowe, Harris and Ashton, Blackwell Science

Fundamental Bacterial Genetics Trun and Trempy, Blackwell Science

Analysis of Genes and Genomes Reece, Wiley

Domains in Integrins Gullberg, Kluwer Academic/Plenum

Molecular Infection Biology: Interactions between microorganisms and cells Hacker & Heesemann, Wiley

Microbial Diversity Ogunseitan, Blackwell Science

The hERG Cardiac Potassium Channel Novartis Foundation Symposium 266, Wiley

Chromosome segregation Nasmyth and Yanagida, Philosophical Transactions: Biological Sciences

Book reviews



Centrosomes in Development and Disease Edited by Erich A. Nigg Wiley VCH 3527-309802 August 2004. 474 pages. Centrosomes in Development and Disease Edited by Erich A. Nigg

The centrosome is a small non-membrane-bound organelle, which has captivated and intrigued cell biologists ever since it was first described over 100 years ago by the early cytologists – indeed the editor compares it to the Mona Lisa's smile in its beauty and mystery!

The centrosome consists of two centrioles - analogues of the basal body of cilia and flagella - surrounded by a complex mix of proteinaceous pericentriolar material that acts to nucleate and anchor microtubules in the cytoplasm of a wide range of organisms. Over the last decade or so, the centrosome field has made huge strides. It has employed techniques as diverse as light and electron microscopy, genetics, biochemistry, mass spectrometry and laser microsurgery, in model organism such as Saccharomyces cerevisiae, Caenorhabditis elegans, Drosophila melanogaster and Chlamydomonas reinhardtii, as well as animal cells in culture. This book therefore comes along with perfect timing. It covers essentially all these areas and furthermore deals with the role of centrosomes in cell cycle control and cancer, their involvement in infectious diseases caused by intracellular pathogens and their significance in the life cycle of human parasites.

The book begins with an elegant historical perspective with reproductions of the original handdrawn illustrations, reflecting the painstaking observations of centrosomes made by the pioneer cytologist Edouard Van Beneden. It describes the early input into theories of centrosome function from a rival young researcher — a certain Theodor Boveri. Moreover, the author, Jo Gall, even shows some of his own microscopy images taken using Boveri's original slides of Ascaris eggs.

A key function of centrosomes is to nucleate the polymers of $\alpha\text{-}$ and $\beta\text{-}$ tubulin that we know as microtubules. The third chapter in the book gives a thorough discussion of the role of $\gamma\text{-}$ tubulin and the large $\gamma\text{-}$ tubulin ring complex, termed $\gamma\text{-}$ TuRC, in the nucleation process and presents a model based on kinetic and structural data. Studies of the spindle pole body – the centrosome analogue in the genetically tractable yeast – have played a major role in increasing our understanding of centrosome function and the book gives an excellent overview

of the state of play in this field. It details the morphology, molecular composition and duplication mechanisms of the spindle pole body and its important role as a signalling platform in the mitotic exit network. The book also covers the contributions to our understanding made by a different unicellular eukaryote, the green alga Chlamydomonas reinhardtii, regarding the role of the basal body/centriole. An outstanding chapter deals with the evolutionary aspects of centrosome function and provides thought-provoking insights. The mass spectrometer has contributed to many areas of cell biology over the last decade. The book gives an excellent overview of the contribution proteomics has made to realising the goal of a complete inventory of the human centrosome.

Many of the studies on the centrosome have focused on the mechanisms controlling centriole duplication and separation during the cell cycle, a topic also well covered in the book. The chapters that stand out in this context are those from Kip Sluder (9) and Alexey Khodjakov and Conly Reider (10). The latter authors elegantly describe the pioneering work using laser microsurgery to selectively ablate not only the entire centrosome but even a single paired centriole, allowing them to show that centrosomes are not required for spindle assembly in somatic cells. Both chapters describe efforts to determine the role the centrosome plays in regulating entry into mitosis and its apparent function in blocking initiation of replication.

The third part of the book covers the role of the centrosome in development and tissue architecture, with a particularly well-written chapter describing the structure and function of the centrosome in the early embryonic development of *C. elegans*, followed by a great chapter on the important contributions *Drosophila* studies have made to our understanding of the developmental aspects of centrosome function in this system. The role of centrosomal and non-centrosomal-nucleated microtubule arrays in the functions of polarized epithelial cells is described in a well written chapter by Mette Mogensen.

In the last section, entitled 'Centrosomes in Disease', several chapters deal with centrosomes and cancer. A very early observation made by Boveri was that certain characteristics of malignant tissues, such as loss of cell polarity and chromo-

some segregation defects, were the results of aberrant centrosome function. Viral effects on centrosome and microtubule networks, as well as the way certain intracellular pathogens use the microtubule cytoskeleton to their advantage, are covered in an interesting penultimate chapter. Lastly, but far from least, is an excellent chapter on the basal body and microtubule cytoskeleton in pathogenic protozoa such as *Trypanosoma brucei*.

Overall, Centrosomes in Development and Disease is a comprehensive book which is well written, concise and has many excellent reviews of the key topics in the field. The book balances the historical with the cutting edge, the background with the detail and is therefore a recommended read for the newcomer and the experienced centrosome researcher alike.

Paul D. Andrews
Division of Gene Regulation and
Expression
Wellcome Trust Biocentre, School of
Life Sciences
University of Dundee
paul@lifesci.dundee.ac.uk

Advanced Genetic Analysis, Finding Meaning in a Genome

R. Scott Hawley and Michelle Y. Walker

Genetic analysis is progressing at a great pace and it is necessary for both researchers and students to keep up to date with the latest analysis methods. When I first looked at the title of this book I was intrigued to find out how the authors had managed to write a book on something as changeable and developing as the field of genetic analysis. Surely such a publication would become out of date very soon.

The book covers important areas of the basic principles that underlie genetic analysis: mutation, complementation, recombination, segregation and regulation. For each of these, the authors explain their definition and uses in genetic analysis. As the authors mention in the preface, this is a book about genetic theory not genetic facts. It is supposed to give the reader an insight into the biological and analytical processes that constitute each of the tools and explain their use.

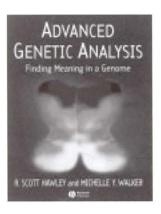
The book is divided into eight chapters describing concepts ranging from meiosis segregation to the

complementation test but, in my opinion, these are presented in a disorganized fashion. It starts by describing mutants, then the use of mutants in genetic analysis, and then complementation tests. So far so good, but in the fourth chapter the reader feels as if returning to near the beginning of the book with a description of Suppression types. From here, the authors explain gene function and 'Genetic fine-structure analysis'. Then in Chapter Seven, meiotic recombination and finally meiotic chromosome segregation are described. I would have preferred to start with these last two topics.

However, this book is intended for an advanced course in genetic analysis and postgraduates should be able to use it to its full purpose, learning new ways of performing genetic analysis and which model organisms to use. This book is also a very good aide memoire for experienced researchers in this field, as it describes concepts in an up to date fashion and in a concise way. There is a helpful list of internet links to useful resources but one has to query how these links will function in the future.

In summary, this book is well written, the information is up to date and it refers to many model organisms.

Monica Mascarenhas Biochemistry & Molecular Biology Department, Royal Free & University College Medical School m.mascarenhas@rfc.ucl.ac.uk



Advanced Genetic Analysis: Finding Meaning in a Genome R. Scott Hawley and Michelle Y. Walker Blackwell Science 1405103361 December 2002

Nuclear Organization in Development and Disease

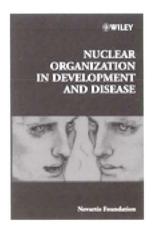
Novartis Foundation Symposium

This book is the product of a Novartis Foundation symposium held in London in January 2004, which brought together 31 leading scientists within this field to present and discuss their research. The book is a collection of the presentations, generally in review form, plus the discussion that followed every talk, giving valuable insight into the minds of the participants and their thoughts on the subject.

The book starts with an introductory review on the nuclear lamins, which are important nuclear proteins for both nuclear structure and function, and indeed the most talked about group of proteins in this book. The roles of lamin and other nuclear envelope proteins in cell division are discussed in the second paper, lamin being thought to have an important role in nuclear envelope assembly and disassembly during this process.

Mutations in the gene encoding Lamin A/C are a primary cause of a group of related diseases termed 'laminopathies', which are discussed in general in the third paper and more specifically later in the book. These diseases include Emery-Dreifuss muscular dystrophy, a partial lipodystrophy, a peripheral neuropathy disorder and premature ageing syndromes. On the surface, these diseases all appear to be very different and it is difficult to understand how the same protein could cause them all, which is a major issue addressed by these scientists in their articles.

Lamin has a wide variety of binding partners, which are thought to determine the disease phenotype

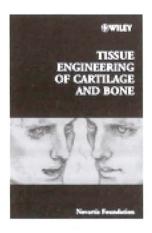


Nuclear Organization in Development and Disease Novartis Foundation Symposium John Wiley & Sons 0470093730 January 2005 according to which particular lamin mutation is present, and some of these are discussed in detail. One of these binding partners is Emerin, a nuclear envelope protein. Emerin mutations result in an X-linked form of Emery-Dreifuss muscular dystrophy that is clinically indistinguishable from the same disease caused by lamin mutations in Emerin-binding regions.

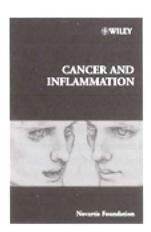
There is a distinct lack of textbooks that cover this area of cell biology and this book fills the gap

nicely. It is a comprehensive introduction to the laminopathies and will be useful for scientists and students in this field. Alongside the background information presented here, there is also recent research, making the book an attractive alternative to trawling the scientific literature in search of upto-date background reading.

Lindsay Emerson, Kings College London. lindsayjemerson@yahoo.co.uk



Tissue Engineering of Cartilage and Bone Novartis Foundation Symposium John Wiley & Sons 0-470-84481-7 March 2003 262 pages



Cancer and Inflammation Novartis Foundation Symposium John Wiley & Sons 0-470-85510-X January 2004 290 pages

Tissue Engineering of Cartilage and Bone Novartis Foundation Symposium 249

The Novartis Foundation receives many proposals for meetings each year, but holds just eight, hence any meeting given the go ahead should be considered particularly relevant. Tissue engineering of cartilage and bone was one such meeting.

This field was initially driven by biomaterial scientists designing novel bioresorbable scaffolds on which cells could be seeded to grow tissues. An enormous amount of work is answering the questions raised by the pioneering work. Current questions surround defining the optimal cell source and the most effective cell scaffolds.

The key issues addressed in this book are wide ranging, from the fundamentals of scaffolds and bioreactors to the qualitative and quantitative assessment of articular cartilage in vivo using magnetic resonance imaging. The book provides presentations and subsequent discourse surrounding the issues raised and mused over by a variety of leading scientists and clinicians within the field. Those with a strong knowledge of the field would find the book very valuable. As appears to be common with these books, a large amount of knowledge surrounding each topic is assumed and it will not be an easy read otherwise. Its main strength is that there are a variety of perspectives discussing these topics and perhaps those with sufficient background knowledge would find an idea or a question raised particularly pertinent.

In summary, the book highlights key science within the field of tissue engineering and identifies emerging ideas utilizing dialogue amongst a broad spectrum of leading experts. The book successfully sets about translating bone and cartilage tissue engineering principles into a clinically relevant science, which should lead to the development of more effective treatments.

Mark Howard, Smith & Nephew, York. mark.howard@smith-nephew.com

Cancer and Inflammation

Novartis Foundation Symposium 256

The Novartis foundation is an international scientific and educational charity, which focuses on promoting international dialogue in scientific research. The Foundation organizes international meetings throughout the year and then publishes highly respected books featuring the presented papers and the ensuing dialogue. This particular book, Cancer and Inflammation, draws together contributions from an international group of scientists and clinicians from diverse disciplines, ranging from epidemiology to immunology, cell biology, molecular oncology, molecular medicine and pharmacology, to debate the research presented and related issues. Thus the book constitutes, in one authoritative reference source, the recent work of many of the major laboratories involved in the study of cancer progression and the putative role of inflammation in facilitating the processes of tumour growth, angiogenesis and metastasis.

Topics covered include the epidemiological links between cancer and inflammation, the parallels between inflammation and cancer, the role of inflammation in cancer, inflammatory genes as risk factors for cancer initiation and progression, inflammation and cancer angiogenesis, and preventative and therapeutic strategies. Whilst generally insightful, the written conversation following each of the presentations is not always easy to follow and on occasion appears to wander. However, the book does contain a number of broader discussions throughout, based on the papers presented thus far which helps clarify these dialogues.

In summary, those with a strong background in the specific topics covered will most likely find this book a useful guide to the considered thoughts of those prominent within their field.

Mark Howard, Smith & Nephew, York. mark.howard@smith-nephew.com

The 44th ASCB meeting in Washington December 2004

As last year's winner of the BSCB poster prize, I received a free ticket to attend the American sister society's meeting. This was held in the brand new Washington Convention Centre in downtown Washington DC, just a block uphill from Chinatown.

By Bernhard Strauss

Though the scale of the meeting was impressive enough, with thousands of cell biologists congregating, the architecture of the convention centre added an extra dimension of grandness to the atmosphere. From the top galleries of the light-filled lobbies, one had a panoramic view over Washington. When you looked down into the entrance hall, all the cell biologists buzzing in and out seemed really small, just like some kind of vesicles trafficking on invisible microtubule tracks (or actin?).

The first day had a heavy schedule even before the evening opening lectures. A number of 'special interest subgroups' met in the afternoon, with topics ranging from 'Actin Nucleation and Organization by Adhesive Contacts' to 'Embryonic Cell Biology', as well as some on career development issues. With the brain so stimulated, in my case by the latest news on microtubule plus-end tracking proteins, I was looking forward to the keynote speakers. I was expecting to hear some big picture views that would put my larval cell biologist's existence into perspective. The title was visionary indeed: "Cell biology: Rising to meet the medical challenges of the next century". The two speakers, Peter Kim from the Merck Research Laboratories, and Nobel laureate Sir Paul Nurse, now at The Rockefeller University, presented their views on how these medical challenges could be tackled. Peter Kim, representing application and product-driven industry research, showed a promising antibody-based approach for an HIV vaccine. He then gave an overview of how bioinformatics should accelerate and optimize drug discovery in the post-genomics era. Only one in 10 potential drug molecules that enter clinical trials finally come to the market. Paul Nurse, his face displayed in almost sub-cellular resolution on three gigantic screens for the entire lecture, presented some historic and conceptual landmarks that might motivate cell biologists for the years to come. His starting point was Virchov's (19th century) suggestion

that malfunctioning of cells might be the basis of disease and therefore that the normal function of the cell needs to be understood first. Although major progress has been made since then, Sir Paul stated that we are still far from such an understanding. In light of increasing pressure from funding bodies to work on questions that will lead to applications, he stressed the importance of basic research to "understand how life works". He went on to suggest that cell biologists need to look for new theoretical approaches to analyse the enormous complexity of cell behaviour. With a little help from the physicists, Paul Nurse is convinced that "the cell is solvable".

Invigorated by this optimistic outlook, I delved into the programme on the next day. Each morning there were two consecutive symposia on offer with a break in between, when everybody was chemotactically attracted by coffee towards the poster and exhibition hall. After the morning symposia, there was time for lunch and poster sessions before a selection of eight minisymposia run in parallel, which made it hard to choose, as there was always something interesting in concurrent sessions.

On the Sunday morning, I went for 'Directed Cell Migration in Development'. **Pernille Rørth** (EMBL, Heidelberg), using border cell migration in *Drosophila* oogenesis as a model system to study invasive migration of cells through tissues, presented data that demonstrated that SRF (serum response factor) and its cofactor MAL-D are required for migration. She showed that SRF/MAL-D is involved in organizing the actin cytoskeleton and that its activity is induced by mechanical cell stretching. She suggested that tension-induced MAL-D/SRF activity and subsequent actin remodelling form a feedback mechanism to regulate cytoskeletal robustness during invasive migration. **Susan McConnell** (Stanford

University), who chaired this session, presented data on migrating neuronal precursors in the developing mammalian brain. In her lab, a matrigelbased culture system is used to image single, migrating cells in vitro. From her time-lapse data, as well as EM studies, she has discovered some important characteristics of such migrating neurons. The initial formation of a leading process seems dependent on Doublecortin, a microtubuleassociated protein. Subsequent movement of the cell body starts with the formation of a cytoplasmic dilation in the direction of future movement into which first the centrosomes move, then the nucleus follows on parallel microtubule arrays. To complete translocation, the activity of non-muscle myosin 2 is required.

As I work on early cell division in Xenopus, I had to hear the news from Michael V. Danilchik (Oregon Health and Science University) on 'Polarity of Embryonic Cleavage Furrow Microtubules'. In several species, such as teleosts, sea urchins and Xenopus, the cleavage furrow microtubules of the first cleavage divisions contain a specialized population of hook-shaped microtubule bundles that are thought to play an important role in vesicular transport at the leading edge of the cleavage furrow, as well as during abscission. Dr. Danilchik could show by live imaging of tubulin-GFP and EB1-GFP that the mid-body and the cleavage furrow microtubules in vivo contain very stable bundles that are not derived from the midzone of the mitotic spindle. (I have seen these in my own movies!) Their formation and stability seems to be independent of the activity of centrosomes and they are centres of microtubule nucleation on their own. He suggested that these specialized bundles are composed of completely overlapping arrays of anti-parallel microtubules.

On Sunday evening, Tom Pollard (Yale University) was honoured with the E.B. Wilson medal and his talk was an overwhelming summary of decades of hard work on the 'Molecular Basis of Cellular Movements'. Yes, he was the one who discovered that a myosin molecule, later termed non-muscle myosin 2, is involved in cell movements. Apart from giving an impressive review of his career and the many contributions to the field, he also conveyed the message that as a cell biologist today one has to try to understand it all: from the descriptive level of studying cell behaviour with ever better microscopy all the way down to the sub-molecular level. He also stressed the importance of mathematical modelling at all levels of analysis. To conduct research aiming for such complete explanations of cell biological questions certainly requires extensive collaborations.

A minisymposium on 'Asymmetry in Development' was on my have-to-go list as some of the talks

were directly relevant to my own research on spindle orientation. Data from Chris Doe's lab (University of Oregon) suggested that Lis1/dynactin independently regulate spindle positioning and mitotic checkpoint inactivation in Drosophila neuroblasts. By studying neuroblast divisions with time-lapse microscopy, they found that spindle poles are already aligned at late prophase with the eventual axis of division in wild-type neuroblasts. During prometaphase and metaphase, spindle poles undergo oscillations that indicate pushing or pulling forces. In Lis1/GI (Glued is a Dynactin domain) mutants, spindle poles were misaligned prior to anaphase and oscillations were reduced indicating a direct effect on force generation. However, at telophase proper spindle orientation is restored and cell fate determinants become segregated properly. From their mutant analysis they suggest that spindle position depends on an early Lis1/GI dependent pathway and a late telophase pathway that is able to rescue final spindle orientation independent of Lis1 and Dynactin.

After sitting through talks with a boiling brain, it was always a relief to be able to walk through the ample grounds of the convention centre to the poster hall. There one could work out a bit by carrying coffee past hundreds of posters. Getting involved in discussions was unavoidable as there were so many interesting topics presented. I also spent hours at my own poster talking to 'customers' who gave me a lot of positive feedback and made helpful suggestions. Working on *Xenopus*, this meeting was a good opportunity for me to see how far other people have progressed in their systems, addressing similar questions.

Though heavily entertained by the meeting programme, I managed to explore some bits of Washington, stuck my nose into the famous museums (to be honest, found most of them a bit stuffy), and jogged along the national mall once a day. In the evenings, discussing one or the other cell biology issue with fellow conferees over a beer (or two), the words "cool stuff" could be heard a lot.

I would like to thank the BSCB for giving me this inspiring opportunity to present my work...

Bernhard Strauss
The Wellcome Trust/Cancer
Research UK Institute
of Cancer and Developmental
Biology,
University of Cambridge
b.strauss@gurdon.cam.ac.uk

Below: 'Single cell injection'



Molecular Regulation of Stem Cells Banff, February 2005

Thanks to a BSCB Honour Fell Travel Award, I attended the Keystone Meeting on Stem Cells in Banff (Alberta, Canada). It was a big and exciting meeting in a fantastic location. During five days, about 40 talks were presented, 300 posters were displayed and two additional workshops competed with the slopes to catch the attention of the more than 600 attendees.

The Fairmont Banff Springs Hotel was like the fairy tale castle we all dreamed about as children. A short distance from the pretty town of Banff, it is an imposing stone building that overlooks the valley and the pine forests. The conference centre is connected to it so that the whole complex embraces a little square dominated by the statue of Baron van Horne fiercely pointing his finger to indicate the way to the stunned scientists. Without his help we certainly would have got lost in the maze of lounges, shops, restaurants and even weddings!

Stuart Orkin (Harvard Medical School, Boston) was the first evening keynote speaker and he introduced some of the recurring topics of the meeting. He showed how many transcription factors play key roles in both the formation of haematopoietic stem cells (HSC) during development and in lineage commitment later on, while different factors seem responsible for stem cell self-renewal. He discussed the analogies between haematopoietic and leukaemic stem cells and the possible mechanisms of leukaemogenesis, as well as the differences between foetal and adult haematopoiesis. He also showed a still uncharacterized subpopulation of non-teratogenic embryonic stem (ES) cells that spontaneously differentiate into muscle fibres. Finally, he introduced the control of pluripotency of ES cells and the still almost totally uncharacterized transcription factor network of which LIF, Oct4 and Nanog must be part.

More data on Nanog, Oct4 and Sox2 were presented by **Shinya Yamanaka** (Kyoto University), who showed how both Sox2 and Oct4 bind a region of the Nanog promoter, but their ability to activate its transcription depends on the acetylation state of this region. He also anticipated the finding of about 20 more genes showing a pattern of expression very similar to Nanog. **Rudolf Jaenisch** (Whitehead Institute, Cambridge, MA) showed how

expression of Oct4 in adult transgenic mice causes the rapid growth of invasive tumours characterized by activated β -catenin and very little differentiation in the skin and the intestine. The tumours regress if Oct4 expression is repressed.

Ihor Lemisckha (Princeton University) presented a genome-wide RNA interference approach for the identification of factors responsible for stem cell self-renewal. The challenge is now to find the targets of all these transcription factors and the mechanisms that regulate their function. He also commented that the whole ES cell culture system may be artifactual, but it is still a great model to look at self-renewal and differentiation. Of a different opinion was Gordon Keller (Mount Sinai School of Medicine, New York), who showed how ES cells in culture reproduce the early stages of development and how, by sorting different subpopulations and following them over time, it is possible to recapitulate the waves of mesoderm differentiation that give rise to haemangioblasts and cardiomyocytes.

James Thomson (University of Wisconsin-Madison) addressed the issue of human ES cell culture and how bFGF and Noggin are able to sustain human ES cell self-renewal. On the last day, Austin Smith (University of Edinburgh) showed that media containing LIF and BMP4 permit the growth of any existent ES cell line in the absence of feeder cells. Both Ronald McKay (NIH, Bethesda) and Austin Smith reported that they are able to generate homogeneous populations of neurons by inducing differentiation of ES cells in vitro. However, while McKay suggested a role for Notch in the asymmetric cell division of neuronal precursors, Smith presented Wnt as an inducer of differentiation.

The role of Notch in regulating stem cell formation and fate determination was discussed in many talks and posters. **Judith Kimble** (University of

By Cristina Lo Celso





Above: Views of Banff Castle and Banff itself.

Wisconsin-Madison) gave a really nice talk on Notch signalling regulating the germ stem cell niche in *Caenorhabditis elegans*. **Leonard Zon** (Children's Hospital, Boston) focused on the fundamental role of Notch signalling in the formation of HSC and showed how, in zebrafish, the longer Notch signalling is activated, the more such cells are generated.

There was much more on HSC. Margaret Goodell (Baylor College of Medicine, Houston) showed the molecular signature of quiescent vs proliferating HSC and introduced the concept of stem cell ageing. In older mice, HSC are more numerous but they are less efficient at repopulating the marrow of recipient mice. HSC derived from old mice express a panel of genes found in other models of ageing and also genes involved in inflammation, while they show a decrease in the expression of DNA repair genes, indicating that stem cells may age like the rest of the body. Irving Weissman (Stanford University) added his considerations on the ageing of HSC and how this phenomenon may be responsible for myeloid leukaemia being more diffuse in elderly people. Sean Morrison (University of Michigan) discussed how ageing of stem cells corresponds to a reduction in their self-renewal capacity, which is regulated by a fine balance between Bmi, p16 and p19.

The epigenetic regulation of stem cell fate was discussed throughout the meeting. Chad Cowan (Harvard University, Boston) introduced the inverse correlation between chromatin remodelling and developmental potency. Rudolf Jaenisch showed how nuclear transfer of somatic cells is an efficient way to generate ES cells only if the somatic nuclei start expressing genes typical of ES cells. Azim Surani (University of Cambridge) talked about epigenetic regulation of germ cells, while Amanda Fisher (Hammersmith Hospital, London) and Brian Hendrich (University of Edinburgh) discussed the role of histone deacetylases in nuclear reprogramming and in the maintenance of the developmental potential of ES cells, respectively.

David Anderson (California Institute of Technology, Pasadena) was the first to talk about the central nervous system. He presented his approach to assessing the multipotency of motoneuron progenitors by sorting them and following their fate both in culture and in transplant assays. This kind of study shows how stem cell fate is regulated by both extrinsic and intrinsic factors.

Fred Gage (Salk Institue, La Jolla) presented some of his recent studies on the mechanisms that regulate adult neurogenesis in the hippocampus. He showed how the small non-coding RNA molecule NRSE triggers neuronal differentiation by interacting with the transcriptional machinery, and also

how in neighbouring cells Sox2 represses while β -catenin/Lef1 promotes NeuroD expression and neuronal differentiation.

Arturo Alvarez-Buylla (University of California, San Francisco) showed how a subpopulation of radial glia cells gives rise to all neural cell types during early postnatal development and also to the astrocytes that sustain neurogenesis in the subventricular zone in the adult brain. Olle Lindvall (University of Lund) described how neurogenesis in the subventricular zone is increased after stroke to generate neuroblasts that migrate in the striatum. Several factors increase the efficiency of this process, but the persistent problem is that most of the newly generated neurons undergo apoptosis, therefore failing to repair the damage. On the last day of the meeting, Jonas Frisen (Karolinska Institute, Stockholm) showed how ephrinA2 and its receptor EphA7 negatively regulate neural stem cell proliferation in the stem cell niche of adult brain. Interestingly, ephrins seem to have the opposite role in the intestine.

Elaine Fuchs (Rockefeller University, New York) discussed the gene expression pattern of epidermal stem cells and described how in the epidermal stem cell niche, the extracellular matrix, the dermal sheath and even the nerves contribute to the quiescence of the stem cells. It will be interesting to examine the changes in gene expression of epidermal stem cells when they exit the quiescent state to promote hair growth or following β -catenin activation.

Fiona Watt (Cancer Research UK, London) discussed how the fate of epidermal stem cells is influenced in different ways by c-Myc and β -catenin and how activation of this last factor in adult transgenic mice is responsible for the formation of ectopic hair follicles containing not only new and functional epidermal stem cells but also melanocyte precursors and neural crest derivative cells.

The last skin-related talk was by **Shin-Ichi Nishikawa** (RIKEN Center for Developmental Biology, Kobe). His group has characterized the melanocyte stem and transit amplifying cells at the single cell level. He showed how c-Kit is necessary for melanocyte formation, Wnt signalling is inhibited in the stem cell niche and involved in the induction of melanocyte proliferation and differentiation, and Notch signalling is necessary for the survival of the melanocytes.

Cristina Lo Celso Cancer Research UK London Research Institute, cristina.locelso@cancer.org.uk



The Biophysical Society Annual Meeting

Despite its name, this huge meeting, held each year in the US, has cell biology as one of its main themes. Interestingly, this meeting attracts many of the leaders in my research field, which is cardiac cell biology and Ca^{2+} signalling. This year, the conference was held in Long Beach, California, helping to increase the attendance to nearly 5000 people.

Fabien Brette
Department of Physiology,
University of Bristol
f.brette@bristol.ac.uk

The meeting is famous for its poster sessions, where around 500 posters are presented every day. That means that you have to focus on what to see and also that formal poster sessions are really busy. However, posters are displayed the evening before, so many delegates start to look at posters then. During the meeting, several symposia are organized, which give the opportunity to hear lectures (25 min) by leaders of the field, while during the platform sessions, short talks (10 min) are given by PhD students and post-docs.

This year, although only one symposium was directly relevant to my field of research, it was extremely interesting. Organized by Clara Franzini-Amstrong (University of Pennsylvania) and Jon Lederer (University of Maryland), the talks focused on the control and regulation of calcium signalling in excitation-contraction coupling. H Cheng (who discovered Ca2+ sparks with Mark Cannell and Jon Lederer) gave a great presentation about local Ca2+ depletion in the sarcoplasmic reticulum of cardiac myocytes. They call these events 'Ca2+ blinks' and showed that they mirror Ca2+ sparks. This area, what happens in the sarcoplasmic reticulum during local Ca²⁺ release, is a hot topic and Eduardo Rios (Rush University, Chicago) presented similar results from skeletal myocytes. Finally, Kurt Beam (Colorado State University), using the state of the art FRET technique, provided new data regarding the conformational coupling of L-type Ca2+ channels and ryanodine receptors in muscle cells.

Several platform sessions were related to my field; for example, **Julie Bossuyt** (from Don Bers' laboratory, Loyola University, Chicago) showed that phospholemman, a small protein regulating Na-K ATPase activity, appears to work in a similar manner to phospholamban regulation of the related sarcoplasmic Ca ATPase. **Marco Weiergräber** (from Tony Schneider's laboratory, University of Cologne, Germany) suggested that a novel sarcolemmal Ca²⁺ channel (CaV 2.3) might be implicated in the regulation of spontaneous heart

beating, although he cannot confirm whether the effect seen was due to the presence of this channel in heart cells or in brain cells (acting on the heart via sympathetic modulation).

I was selected to present my results as a talk during the 'Ca2+ sparks, waves and fluxes' session. Talking about the heart on Valentine's day! It was a little daunting to speak in front of such a large and smart audience, but all seemed to go well and I was asked a lot of questions (constructive and not nasty!). The results I presented were obtained using the technique developed by Prof Clive Orchard (University of Bristol) to study the role of membrane invaginations (transverse tubules) in cardiac cells (acute detubulation) and provided new information about Ca2+ signalling in cardiac myocytes. In the same session, Valerie De Crescenzo (from John Walsh's laboratory, University of Massachusetts) presented interesting data showing that in neuron terminals, Ca2+ release can be activated in a similar manner as in skeletal muscle, via direct coupling of the sarcolemmal Ca2+ channel and the ryanodine receptor. Finally, Jianwei Shuai (from Ian Parker's Iaboratory, University California Irvine) provided evidence that Ca puffs involve the synchronous opening of 25-50 IP, receptors.

My thanks go to the British Society for Cell Biology for the Honor Fell Travel Award, which went towards the costs of my attendance at an exciting and useful meeting.



Right: An early morning in front of the convention centre!

American Society for Microbiology DNA Repair and Mutagenesis

When I heard that my abstract had been accepted for the American Society for Microbiology DNA Repair and Mutagenesis meeting in Bermuda, I was more than a little excited. Not only would I get to enjoy a bit of sunshine in November, but I would have the opportunity to meet and listen to leading scientists from around the world. The seven day conference attracted over 600 participants and 511 posters and 91 talks were presented.

By Jonathan Frampton

The conference commenced with lectures from **Graham Walker** (Massachusetts Institute of Technology), **Priscilla Cooper** (Lawrence Berkeley National Laboratory), **Phil Hanawalt** (Stanford University), **Thomas Lindahl** (Cancer Research UK London) and **Errol Friedberg** (UT Southwestern Medical Center). We were then given a rendition of the 'DNA Repair Blues' with Graham Walker on guitar/vocals and Errol Friedberg on the didgeridoo, an experience I will never forget...however hard I try!

The first full session, on excision repair, started bright and early with a discussion by **Susan Wallace** (University of Vermont) of processing of oxidative DNA base damage. **Keith Caldecott**, my colleague from the Genome Damage and Stability Centre, University of Sussex, gave an excellent presentation on chromosomal singlestrand break repair and neurodegenerative disease. Following a very informative talk by **Cynthia McMurray** (Mayo Clinic Rochester) on OGG1 cooperating with MSH1 in causing DNA expansion underlying neurodegenerative disease, we all adjourned to the beach for lunch, sun-bathing and snorkelling.

The highlight of the afternoon session was the presentation by **Jan Hoeijmakers** (Erasmus University, Rotterdam). From a number of single and double mutant mouse models, he has evidence supporting the idea that oxidative DNA lesions compromise transcription, inactivate genes and trigger apoptosis, and possibly senescence, inducing ageing. On the other hand, lesions or defects in genetic stability mechanisms causing enhanced levels of DNA damage-induced mutagenesis correlate with increased carcinogenesis.

The talks on day two focused on the interplay of repair and transcription; the two that stood out for me were by **John Diffley** and **Helle Ulrich** (both

Cancer Research UK). John spoke about work in Saccharomyces cerevisiae regarding the mechanisms regulating pre-replication complexes; in particular, the novel mechanism by which Cdc6 function is inhibited by the mitotic CDK (Clb2/Cdc28). Helle presented results implying a novel repair role for the post-replication repair proteins Rad18/Rad6/Rad5 distinct from PCNA modification after ionizing radiation.

Steve West (Cancer Research UK) opened the talks on Wednesday with interesting data regarding DNA double-strand break repair by homologous recombination, focusing particularly on Rad51 paralogues. There was a good talk by Penny Jeggo concerning the connection between ATM signalling and DNA double-strand break repair and a short talk by Aidan Doherty on how mycobacterial Ku and Ligase proteins constitute a two-component NHEJ repair machine. Both are colleagues at the University of Sussex.

Thursday was a whole day dedicated to alternative DNA polymerases. My supervisor, **Alan Lehmann**, instigated a long discussion with his very good talk on the structure, function and regulation of DNA polymerase eta by PCNA ubiquitination. In the evening, I presented my poster 'The role of *Schizosaccharomyces pombe* Ubc13 and Mms2 in Response to DNA Damage'. Not only did I receive a lot of useful feedback and new directions for my PhD, I also set up a small collaboration with **Anke Schirer** of Wilfried Kramer's laboratory (University of Göttingen), who presented a poster adjacent to mine entitled 'Involvement of Baker's yeast MPH1 and its Homolog from Fission Yeast in Error-Free Bypass'.

The final day, concentrating on controlling mutations, was opened by **Lorena Beese** (Duke University Medical Center), who showed data suggesting DNA lesions can form mismatches that







evade the polymerase error-detection mechanism, potentially leading to the stable incorporation of lethal mutations. The concluding talk of the conference, was an interesting one by **Judy Campisi** (Lawrence Berkeley National Laboratory) who discussed the functions of BLM and WRN during S phase.

Aside from the talks and posters, there was a great deal of opportunity to meet and discuss science with a range of people, from fellow PhD students to professors. The evening events and the local 'Dark and Stormy' drink helped to stimulate relax-

ing scientific discussions, which often went on long into the night and sometimes even into the hot tub! The conference banquet was a huge success, the open bar and disco certainly went down very well.

The whole ASM Bermuda conference was an incredibly useful and enjoyable experience, and my thanks go to the British Society for Cell Biology for the Honor Fell Travel Award which allowed me to attend.

Jonathan Frampton
Genome Damage and Stability Centre, University of
Sussex. J.M.Frampton@sussex.ac.uk

Above, from left to right: The beach in November; late scientific discussions in the hot tub; and snorkelling with a pufferfish.

Cell interactions in development and disease

Hyderabad, India

I attended this meeting at the Centre for Cellular and Molecular Biology, Hyderabad, which featured more than 28 speakers from all around the world. Some talks were quite basic and descriptive, so very easy to follow. The lecturers included Alex Hajnal, Ben-Zion Shilo, Sarah Bray, Carl-Henrik Heldin, Juergen Knoblich, Mariano Barbacid and Matthew Freeman.

There were more than 50 posters, most of them on *Drosophila* cell biology. I presented my poster 'Development of *Drosophila* model system: Nemaline Myopathy'. One thing I noticed in this meeting was that the students were given more oppportunities to present their work than is sometimes the case. The posters were displayed

throughout the meeting and lunch was arranged in the poster area, hence I had a really good chance to talk to people and get helpful suggestions and criticism regarding my work.

Apart from the academic activities, there was a very interesting cultural programme which enlivened our three days. Folk dance and music were performed on the stage by local artists and students of the Centre. Overall, I benefited greatly from this meeting and I am grateful to BSCB that my travel expenses were supported.

Vikash Kumar, Dept of Biology, University of York vk10@york.ac.uk

Keystone: Cell Migration and Adhesion Salt Lake City, April 2005

Thanks to an Honor Fell Travel Award from the BSCB, I was lucky enough to be able to attend the April 2005 Keystone meeting on Cell Migration and Adhesion in the beautiful Snowbird Resort near Salt Lake City in Utah, USA. I arrived at the resort early and decided to make the most of the extra time by taking to the slopes and exploring the amazing mountain. As I arrived and set out with my snowboard, the sun was shining but soon the clouds came in and it began to snow, resulting in a dramatic decrease in visibility. This, combined with a range of steep ski runs and my complete lack of knowledge of the area, led to an interesting experience attempting to navigate my way down the mountain! Still I persevered and managed to find a way down in time for the meeting.

By Sam Passey

The Keynote Address by John Condeelis (Albert Einstein College of Medicine, New York) was an exciting talk about his research into the contribution of actin polymerizing pathways in vivo to tumour invasion and metastasis. His lovely movies illustrated the use of multiphoton microscopy in whole living animals to study the migration of cells in tumour invasion, allowing direct visualization of the migration of macrophages and carcinoma cells in a mouse model of mammary carcinoma.

After a delicious buffet breakfast the following morning, we began in earnest at 8 am with the *Cellular Protrusions* session, chaired by **Gary Borisy** (Northwestern University Medical School) who also presented the first talk. He described the use of correlative light and electron microscopy to allow direct comparison of the last known actions of a cell with the structure of the actin cytoskeleton observed by scanning electron microscopy of the same cell. This technique has allowed visualization of different actin structures within the cell, including lamellipodia and filopodia, and comparison of the actin arrangement with different morphologies or cell activities.

The morning session was followed by a break until 4.30pm which allowed people the opportunity to participate in a range of activities available in the resort, not only the obvious skiing and snowboarding but also heated outdoor swimming pools and spas, including the world renowned Cliff Spa where visitors could pamper themselves with massages, facials and skin treatments along with yoga and pilates classes.

The evening *Cell Adhesion* session began with a talk by **Alan Rick Horwitz** (University of Virginia, USA) on 'The Regulation of Adhesion Turnover and Protrusion'. He discussed the work of his lab on the adaptor protein GIT-1, which is proposed to be involved in mediating Rac activation at the leading edge of migrating cells by promoting the formation of a signalling complex containing Rac activators and effectors and also through signalling to Arf6.

The Cell Polarity session kicked off with Peter Devreotes from Johns Hopkins University School of Medicine presenting work on the directional sensing role of the lipid, phosphatidyl inositol (3,4,5) triphosphate (PIP3). He showed that by using latrunculin to inhibit the motility and polarity aspects of cell migration, the directional sensing aspects can be visualized as a concentration of PIP3 at the membrane in a crescent at the point nearest the high end of the gradient of what is being sensed. A corresponding increase in PTEN, which degrades PIP3, at the membrane near the low end of the gradient allows for directional sensing and regulation of cell protrusion to establish polarity.

A short talk by **William Wood** (Instituto de Medicina Molecular, Lisbon, Portugal) presented spectacular in vivo imaging in *Drosophila* for the study of macrophage chemotaxis towards a wound. He concluded that these cells migrate to the wound site in response to a chemoattractant gradient generated by the action of phosphoinositol 3-kinase (PI3K), and that this migration is inhibited by PI3K inhibitors. His work also pointed to PI3K independence for migration of cells during development. Both types of migration were



dependent on the Rho GTPase Rac, indicating that although the processes are similar in some aspects they arise in response to different signalling cues.

The afternoon session on Signalling Pathways featured an interesting talk by **Mark Ginsberg** (University of California, San Diego, USA) about integrin signalling in cell migration. He described a nice model for how $\alpha 4$ integrins and paxillin play a role in maintaining cell protrusion by inhibiting Rac activation on the lateral edges of the cell but not at the front, so allowing protrusion for migration at the leading edge whilst inhibiting protrusion along the sides.

To end the day, **Ronald Vale** (University of California, San Francisco, USA) gave a short talk about large-scale genomic screens in *Drosophila* in which he used high resolution microscopy to screen for cytoskeletal phenotypes in cells treated with RNAi. A number of actin-based morphologies have been identified using this technique and the genes responsible can then be identified.

Each evening was taken up with a social hour with food and a free bar followed by the poster sessions which were interesting and very well attended. I showed my poster on the Monday evening and found it an intense but also very enjoyable and informative experience.

The Tuesday morning session on *Cytoskeletal Dynamics* featured interesting talks on a variety of subjects ranging from unconventional myosins by **Margaret Titus** (University of Minnesota, USA) to the role of cortactin in cell motility by **John Cooper** (Washington University, USA). **Gregg Gunderson** (Columbia University, New York) presented exciting work on the role of microtubules in cell migration and the regulation of microtubule reorganization by Rho GTPases. He showed fascinating images illustrating how the cells at the leading edge of a migrating epithelial sheet become polarized as the nucleus is dragged backwards and

the microtubule organizing centre becomes positioned in front of the nucleus.

Tuesday afternoon featured a special session hosted by Alan Rick Horwitz and J. Thomas Parsons (University of Virginia Health Science Centre, USA) to introduce the Cell Migration Consortium. This involves approximately 35 investigators in 16 institutions at present: it is not an exclusive club but an open organization. It was

established with the aim of identifying and overcoming barriers to migration research by facilitating the development of new collaborations and encouraging the sharing of information and technology in this field. The website at www.cellmigration.org details the ongoing work of the consortium and the reagents developed so far. Much progress has been made and the consortium is sure to prove an invaluable resource in the future.

The evening session on Tuesday focused more on migration of cells in vivo, concentrating on epithelial cell migration. Mark Peifer (University of North Carolina, USA) chaired the session and presented an excellent talk about epithelial morphogenesis in Drosophila in which he highlighted the role of Ena and Abl in the regulation of dorsal closure. This theme was followed up on Wednesday with two awe-inspiring sessions on Axon Guidance and Migration In Vivo. The morning session was highlighted for me by a talk from Frank Gertler (MIT, Boston, USA) on the role of Ena/VASP proteins in axon guidance, whilst the afternoon Migration In Vivo session was packed with exciting in vivo imaging from the likes of Denise Montell (Johns Hopkins School of Medicine, New York) and Ruth Lehman (New York University, USA) who both presented lovely live images of Drosophila embryos. Paul Martin (University of Bristol, UK) gave an amusing and visually stunning talk on 'Live Studies of Wound Healing and Inflammation', a topic beautifully introduced by William Wood earlier in the week.

The meeting ended with the usual social hour followed by an entertainment event with a DJ. Much fun was had by all and the salsa music seemed to be particularly popular. Overall, I think the meeting was a fantastic success and very well organized by Frank Gertler and Denise Montell. I enjoyed every minute, learned a lot and met some lovely people – and the snow was great too!

Sam Passey
Department of Biochemistry,
University of Bristol
Sam.Passey@bristol.ac.uk

BSCB/BSDB Joint Spring Meeting

Warwick University, 6–9 April 2005

This Spring, the BSCB returned to Warwick and once again organized a Joint Meeting with the British Society of Developmental Biology. The BSCB meeting was larger than usual, with nine sessions, loosely based around a theme of 'The Asymmetric Cell'. There were 425 participants and 139 posters were presented (see page 2 for the winners), plus two special lunches, following the precedent established last year. This report was contributed by several 'volunteers' who were pressed into service at the last minute.

Session 1: mRNA localization

Ilan Davis, who had built a session where the talks nicely supported each other, chaired the session on mRNA localization. The first word was given to Robert Singer (Albert Einstein College of Medicine, New York), who discussed work based on a clever construct that makes it possible to monitor an RNA's entire path from synthesis to localization in real time. He discussed mathematical models for transcription and movement of RNA. On the controversial subject of mRNA movement within the nucleus, his results supported a diffusionbased model. It has long been known that the mobility within the nucleus is decreased under conditions of energy starvation, but Robert neatly showed that this is not because mRNA movement is an energy-based process. The mRNAs were still 'buzzing' around but under extremely restricted conditions imposed by the expanded DNA.

Isabel M Palacios (University of Cambridge, UK) discussed the field her newly started lab is addressing. Her interest is the mechanisms of kinesinmediated transport during Drosophila oogenesis. Khc is required for localization of oskar mRNA to the posterior of the oocyte. However, it is still not clear whether oskar is localized in a Khc-driven complex or moves by the cytoplasmic flow generated by Khc. Khc co-localizes with the oskar mRNA to the posterior crescent, but a direct link between Khc and oskar mRNA or the cytoplasmic flow remains unestablished. None of the obvious Khc adaptors, Sunday driver or kinesin light chain, is required for these processes. This leads to the question: how is the kinesin-dependent transport in the oocyte mediated?

The main focus of **Anne Ephrussi's** (EMBL, Heidelberg, Germany) talk was the discovery that components of the exon–exon junction complex

are required for oskar localization in the *Drosophila* oocyte. This was surprising as oskar mRNA localization had not previously shown requirements for splicing. Her lab has now discovered that an oskar mRNA from a cDNA construct could not localize correctly in oskar null oocytes but mRNA from a genomic oskar construct containing introns could. Thus, splicing is essential for oskar mRNA localization. This work also showed that splicing of the first oskar intron is required and sufficient for its localisation. Finally, Anne described how the 3' UTR of oskar mRNAs associates into higher order complexes.

Alejandra Clark and Ilan Davis (both University of Edinburgh) discussed localization of another mRNA in the *Drosophila* oocyte – gurken. Alejandra Clark had carried out challenging experiments injecting fluorescently labelled RNA and tracking its movement from the nurse cells into the oocyte and within the oocyte cytoplasm to the nucleus at the dorsal anterior corner. Her results support a model in which there are two populations of microtubules within the oocyte: one orientated in an anterior to posterior manner and another orientated from the dorsal anterior corner towards the ventral side. She then showed results suggesting that grk mRNA is actively transported by dynein from the nurse cells to the oocyte during mid-oogenesis.

llan Davis described the work of a postdoc in his lab, **Veronique Van De Bor**, identifying a consensus RNA signal in the coding region of grk and the I factor retrotransposon that directs both mRNAs to a dorsoanterior crescent near the oocyte nucleus. Their data suggest that I factor transposition causes a disruption of grk mRNA localization by competing for shared mRNA localization factors.

Sponsors

The BSCB is very grateful to all the sponsors who helped to make Warwick such a fascinating and informative meeting: The Company of Biologists for their overall support; Science Press who provided the drinks for the poster reception; Garland Press and Cancer Research UK who supported two of our speakers, and the American Society of Cell Biology, Nature Publishing Group and Current Biology who donated poster prizes.

Then Ilan Davis described the work of another postdoc, Renàld Denaloue, who has been studying the mechanism of localization and anchoring of pair-rule mRNAs to the apical cytoplasm in the Drosophila blastoderm embryo. The lab's previous results showed that the localization involves Dynein-based transport along microtubules. Their new data show that apical anchoring also requires Dynein, but in this case the motor acts as a static anchor, rather than being required for continuous active transport.

Simon Bullock (MRC-LMB, Cambridge, UK) went into more depth with the actual tracking of the pair-rule mRNAs. His results support the idea that the mRNA localization complexes contain both microtubule plus- and minus-end directed motors and that all mRNAs are tethered to these complexes. Simon Bullock further suggested that the overall direction of motility of the mRNA localization complexes might be modified by interaction between the motor complex and the localization sequences within the mRNA.

Katja Dahlgaard;The Wellcome Trust/Cancer Research UK Gurdon Institute, University of Cambridge. k.dahlgaard@gurdon.cam.ac.uk

Session 2. Neural stem cells

The neural stem cell session was opened by Charles ffrench-Constant (University of Cambridge) who gave an overview of how integrins and their ligands in the extracellular matrix regulate growth factor signalling to provide precise temporal and spatial control within the stem cell niche. He also described how in tenascin-C deficient mice, neural stem cells show reduced sensitivity to FGF-2 and enhanced activity in response to BMP-4. He illustrated the expression of laminins within the neural niche and demonstrated their roles in maintenance.

Jun-An Chen (Wellcome/CRUK Gurdon Institute, Cambridge) described a novel cell type-specific cyclin (cyclin Dx) that is required for maintaining ventral neuronal progenitors in the spinal cord. He suggested that motor neuron progenitors differentiate prematurely when the concentration of cyclin Dx falls. These results support the hypothesis that the coordination of cell proliferation and cell fate determination is regulated by cell cycle components.

It was a pity that Magdelena Gotz (Max Planck Inst Neurobiology, Germany) was ill and could not come to this meeting. However, a post-doc from her lab presented evidence of how Pax6 plays a master role in the control of neurogenesis. He showed that neurogenesis becomes fully Pax6-dependent in the neurosphere culture system, independent of the region of origin, and that Pax6 overexpression is sufficient to direct almost all neurosphere-derived cells towards neurogenesis.

Kate Lewis (University of Cambridge) described the advantages of using zebrafish to study ventral interneuron specification and patterning. Many of the transcription factors (Evx1, Eng1b, Chx10) implicated in interneuron specification in amniotes are also expressed in the embryonic zebrafish spinal cord, suggesting that the mechanisms of interneuron specification are conserved across vertebrate species. She also showed a transgenic line of zebrafish in which GFP is expressed in cells that express Pax2, a ventral interneuron transcription factor. This tool will be very useful for future functional studies.

Derek van der Kooy (University of Toronto, Canada) showed how primitive neural stem cells are formed directly from single ES cells in a manner dependent on exogenous LIF and endogenous FGF. Embryonic stem cells quickly acquire neural identity and give rise to neurons and glia in minimal culture conditions. Moreover, experiments in vivo with mouse chimeras reveal that these primitive ES-derived neural stem cells have a broad range of neural and non-neural lineage potential. These results support a model whereby definitive neural stem cell formation is proceeded by a primitive neural stem cell stage during neural lineage commitment.

Finally, Wieland Huttner (Max Planck Institute, Dresden, Germany) demonstrated how it is possible to distinguish between proliferating and neuron-generating neuroepithelial cells using the anti-proliferative gene TIS21. Using time-lapse microscopy of neuron-generating divisions of neuroepithelial cells in a transgenic TIS21-GFP mouse embryo, he discovered the existence of a novel neuronal progenitor dividing at the basal side of the neuroepithelium. In addition, he described using prominin-1 to define the symmetric and asymmetric distribution of apical plasma membrane during proliferating and neuron-generating divisions of neuroepithelial cells.

Jun-An Chen
The Wellcome Trust/Cancer
Research UK Gurdon Institute and
Department of Zoology, University
of Cambridge.
jc393@cam.ac.uk

Session 2. Polarized Secretion of Endocytic Organelles

One of the fantastic things about the BSCB spring meeting is the broad range of topics covered and the opportunities this presents to discover (or rediscover) exciting areas of research that unfortunately one never seems to have the time to keep up with. Thursday presented me with such an opportunity and also a dilemma: which session should I choose? Finally plumping for Polarized secretion of endocytic organelles, I headed over to social sciences to see what I could learn.

The session was chaired by Gillian Griffiths (University of Oxford, UK) who kicked off with a fantastic account of how T lymphocytes achieve polarized secretion, allowing them to kill target cells. Brilliantly, Griffiths has been able to exploit clinical samples to get a handle on the process. She outlined both what this had taught us about players in the biological processes and the understanding this conferred of clinical aspects of the syndromes, highlighting how much can be gained by the availability of clinical samples to the research community.

In a short talk, Alistair Hume (Imperial College London) then gave us a summary of the melanocyte assay he has been using for his research and insights it has provided into the role of melanophilin in melanosome transport. Next, Phillipe Chavrier (Institut Curie, Paris) gave an excellent account of his work on membrane

delivery to the cell surface during phagocytosis and of the interplay of formins and arp2/3 in actin dynamics. **G. Michaux** (University College London) followed with an outline of his functional analysis of P-selectin trafficking in endothelial cells.

After a coffee and biscuit pit-stop, I heard Susan Eaton (Max-Planck-Institute, Dresden, Germany) address a packed audience. She spoke of how gradients of lipid-linked morphogens are achieved during Drosophila development. I was intrigued by her research on argosomes - membranous particles that may play a role in the process. These particles sounded fantastic - a novel solution to an old question. Eaton went on to present progress she is making in dissecting the argosome which highlighted just how difficult some questions are to address and yet how with some ingenuity and determination we can move forwards. Last, but definitely not least, was Ira Mellman (Yale, New Haven, USA) who wowed us with some fantastic images of endocytosis in action. She demonstrated that with careful analysis of such data we can get a crucial understanding of the processes in question.

As the session ended, I reflected on the fantastic opportunity I had been afforded. I had hoped to get an insight into this topic unfamiliar to me and had been lucky enough to spend the afternoon listening to cutting edge research by world class scientists. Not something I have the luxury of doing everyday!

Nina Peel, Wellcome CRUK Gurdon Institute, Cambridge. np257@hermes.cam.ac.uk

Session 3. Neuronal transmitters in health and disease

Bruno Goud (Institut Curie, Paris) started this session with evidence on the involvement of Rab6 isoforms, Rab6A and Rab6A', in mitosis. His data indicated that these two isoforms act in two different pathways regulating the metaphase/anaphase transition and cytokinesis. The Rab6A' pathway involves the interaction of this protein with the P150Glued subunit of the dynein/dynactin complex in a Mad2-dependent manner. This interaction results in the activation of the complex at the kinetochores and the inactivation of the Mad2-spindle checkpoint. Rab6A, on the other hand, interacts with Rabkinesin-6 (RK6) independently of Mad2 activity.

Andrew Grierson (University of Sheffield) introduced a zebrafish model in which the GTPase guanine exchange factor 'alsin' has been knocked down. Alsin mutations in humans cause a rare autosomal recessive form of amyotrophic lateral sclerosis (ALS2). Andrew presented data indicating 50–60% identity at the amino acid level between the zebrafish and human alsins and 75–86% amino acid identity in the Rab5 and RhoGEF domains. A role for this protein in development was proposed based on the observed defects in morphology and motility in the alsin knock-down zebrafish. As alsin is expressed in neurons and not in muscle during development, Andrew suggested the observed defects could be caused by primary neuronal defects and supported this with data on impaired neuronal migration in this model system.

Giampietro Schiavo (Cancer Research UK, Lincolns Inn Fields) described his laboratory's findings on the mechanisms of tetanus neurotoxin (TeNT) transport in mammalian motor neurons. His data indicated that TeNT is retrogradely transported in a mixed population of tubules and vesicles in which, unlike most other vesicles, the pH is neutral. This group have developed a novel method utilizing poly-cysteine-tagged TeNT Hc (a fragment of the tetanus toxin) conjugated to magnetic beads. Results using this method implicate the small GTPase Rab7 in regulating the retrograde transport in mammalian motor neurons; a hypothesis supported by evidence on the co-localization of Rab7-GFP on TeNT Hc carriers in the axons and soma of motor neurons, and by specific inhibition of the retrograde transport by a dominant negative form of Rab7.

Majid Hafezparast (University of Sussex) highlighted the importance of axonal transport in the maintenance and survival of motor neurons by presenting data on the retrograde axonal transport defect in the legs at odd angles (Loa) mouse. This mouse has a mutated cytoplasmic dynein heavy chain (DNCHC1) in the overlapping homodimerization and intermediate-chain binding domains. Using fluorescently labelled TeNT Hc, Majid showed how this mutation impairs the fast component of dynein-mediated retrograde transport in motor neurons. His data on impaired reassembly of the Golgi in mouse embryonic fibroblasts after disruption with cold and nocodazol illustrated the similarity between the Loa mouse and the human motor neuron disease, ALS. Thus, the observed Golgi fragmentation observed in motor neurons of ALS patients may be caused by disruption of the

dynein function as a result of some form of stress in these neurons.

Folma Buss (University of Cambridge) continued the discussion on motor proteins. She presented data on myosin VI, an actin-associated motor protein. Using siRNA knock down, overexpression of dominant negative myosin VI, and cell lines derived from the myosin VI knockout mouse, she provided evidence for the involvement of this motor protein in multiple pathways in endocytic and exocytic trafficking. She also described a novel binding partner for myosin VI in the Golgi and showed that the Golgi is disrupted when the expression of this novel protein is knocked down with siRNA.

Finally, Mike Fainzilber (Weizmann Institute of Science, Israel) presented his laboratory's data on the retrograde signalling response to nerve injury. He provided evidence that dorsal root ganglion neurons dissected from rats with a crushed sciatic nerve exhibit process elongation after a few days in culture because of signalling from the crush site or block of signal by the crush. He showed how injury results in local translation and increased concentrations of importins and vimentin at the site of injury. He then described some elegant experiments showing the interactions of these factors with dynein to form a retrograde injury signalling complex, which transports kinase signals over long intracellular distances protected from phosphatases.

Majid Hafezparast, Department of Biochemistry, School of Life Sciences, University of Sussex. m.hafezparast@sussex.ac.uk

Session 3. Asymmetric Cell Division

The session on asymmetric cell division was chaired by Jürgen Knoblich (IMP, Vienna), who started by talking about polarization of recycling endosomes during asymmetric cell division in the Drosophila nervous system. The hallmark of asymmetric cell division is segregation of cell fate determinants, the first of which to be identified was Numb. In the endocytic pathway, recycling endosomes are generated and accumulate around the centrosome of only one of the daughter cells. Rab11 is the marker for these recycling endosomes and is suppressed in cells that do not inherit Numb. Rab11 binds Nuf, a centrosomal protein that binds and accumulates on only one of the centrosomes. Nuf and Numb act redundantly in asymmetric cell division.

Rita Sousa-Nunes (King's College, London) described a mutant obtained in a screen to identify new genes involved in the asymmetric division of the *Drosophila* neuroblast. This mutant has the intriguing phenotype of enhanced detection of centrosomal Miranda. Continuing the studies on *Drosophila*, François Schweisguth (Ecole Normale Superieure, Paris) spoke about Neuralized, which, along with Numb, regulates Notch-mediated binary fate decisions. Bearded is a partner of Neuralized; overexpression and deletion experiments suggest that negative regulation of Neuralized by Bearded is at least partly responsible for the spatially restricted distribution of Delta (Notch ligand).

Arwen Wilcock (School of Life Sciences, Dundee) outlined a strategy to build extensive maps of cell lineage using electroporation of the spinal cord of chick embryos with GFP tubulin, followed by time-lapse 3D imaging. After the coffee break, Pierre Gönczy (ISREC, Switzerland) described the importance of G protein signalling pathways for asymmetric cell division in *C. elegans* embryos.

Finally, Magda Zernicka-Goetz (Gurdon Institute, Cambridge) presented a non-invasive lineage tracing study of the early mouse embryo. The aim is to determine whether development of blas-

tocyst pattern shows any correlation with the orientation and order of the second cleavage divisions that result in specific positioning of blastomeres at the 4-cell stage. The results suggest that the spatial arrangement of individual 4-cell stage blastomeres and the order in which they are generated correlate with blastocyst pattern in the mouse embryo.

Teresa Barros
The Wellcome Trust/Cancer Research UK Gurdon
Institute, University of Cambridge.
t.barros@gurdon.cam.ac.uk

Session 4: Cell Biology of behaviour

The session on the cell biology of behaviour featured some great examples on how model organisms can be exploited to analyse the cellular and molecular features underlying behaviour. Focusing on the function of neuromodulators such as serotonin and dopamine in *C. elegans* (which alter their locomotive behaviour and slow down in response to serotonin), **Stephen Nurrish** (University College London) shed light on the downstream pathways involved in this behavioural response. He showed that the activity of DGK-1, a common component of the cellular response to serotonin and dopamine, is regulated by binding to Rho-1, and that altering the levels of Rho-1 results in altered locomotive behaviour.

Nurrish's co-worker **PR Morrison** expanded on the topic of DGK-1-interacting proteins by describing the function of KIN-4, a newly identified binding partner. Mutations in this protein partly mimic the dopamine resistance seen in dgk-1^{-/-} animals.

Graeme Davis (UCSF, USA) reported on the mechanisms involved in synapse stabilization and disassembly in *D. melanogaster*, using an elegant visual assay of localized synaptic retraction, the 'postsynaptic footprint'. In this, the remnants of postsynaptic structures, present even after synapse retraction, are immunolabelled and provide a quantitative means of analysing the effect of mutations on synapse stability. Using this, Graeme demonstrated the function of retrograde BMP signalling and dynactin retrograde transport in this system.

The following two talks concentrated on the signalling that underpins bipolar disorder, for which all current treatment options have been discovered serendipitously and have undesirable side-effects. RSB Williams (University College London) demonstrated in a stimulating short talk how the InsP3-regulated life cycle of the soil amoeba Dictyostelium discoideum can be used to screen for the InsP3-depletive effect of newly designed drugs structurally related to valproic acid. This is a widely used drug that has teratogenic effects. The new candidate substances might improve the treatment of bipolar disorder. Anne Mudge (University College London) extended the investigation of the role of InsP3 signalling in bipolar disorder to conventional antidepressants and showed that these affect InsP3 levels when administered in combination with mood stabilizers.

Finally, Mario de Bono (MRC Laboratory of Molecular Biology, Cambridge) returned to the flatworm and topics already touched on by Cori Bargmann in her BSDB keynote lecture, analysing the difference between roaming and dwelling strains, and identifying two pathways involved in the regulation of aggregative or dispersing behaviour — the first being responsive to food cues, the second responding to oxygen levels, leading to an integrated behavioural response that also takes into account global neuroendocrine signals.

Uli Foster, Department of Pathology, University of Cambridge, buf20@cam.ac.uk

Women in Cell Biology lunch

This lunch addressed the particular challenges faced by women in Cell and Developmental Biology in pursuing academic careers in the UK. In particular, we discussed possible solutions to the 'Leaky Pipeline', i.e. while biology attracts a majority of female undergraduates, there is only a small percentage of female lecturers and professors. This translates into a significant loss of talent for the scientific community as a whole. We had three very entertaining and inspiring talks from successful women who have followed different career paths: Professor Scottie Robinson is a Wellcome Principal Research at the University of Cambridge, Dr Michele West is a career postdoc at the University of Dundee and Dr Helen Arthur held a Wellcome Return-to-Work Fellowship before obtaining a permanent position at the University of Newcastle. Following their presentations, there was a lively discussion with the audience, which included issues such as the possibility of establishing a better career structure for long-term postdocs, being a scientist and being a Mum, and what organizational changes could be implemented in the workplace and at conferences to facilitate women.

Liz Smythe University of Sheffield e.smythe@sheffield.ac.uk

The presenters have kindly made their powerpoint slides available; if anyone would like to see these, please contact Joan Marsh (jmarsh@wiley.co.uk).

Session 4: Micro-RNAs

The micro-RNAs session was devoted to small, noncoding RNAs that regulate gene expression at the post-transcriptional level. It was opened by Steve Cohen (EMBL, Heidelberg), who described a combined experimental and computational approach to study genome-wide micro-RNA functions. Given the large number of micro-RNA-encoding genes (over 100 in Drosophila), the time that would be required for functional analysis by genetics alone has prompted the use of computational methods to infer potential roles for these genes. Although the variability in base pairing makes it hard to predict the identity of candidate targets for micro-RNAs, Steve described how comparisons between known micro-RNA targets reveal that base pairing is more consistent at the 5' end and this 'seed' region appears to contain most of the important information, whilst the targeting of micro-RNAs to a suite of genes with related functions facilitates functional annotation.

Jan Rehwinkel (EMBL, Heidelberg) then described a genome-wide analysis of RNAs regulated by Drosha and Argonaut proteins in Drosophila, using microarray expression profiles. Of the various transcripts upregulated when these proteins were depleted, most were known to be involved in axon guidance, cell adhesion, organogenesis or apoptosis (including the validated micro-RNA targets hid and reaper).

The role of RNAi in transposon silencing was explored by Ron Plasterk (Hubrecht Laboratory, Utrecht), who pointed out that even though there are multiple copies of transposons in the Caenorhabditis elegans genome, none of these are mobile in the germline. However, in 'mutator' mutants (which lose the activity of genes owing to

the aberrant activation of a subset of transposons in the germline), it was found that RNAi was also defective, suggesting that RNAi might protect the genome against transposon activity. Describing RNAi as the 'immune system of the genome', Ron pointed out how the amplification of RNAi signals might be compared to clonal selection, given that a brief episode of RNAi activity may lead to stable germline gene silencing that is heritable over 30 generations! Changing track, Ron then highlighted how the sequencing of micro-RNAs from a host of primates might facilitate the discovery of new micro-RNA genes through 'phylogenetic shadowing' and described ongoing functional studies of micro-RNAs in zebrafish development.

The role of micro-RNAs in C. elegans development was picked up by Eric Miska (Gurdon Institute, Cambridge), who described a combined functional genomics approach involving GFP expression studies and the generation of knockout mutants. In this way, the lin-4 micro-RNA and four members of the evolutionarily conserved let-7 family were shown to yield heterochronic phenotypes in mutants. Eric then described the downregulation of micro-RNAs in primary human tumours. The session was concluded by David Baulcombe (Sainsbury Laboratory, Norwich), whose presentation focused on the role of siRNAs in chromatin silencing in Arabidopsis. He related how enhanced and reduced silencing phenotypes were observed in a host of mutants for homologues of RNA processing enzymes, presumably by affecting the turnover of RNA sequences entering the RNA silencing pathway and their subsequent direction of sequence-specific epigenetic modifications.

Neville Cobbe
Wellcome Trust Centre for Cell
Biology, University of Edinburgh.
NCobbe@hgmp.mrc.ac.uk

Session 5. Regulation of Cell Death

Programmed cell death is an essential event for normal development and maintenance of tissue homeostasis. Apoptosis is the major form of programmed cell death in metazoans and it is controlled mainly by the activity of three families of proteins: the Bcl-2 family, inhibitor of apoptosis proteins (IAPs) and caspases, the cellular 'bulldozers' responsible for the destruction of the cell.

Caspases are proteases that cleave target proteins specifically after aspartate residues. Although hundreds of putative caspase substrates have been identified in vitro, few have been validated in vivo.

Seamus Martin (Trinity College, Dublin) reported the identification of evolutionarily conserved caspase substrates. By analysing putative caspase substrates from *C. elegans* to humans, he was able to confirm previously described substrates, as well as expose new targets. Since these caspase substrates are evolutionarily conserved, it is likely that their cleavage during cell death is significant. While caspases trigger the onset of apoptosis, the morphological changes of the dying cell are thought to depend on actomyosin forces. However, Jon Lane (University of Bristol) showed new evidence implicating microtubules in the packaging of cellular fragments into

apoptotic bodies. Although microtubules are rapidly depolymerized during the initial stages, they are subsequently replaced by rigid microtubule bundles that are necessary for directing condensed chromatin into apoptotic bodies.

New insights into the mechanism of caspase regulation by IAPs were presented by **Pascal Meier** (Institute of Cancer Research, London). The current dogma states that IAPs act as inhibitors that neutralize caspases by binding to their active site pockets. Pascal's data challenge this view, as mammalian c-IAP1 and the *Drosophila* DIAP1 inhibit caspases through a distinct mechanism. During processing from the zymogenic to the fully active form, caspases expose an IAP-binding motif similar to that of IAP antagonists. Surprisingly, the IAP-bound caspase remains catalytically active and after binding it cleaves the IAP. This targets the IAP for proteasomal degradation leading to co-degradation of caspases.

Luis Miguel Martins (MRC Toxicology Unit, Leicester) presented new data on Omi/HtrA2 knockout mice. Omi/HtrA2 is a mitochondrial serine protease that also fuctions as an IAP-antagonist when released into the cytoplasm. Interestingly, Omi/HtrA2 knockout mice did not show an apoptosis-related phenotype. Instead, they showed a parkinsonian phenotype, indicating that Omi/HtrA2 is neuroprotective. Thus, Omi/HtrA2's main function is to maintain mitochondrial integrity in specific subsets of neurons, rather than to relieve IAP-mediated inhibition of caspases.

Doug Green (La Jolla Institute for Allergy and Immunology, San Diego, USA) presented surprising results on caspase-independent cell death. By conducting a functional screen with a retroviral library, he searched for genes that confer resistance to death when cells are triggered to die but caspases are blocked. Surprisingly, GAPDH allowed cells to survive in the presence of caspase inhibitors following apoptosis induction. Although the mechanism by which GAPDH accomplishes this is obscure, it is conceivable that GAPDH allows cells to maintain their energy levels while mitochondria are repaired.

Programmed cell death was first described about 150 years ago but, as seen in this meeting, crucial questions are still unresolved and there is a lot more going on in dying cells that one might expect.

Paulo Ribeiro
The Breakthrough Toby Robins
Breast Cancer Research Centre,
Institute of Cancer Research
Paulo.Ribeiro@icr.ac.uk

Session 5. Mitosis

The Saturday Mitosis session could have been regarded as 'the morning after the night before', coming as it did after an evening of food, wine and salsa dancing. Instead, the quality of the speakers ensured this series of talks was one of the most interesting of the meeting. Tony Hyman (Max-Plank-Institut, Dresden, Germany) opened with an excellent talk describing the function of Xenopus TACC3 in regulating microtubule dynamics. He showed that TACC3 interacts with XMAP215, enhancing the ability of the latter to stabilize microtubules, and that Aurora A is responsible for phosphorylating and activating TACC3 specifically at the centrosome. He went on to suggest a model in which stabilization of microtubules at the centrosome through Aurora A, TACC3 and XMAP215 would allow centrosomes, when present, to become the dominant site of microtubule growth.

Monica Bettencourt-Dias (University of Cambridge) presented work carried out in David Glover's lab investigating the function of two Drosophila kinases involved in cell cycle progression: PvrR, a homologue of the PDGF/VEGF receptor, and SAK, a member of the MAPK family. Iain Porter (University of Dundee) described a biochemical approach leading to the identification of 213

proteins unique to mitotic chromatin in *Xenopus*. He showed that one of these, Fam44, localizes to the outer part of kinetochores during mitosis and is involved in aligning chromosomes on the metaphase plate. **Yixian Zheng** (HHMI Carnegie Institute, Baltimore, USA) provided more evidence for the function of Fam44 in her excellent talk. She showed that Fam44 is capable of de-ubiquitinating the chromosomal passenger protein Survivin on a conserved Lysine (K64). Suprisingly, evidence from her lab suggests that ubiquitination does not target Survivin for proteolysis, but instead regulates the localization of Survivin and Aurora B to kinetochores.

The session ended with a talk by Jordan Raff (Gurdon Institute, Cambridge), who showed that the pericentriolar material of the centrosome is dynamic and that its structural integrity in Drosophila is regulated by the Pericentrin-like protein, D-PLP. Cells lacking D-PLP are able to form spindles and undergo mitosis, but the recruitment of the pericentriolar material is disrupted. As this phenotype can be reversed when microtubules are depolymerized, Jordan suggested that D-PLP normally functions to counteract the microtubule-dependent dispersal of the pericentriolar material.

James Wakefield, Department of Zoology, University of Oxford, james.wakefield@zoology.oxford.ac.uk

Hooke Medal Lecture

This year the Hooke Medal was awarded to Frank Uhlmann (CRUK, London), whose work on chromosome segregation in yeast has contributed greatly to our understanding of the mechanisms by which sister chromatids are tethered prior to, and separated during, mitosis.

The medal lecture very appropriately began by describing the work of Robert Hooke, then made a beautiful transition to the present day, with reference to others who made key observations relating to cell division, upon which we have built our current knowledge.

After a brief summary of the mechanism of the spindle assembly checkpoint to orientate those not working in this field, Frank Uhlmann went on to address recent work in his lab. The cohesin ring complex binds to chromosomes, keeping sister chromatids together until their timely separation at anaphase, following its cleavage by separase. Results from this lab indicate that cohesin binds to intergenic regions of the chromosomes, where open reading frames from both orientations converge. Frank proposes that cohesin is moved along chromosomes during transcription, away from sites of loading, to ensure they do not subsequently fall off.

The second phase of the lecture addressed the issue of mitotic exit; how does a cell know when anaphase has finished, so that it can prepare for division? Studies in the Uhlmann lab have shown that separase, in addition to its role in the cleavage of cohesin, is also essential to trigger mitotic exit, in a manner independent of its protease activity. This activity of separase is essential for the release of the phosphatase Cdc14 from the nucleolus during anaphase, allowing it to assume its role in the mitotic exit network (MEN). This work has shown not only that chromosome segregation does not directly trigger mitotic exit, but also that it is not even essential for this process!

Intriguingly, the lab has also found that separase has a role in the regulation of microtubule dynamics during anaphase. During the lecture, experiments were also described that address the question of the segregation of ribosomal DNA. This talk was very enjoyable, accessible to all, and demonstrated that Frank Uhlmann is a very deserving winner of the Hooke Medal.

Carly Dix

The Gurdon Institute, Cambridge cd325@cam.ac.uk

Lunch meeting: Careers in Biological Sciences

This lunch provided ample fodder for researchers striving for liberation from the fetters of postdoctoral servitude. Senior scientists and representatives of the Wellcome Trust, Medical Research Council and Cancer Research UK gave us their expert insight into the selection process for career development awards, which can be distilled into the three Ps of person, project and place.

Person: Funding bodies support scientists who are able to demonstrate independence, as well as having a strong track record. Therefore, it is important to carve out your own research niche, by setting up your own collaborations and negotiating with your mentor which projects you can take from the lab when you leave.

Project: The primary aim of the research councils is to fund good science, which means that hypothesis-driven projects are favoured. The feasibility of the proposed research is also important, so include preliminary data where possible.

Place: Since career development awards are also viewed as an opportunity for extended training, it is beneficial if you can

demonstrate that you will be learning new scientific approaches. You should select an institution noted for its research excellence in your intended field. It is recommended that you move to a new institution/department to foster your independence from your postdoctoral adviser.

The speakers also advised advanced planning, given that some fellowships have only one application round per year. They suggested that people with eligibility concerns contact the fellowship offices directly, since these are flexible under certain circumstances. If you are an aspiring PI but don't yet know an NIA from a CDA, you should visit the websites of the main funding bodies, which contain details of the application procedures. Then, employ the final piece of advice we received: Go for it!

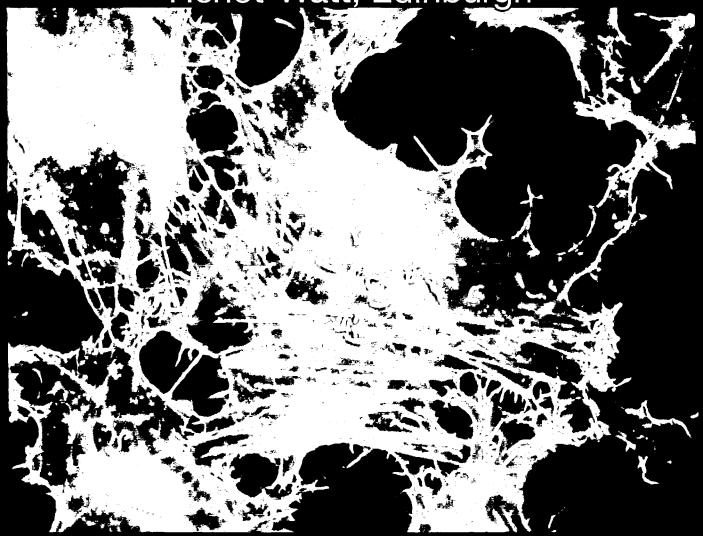
Dr. Elizabeth Callery, The Gurdon Institute University of Cambridge. emc13@cam.ac.uk

Powerpoint slides of the presentations are available from Joan Marsh (jmarsh@wiley.co.uk

Signalling and Cytoskeletal dynamics during infection

British Society for Cell Biology meeting organised by Michael Way

2nd - 5th October 2005 Heriot-Watt, Edinburgh



Confirmed Speakers include:

Pascale Cossart, Pete Cullen, Urs Greber, Gareth Griffiths, Ari Helenius, John Heuser, David Holden, Daniel Kalman, Vassilis Koronakis, John Leong, Tim Newsome, Guy Tran Van Nhieu, Lucas Pelkmans, Quentin Sattentau, Greg Smith, Dominique Soldati, Michael Steinert,

Michael Way, Matthew Welch, Tom Wileman, Xiaowei Zhuang



BSCB Autumn Meeting programme

The Edinburgh Conference Centre, Heriot Watt University. 2–5 October 2005

On the	BSCB website	Sunday 2	2nd October
Registration fo	rm		
Abstract submi Travel direction		14.00–18.00	Registration in James Watt Centre I Concourse
www.bscb.org		17.00–19.00	Buffet Dinner in Middle Floor Dining Room, Hugh Nesbit Building
***************************************	565.6.8	19.00–20.00	Plenary I in James Watt Centre I – Main Auditorium
			Pascale Cossart Exploitation of the cytoskeleton by bacterial pathogens: The Listeria paradigm
		20.00-00.00	Cash Bar in The Lectern Bar
Monday	3rd October		
07.30-09.00	Breakfast in Middle Floor Dining Room, Hugh Nesbit Building		
Session	1: Invasion	Session	2: Signalling at the plasma
	Main Auditorium, Chair: Ari Helenius	membrane and beyond	
09.30-10.00	Ari Helenius (Switzerland) New pathways of virus entry	Main Auditorium, Chair: Urs Greber	
10.00-10.30	Dominique Soldati (Switzerland) Gliding through life – the apicomplexan approach to invasion	14.30–15.00	Urs Greber (Switzerland) Adenovirus trafficking – from the plasma membrane to the nucleus
10.30-10.45	Short Talk from posters	15.00–15.30	Vassilis Koronakis (UK)
10.45-11.15	Tea/Coffee/Posters in Exhibition Area/Concourse	45 30 45 45	Host actin remodeling by enteropathogenic bacteria
11.15-11.45	Lucas Pelkmans (Germany)	15.30–15.45 15.45–16.15	Short Talk from posters Tea/Coffee/Posters in Exhibition Area/Concourse
5	Systems virology to dissect endocytic pathways in mammalian cells	16.15–16.45	Xiaowei Zhuang (USA) Cellular entry and trafficking of individual influenza
11.45–12.15	John Leong (USA)		viruses
	Actin pedestal formation by enteropathogenic and enterohemorrhagic E. coli	16.45–17.15	Pete Cullen (UK) Title to be advised
12.15-12.30	Short Talk from posters	17.15–17.30	Short Talk from posters
12.30-14.30	Lunch and Posters in Exhibition Area/Concourse		•
		18.00–19.30	Dinner in Middle Floor Dining Room, Hugh Nesbit Building
		19.30–21.30	Poster Session with Sponsored Drinks in Exhibition Area/Concourse
		21.30-00.00	Cash Bar in The Lectern Bar

Tuesday 4th October

07.30-09.00

Breakfast in Middle Floor Dining Room, Hugh Nesbit Building

Session 3	3: Cytoskeleton and	Session	4: Getting to cell periphery
signalling	during replication		Main Auditorium, Chair: Michael Way
	Main Auditorium, Chair: David Holden	14.30-15.00	Greg Smith (USA) Herpesvirus assembly of a neuronal egress apparatus
09.30–10.00	David Holden (UK) Activities of Salmonella during intracellular	15.00–15.30	Tim Newsome (UK) Src family kinases regulate vaccinia virus transport
	replication	15.30-15.45	Short Talk from posters
10.00-10.30	Michael Way (UK)	15.45-16.15	Tea/Coffee/Posters in Exhibition Area/Concourse
	Vaccinia and RhoA signalling	16.15-16.45	Matt Welch (USA)
10.15-10. 4 5	Short Talk from posters		Role of Arp2/3 complex-mediated actin nucle-
10.45-11.15	Tea/Coffee/Posters in Exhibition Area/Concourse		ation in bacterial and viral pathogenesis
11.15-11.45	Gareth Griffiths (Germany)	16.45-17.15	Tom Wileman (UK)
	Phagosomes, actin and the killing of mycobacteria		Role of cytoskeleton during movement of African
11.45-12.15	Michael Steinert (Germany)		Swine Fever virus from aggresomes to the tip of
	Custom-tailored Dictyostelium cells contribute to the		filopodia
	roadmap of Legionella infection	17.15-17.30	Short Talk for posters
12.15-12.30	Short Talk from posters	19.00-Late	Scottish Banquet and Ceilidh in James Watt
40.00.44.00			•

Centre II Main Hall

Wednesday 5 October

07.30-09.00

12.30-14.30

Breakfast in Middle Floor Dining Room, Hugh Nesbit Building

Lunch and Posters in Exhibition Area/Concourse

Session 5: Exit at the plasma membrane

Main Auditorium, Chair: Dan Kalman

10.00–10.30	Dan Kalman (USA) Disabling pathogenic E coli and orthopox viruses	11.45–12.15	John Heuser (USA) High-resolution 3-D EM imaging of cytoskeletal
10.30–11.00	Guy Tran van Nieu (France) Signals amplification and Shigella spreading in epithelial cells		developments at spots where poxviruses attach to the plasma membrane: a highly provocative situation
11.00–11.15	Short Talk from posters	12.15–12.45	Quentin Sattentau (UK) Title to be advised
11.15–11.45	Tea/Coffee/Posters in Exhibition Area/Concourse	12.45–13.00 13.00–14.30 DEPART	Short Talk from posters Lunch and Posters in Exhibition Area/Concourse

The ASCE 45th Annual Meeting

December 10-14, 2005, San Francisco

KEYNOTE SYMPOSIUM

Saturday, December 10

Clare Fraser, The Institute for Genomic

SYMPOSIA

Sunday, December 11 Quantitative Studies of Cell

Marc Kirschner, Harvard Medical School

Harold Erickson, Duke University Medical

Christine Jacobs-Wagner, Yale University Dyche Mullins, University of California, San

Monday, December 12 Wiring the Nervous System—8:00 am

Anirvan Ghosh, University of California,

Yishi Jin, University of California,

Adapting to Stress: Spotlight on

Tom Rapoport, Harvard Medical School/

David Ron, New York University School of

Richard Youle, National Institute of Neuro-

logical Disorders & Stroke/NIH

Tuesday, December 13

John Gurdon, Wellcome Trust/Cancer

Helen Blau, Stanford University

Research UK

University

Reprogramming Cell Fate-8:00 am

Markus Grompe, Oregon Health & Science

Santa Cruz/HHMI

Organelles-10:30 am

Hollis Cline, Cold Spring Harbor Laboratory

Signaling Networks-8:00 am

Garry Nolan, Stanford University Peter Sorger, Massachusetts Institute of

Prokaryotic Origins of the Cytoskeleton-10:30 am

Technology

Francisco

San Diego

HHMI

Medicine

search Organization/HHMI

Big Science, Little Science Linda Buck, Fred Hutchinson Cancer Re-

MINISYMPOSIA

Building Sensory Networks

Herwig Baier, University of California, San Francisco Gero Miesenboeck, Yale University School of Medicine

Cargo Sorting & Vesicular Transport

Robert Piper, University of Iowa Anne Spang, Max Planck Institute, Tuebingen

Cell Biology of the Synapses

David Colman, McGill University Janet Richmond, University of Illinois

Cell Migration/Motility

Peter Friedl, University of Würzburg Carole Parent, National Cancer Institute/NIH

Chromatin Dynamics

Terumi Kohwi-Shigematsu, Lawrence Berkeley National Laboratory Danesh Moazed, Harvard Medical School

Coordinating Adhesion & Signaling

Avri Ben-Ze'ev, Weizmann Institute of Science Vania Braga, Imperial College London

Coordination of Cytoskeletal Networks

William Bement, University of Wisconsin, Madison Talila Volk, Weizmann Institute of Science

Cytoskeletal Dynamics in Living Cells

Velia Fowler, The Scripps Research Institute Steven Gross, University of California, Irvine

Cytoskeletal Molecular Motors

Susan Gilbert, University of Pittsburgh Margaret A. Titus, University of Minnesota

Differentiation & Cancer

John Cleveland, St. Jude Children's Research Hospital Xi He, Children's Hospital, Boston

Epithelial Morphogenesis & Polarity

David Bilder, University of California, Berkeley Heike Fölsch, Northwestern University

Extracellular Matrix & Signaling

Josephine Adams, The Cleveland Clinic Foundation Joanne Murphy-Ullrich, University of Alabama at Birmingham

Formins & Arp2/3: Regulators of Actin

Henry Higgs, Dartmouth Medical School Matthew Welch, University of California, Berkeley

Intermediate Filaments

Ueli Aebi, University of Basel Bishr Omary, Palo Alto VA/Stanford University

Intersection of Signaling & Trafficking: Small GTPases

Host-Pathogen Interactions—10:30 am Pascale Cossart, Institut Pasteur, Paris David Roos, University of Pennsylvania Wesley Sundquist, University of Utah

Wednesday, December 14

Cell Growth & Division-8:00 am Ernst Hafen, Universität Zurich Tim Hunt, Cancer Research UK Yixian Zheng, The Carnegie Institution of Washington/HHMI

Harry Mellor, University of Bristol Lipid-Mediated Signals

Jim Casanova, University of Virginia

Antonella DeMatteis, Consorzio Mario Negri Sud Julie Saba, Children's Hospital/Oakland Research Institute

The Membrane Cytoskeleton

Vann Bennett, Duke University Medical Center/HHMI Elizabeth McNally, University of Chicago

Mitosis & Meiosis

Dean Dawson, Tufts University William Earnshaw, University of Edinburgh

Neuronal Polarity & Axo-Dendritic Growth

Lorene Lanier, University of Minnesota Liqun Luo, Stanford University

Nuclear Compartments

Joseph Gall, The Carnegie Institution of Washington Angus Lamond, University of Dundee

Nuclear Envelope Functions

Valérie Doye, Institut Curie, Paris Howard Worman, Columbia University College of Physicians & Surgeons

Organelle Dynamics

David Chan, California Institute of Technology Andreas Mayer, University of Lausanne

Pathogens Co-opting Host Cell Functions

Marcia Goldberg, Massachusetts General Hospital Michael Way, Cancer Research UK

Protein Folding & Quality Control

Judith Frydman, Stanford University Jonathan Weissman, University of California, San Francisco/HHMI

Protein Misfolding & Disease

William Balch, The Scripps Research Institute Harry Orr, University of Minnesota

Regulating Intercellular Junctions

Andrew Kowalczyk, Emory University School of Medicine Yoshimi Takai, Osaka University

Regulation of the Cell Cycle

Alison Lloyd, University College London Peter Sicinski, Dana Farber Cancer Institute

RNA Silencing Mechanisms

Bonnie Bartel, Rice University Greg Hannon, Cold Spring Harbor Laboratory

Signaling in the Immune System

Jason Cyster, University of California, San Francisco/HHMI Michael Dustin, New York University School of Medicine

Signaling in 3D Environments

Jeffrey Hubbell, Swiss Federal Institute of Technology Senthil Muthuswamy, Cold Spring Harbor Laboratory

Stem Cell Niches

David Scadden, Massachusetts General Hospital Allan Spradling, Carnegie Institution of Washington/HHMI

Trafficking Proteins & Complexes

James Hurley, National Institute of Diabetes & Digestive & Kidney Diseases/NIH

Sean Munro, MRC Laboratory of Molecular Biology, Cambridge

For more information, contact the ASCB at 301-347 9300; ascbinfo@ascb.org or www.ascb.org

Other forthcoming meetings

2005

30th FEBS Congress and 9th IUBMB Conference

2–7 July, Budapest www.FEBS-IUBMB-2005.com

Identifying Extracellular Matrix and Adhesion Molecules

8 July, Birkbeck College, London www.euroscicon.com

Applied Functional Genomics

20–23 August, University of Aarhus, Denmark www.mbio.au.dk/~clark/workshop/homepage.htm

FEBS/ESF workshop on Integrated Approaches in Cytoskeleton Research

21–31 August, Luxembourg City cytoskeleton.crp-sante.lu

Modelling Metabolic and Signal Transduction Networks

ESF Training Course 1–4 September, St Hugh's College, Oxford mudshark.brookes.ac.uk/ESF

15th International Society of Developmental Biologists Congress

3–7 September, Sydney www.isdb2005.com

The 6th UK Cord Blood Immunology Group Meeting

9 September, The Centre for Life, Newcastle www.euroscicon.com

Major steps in cell evolution: evidence, timing and global impact

26–27 September, Royal Society, London www.royalsoc.ac.uk/events

Epithelial Mesenchymal Transition Conference

1–3 October, Vancouver, British Columbia Maria Freeman, Maria@malachite-mgmt.com

BSCB Autumn meeting: Signalling and cytoskeletal dynamics during infection

2-5 October, Heriot Watt University, Scotland Michael. Way@cancer.org.uk

SNP mapping

21 October, Birkbeck College, London www.euroscicon.com

Assaying Chemokines and Chemotaxis

28 October, Birkbeck College, London www.euroscicon.com

Regenerative Medicine

4 November, Birkbeck College, London www.euroscicon.com

High-throughput Technologies and Data Analysis

9 November, Birkbeck College, London www.euroscicon.com

Proteomics Challenges and Emerging Technologies

11 November, Birkbeck College, London www.euroscicon.com

2D Electrophoresis: the way forward

18 November, Birkbeck College, London www.euroscicon.com

ASCB 45th Annual Meeting

10–14 December, San Francisco www.ascb.org

2006

BSCB/BSDB Joint Spring Meeting

(Mon 20) Tues 21 – Thurs 23 March 2006, University of York

Imaging Membrane Dynamics: Visualization of Trafficking Pathways

A joint meeting of the BSCB and Royal Microscopical Society 14–17 September, Royal Holloway College, University of London, Egham, Surrey david.stephens@bristol.ac.uk

2007

16th International Congress of Cytology 13–17 May, Vancouver, BC, Canada

www.venuewest.com

Techniques in Molecular Biology, 2005

University of Hertfordshire, Hatfield, Herts, UK

Details and application forms from Dr Ralph Rapley, School of Life Sciences, University of Hertfordshire College Lane, Hatfield, Herts AL10 9AB UK.
Tel: (01707) 285097
R.Rapley@herts.ac.uk
www.herts.ac.uk/stc

RNA extraction and analysis 30 June: One-day laboratory/lecture course

PCR methods and applications
1 July: one-day laboratory/
lecture course

Introduction to bioinformatics
5 July: one-day practical/
lecture course

Immunology: basic terms and techniques 7 July: one-day laboratory /lecture course

Molecular biology: basic terms and techniques 8 July: one-day laboratory/lecture course

Proteins and proteomics 5–6 September: two-day laboratory course

Nucleic acids and genomics 7–9 September: three-day laboratory course

Application to join the BSCB

Please complete and return along with a signed Direct Debit mandate to: Margaret Clements, Department of Zoology, Downing Street, Cambridge, CB2 3EJ.

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	
	e of its members on the BSCB web page. This list is ur details will be included only if you tick this box	s not sold
Applicant's signature:		Date:

British Society for Cell Biology



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, Department of Zoology, Downing Street, Cambridge, CB2 3EJ.

To The Manager,	Bank/Building Society	Originator's identification number 9 4 1 4 5 1		
Address		FOR BSCB USE ONLY This is not part of the instruction to your bank/building society		
	Postcode	5. Originator's BRITSO (for office use only)		
Please write the full postal add Name of account holder	dress of your branch in the box above.	6. Instructions to the Bank or Building Society Please pay the British Society for Cell Biology Direct Debits from the account		
2. Name of account noider		detailed on this Instruction subject to the safeguards assured by the Direct Debit Guarantee.		
3.Account number		Signature		
4. Sort code		Date		
Banks/Building Societies may ref from some types of account.	use to accept instructions to pay direct debits			

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 younger BSCB members at the beginning of their research careers to attend meetings. They are aimed at PhD students and postdocs. 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 £300 for UK meetings (except for BSCB Spring Meeting for which the registration and accommodation costs will be made, even in excess of £300), up to £400 for European meetings and up to £500 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.
- Normally, no applicant will receive more than one award in each calendar year and three in toto.
- The applicant must be contributing a poster or a talk.

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 following:

- the completed and signed application form (below)
- · a copy of the abstract being presented
- 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 Work address (write clearly – this will be used as a return label)	Meeting for which application is made (title, place, and date):		
	Estimated expenses: Travel: Subsistence: Registration:		
E-mail address: Age: BSCB Membership number: The years of previous Honor Fell awards:	Have you submitted any other applications for financial support? YES NO (delete as applicable). If YES, give details including source and whether these monies are known to be forthcoming:		
Degrees (with dates):	Supporting statement by Head of Department: 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. Signature: Name:		
Number of meetings attended last year:	Applicant's signature:		

The British Society for Cell Biology

BSCB President's report, 27th April 2005

Society business

It has been another exciting and productive year for the BSCB. Our Autumn and Spring meetings were a great success and I thank everyone involved in their organization for all their hard work. The Autumn programme was put together by Mary Herbert, while Jordan Raff was responsible for the Spring programme. This year's Spring meeting was held jointly with the BSDB, and the two meeting secretaries, Kairbaan Hodivala-Dilke and Nancy Papalopoulu, worked extremely well together. The meeting was oversubscribed and the standard of the posters and short talks selected from the abstracts was truly outstanding.

As you can see from our accounts, the BSCB is in a sound state financially, largely due to the efforts of our treasurer, Mark Marsh (who nevertheless turned a blind eye to our massive over-expenditure on Honor Fell travel awards!). As always, our Sponsors are essential for the success of our meetings. We are very grateful to all of them, and this year I would particularly like to thank Science magazine for generously funding the drinks reception at the Spring meeting poster session.

The rest of the BSCB committee has continued to work hard on behalf of our members. We are indebted to those members retiring from the committee and thank them for all their efforts. We are delighted to welcome Sean Munro and Sylvie Urbé, who have recently joined the committee, as well as a large number of new BSCB members, listed opposite.

Jonathan Pines is running the BSCB Ambassadors scheme and would like to hear from anyone who is willing to publicize the Society in her or his own University Department or Institute. Being an ambassador does have its perks, in particular enjoying free drinks in the bar with Jonathan.

At the AGM it was decided that the BSCB would remain affiliated to the International

Federation for Cell Biology, rather than becoming a full member.

BSCB lunchtime meetings

For the second year running, we held a 'Careers' lunch and a 'Women in Cell Biology' lunch at our Spring meeting. The topic of the Careers lunch, organised by Michael Whitaker, was the various fellowship schemes that are available to enable you to establish yourself as an independent investigator. We had a superb opening talk from Clare Isacke, who described her experiences in both University and Institute settings. Shabi Syed guided us through the MRC fellowship schemes. Fiona Hemsley and Simon Vincent put the Cancer Research UK perspective, while Helen Fisher described the Wellcome Trust schemes available. Clare, Simon and Helen kindly donated their PowerPoint presentations to me and these are available on request.

Liz Smythe organized the Women in Cell Biology lunch and introduced the issue of the 'leaky pipeline', the steady loss of talented women during the course of a scientific career. Michelle West described what it is like to choose to be a long-term postdoc, working four days a week instead of full time, funded on her boss' programme grant. We looked enviously at her publication list and agreed that there should be a way of recognizing Michelle's situation as a positive career choice, rather than describing it as being 'just a postdoc'. Helen Arthur recounted how she had successfully reentered science after a 10 year career break (heartening news for members of the audience who thought six months might be too long) and discussed the various schemes that are designed to help women re-enter science after a spell at home raising children. Scottie Robinson gave a very entertaining account of the adventures as a PhD student that led to her late start as an independent, and highly successful, group leader. The tone of this lunch was very upbeat and much appreciated by the audience. I am very grateful to Michael and Liz for organising these events. Next year's lunches will deal with how to get your papers published, and – to fit with our stem cell theme – scientific ethics. We would welcome suggestions for topics for future meetings.

Reflections on entering middle age Finally, I will be retiring as BSCB president next year and this, combined with the BSCB's 40th birthday, has put me in a reflective mood. I first went to a BSCB meeting when I was a PhD student and I'll never forget the excitement of hearing talks by people whose papers I had read. I'd assumed that all the speakers would be elderly, so it was a revelation that some of them were not much older than me and that they were all very accessible, happy to talk with students and postdocs over coffee or drinks. Later I became treasurer of the BSCB and I spent many Sunday afternoons writing out cheques for Honor Fell travel awards - a very satisfying way to spend money (if not Sunday afternoons).

Along the way, I've organized BSCB meetings, spoken at them and had the pleasure of seeing my PhD students win the poster prize competition. As the Society gets older, its essential character remains unchanged, even though the science is moving incredibly fast and we are constantly developing new ways to benefit our members. Last, but not least, the BSCB is a showcase for British science and overseas speakers come away from our meetings with the impression of a talented and vibrant cell biology community. So, we may be 40 but we look pretty good, don't you think?

Fiona Watt

British Society for Cell Biology

New members

Almiro do Vale, Maria I.

Baker, Natasha Baria, Katherine Barrett, Rachel Bausek, Nina Bennett, Lara Benson, Elizabeth Bhatti, Saeeda Bickmore, Dr. Wendy Bright, Michael D.

Britt-Compton, Bethan Buckland, Gemma L. Buttrick, Graham Byron, Adam Charolidi, Nicoletta

Cibert-Goton, Vincent Childs, Andrew

Clark, Anna Cobbold, Laura Collin, Dr. Ludovic Cooley, Carol Cooper, Oliver Cotterell, James

Cremona, Catherine Cross, Janet Crouchman, Sophie D'Amico Lago, Gabriela Di Fiore, Dr. Barbara Doceul, Virginie Doyle, Alexander Evans, Geraint

Fernius, Josefin Foley, Mathew J. Galli, Dr. Cesare Gallo, Federico

Gao, Mr. Shan Germain, Mitchel Graham, Joanna

Handley, Mark Hanson, Kirsten Harley, Margaret Harrison, Richard

Heath, Robert J.W. Heywood, Hannah Hickinson, David

Holt, Oliver Hornsby, Lucy Hughes, Julian Hussain, Kamran Jafari, Gholamali Johnson, Matt

Keramari, Maria Kershaw, Tom Kisielewska, Jolanta Kong, Yi Wen

Krause, Matthias Kumar, Vikash Kuo, Hsiao-Che Linden, Andreas Jan Mascarenhas, Monica McCormick, Laura Jane McTaggart, Malcolm Metcalf, Daniel Millard, Dr. Tom

Miller, Jayne Moore, Dr. Tara Murray, Andrew Nayak, Dr. Vrinda Nedjai, Belinda Neyen, Claudine

Ngoc-Sa, Nguyen Huu O'Donoghue, Jean Palmer, Zoe Passey, Samantha Patterson, Lucy

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Rahman, RumanRalph, Emma

Rengifo, Andrea

Reynolds, Dr. Andrew R.

Roberts, Kirsty Schirmer, Dr. Eric Scott, Dr. Kate Seifert, Anne Shackleton, Dr. Sue

Shaw, Lisa

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Small, Donna Staniland, Amelia Stansfield, Peter Stones, Dr. Rachel Strauss, Bernhard Stucke, Dr. V.M. Taylor, Eleanor K. Thompson, Oliver Urbe, Dr. Sylvie Venn, Neil

Vermeren, Dr. Matthieu

Wagstaff, Laura Wang, Chiuhui Mary Williams, Tomos Witter, Daniel Wright, Graham Zahra, Rabaab

British Society for Cell Biology

Committee Members 2005



President Dr Fiona Watt Keratinocyte Laboratory, Cancer Research UK, 44, Lincoln's Inn Fields, London, WC2A 3PX Tel: 020 7269 3528 f.watt@cancer.org.uk Appointed 2000; retires 2006



Secretary Professor Michael Whitaker Dept Physiological Sciences, The Medical School, Framlington Place, Newcastle upon Tyne, NE2 4HH Tel: 0191 222 5264 Fax: 0191 222 5296 michael.whitaker@ncl.ac.uk Appointed 2000; retires 2006



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
m.marsh@ucl.ac.uk
Appointed 2001; retires 2007



Meetings Secretary
Dr Kairbaan Hodivala-Dilke
The Cell Adhesion and Disease Laboratory
Department of Tumour Biology
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 7014 0401
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 2000; retires 2006

Newsletter editor

(to whom material should be sent

— see guidelines for contributors)

Dr Joan Marsh

John Wiley & Sons, International House,
Ealing Broadway Centre, London W5 5DB.

Tel: 020 8326 3846

Fax: 020 8326 3802

jmarsh@wiley.co.uk

Appointed 2001; retires 2007



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, UK Tel: +44 (0) 20 7848 8056 Fax: +44 (0) 20 7848 6435 tony.ng@kcl.ac.uk



UKLSC/IOB Liaison

Dr Stephen Nurrish
MRC Laboratory for Molecular Cell Biology,
University College London, Gower St,
London, WC1E 6BT
Tel: 020 7679 7267
s.nurrish@ucl.ac.uk
Appointed 2002; retires 2005



Committee members

Dr Vania Braga
Cell & Molecular Biology Section,
Division of Biomedical Sciences,
Faculty of Medicine,
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



Dr Gillian Griffiths
Sir William Dunn School of Pathology
University of Oxford
Oxford OX1 3RE
Tel: 01865 275 571
gillian.griffiths@path.ox.ac.uk
Appointed 2002; re-election due 2005





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





Professor Angus Lamond Wellcome Trust Biocentre, University of Dundee, MSI/WTB Complex, Dundee DD1 5EH Tel: 01382 345473 Fax: 01382 345695 a.i.lamond@dundee.ac.uk Appointed 2000; retires 2006





Dr Sean Munro MRC Laboratory of Molecular Biology Hills Road Cambridge CB2 2QH Tel: 01223 402236 sean@mrc-lmb.cam.ac.uk Appointed April 2005: re-election due 2008



Appointed April 2005: re-election due 2008





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 r.a.quinlan@dur.ac.uk Appointed 2001; retires 2007



Schools Liaison Officer

Reading RG6 7NB

Tel: 0118 962 2045 d.archer9@ntlworld.com

194 Silverdale Rd, Earley

David Archer

Dr David Stephens

University of Bristol, School of Medical Sciences,

University Walk,

Tel: 0117 928 9955

Dr Sylvie Urbé

Crown Street

urbe@liv.ac.uk

Liverpool L69 3BX

Fax: 0151 794 4434

Bristol

BS8 1TD

Department of Biochemistry,

david.stephens@bristol.ac.uk

The Physiological Laboratory

University of Liverpool

Tel: 0151 794 5432/5729

Appointed 2004; re-election due 2007

BSCB assistant Margaret Clements Department of Zoology, Downing Street, Cambridge, CB2 3EJ Tel: 01223 336655 Fax: 01223 353980 BSCB@zoo.cam.ac.uk





Honor Fell Travel Awards Dr Jordan Raff Wellcome/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 2007



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Tel: +44 (0)1223 336655 Fax: +44 (0)1223 353980,

E-mail: BSCB@zoo.cam.ac.uk

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