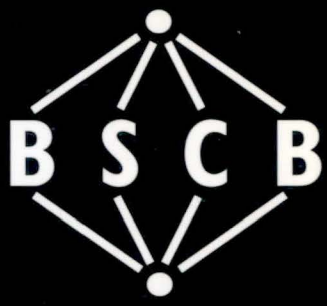
BSCB NEWSLETTER



Winter 1999

BSDB/BSCB Joint Spring Meeting

29-31 March 2000 University of Warwick

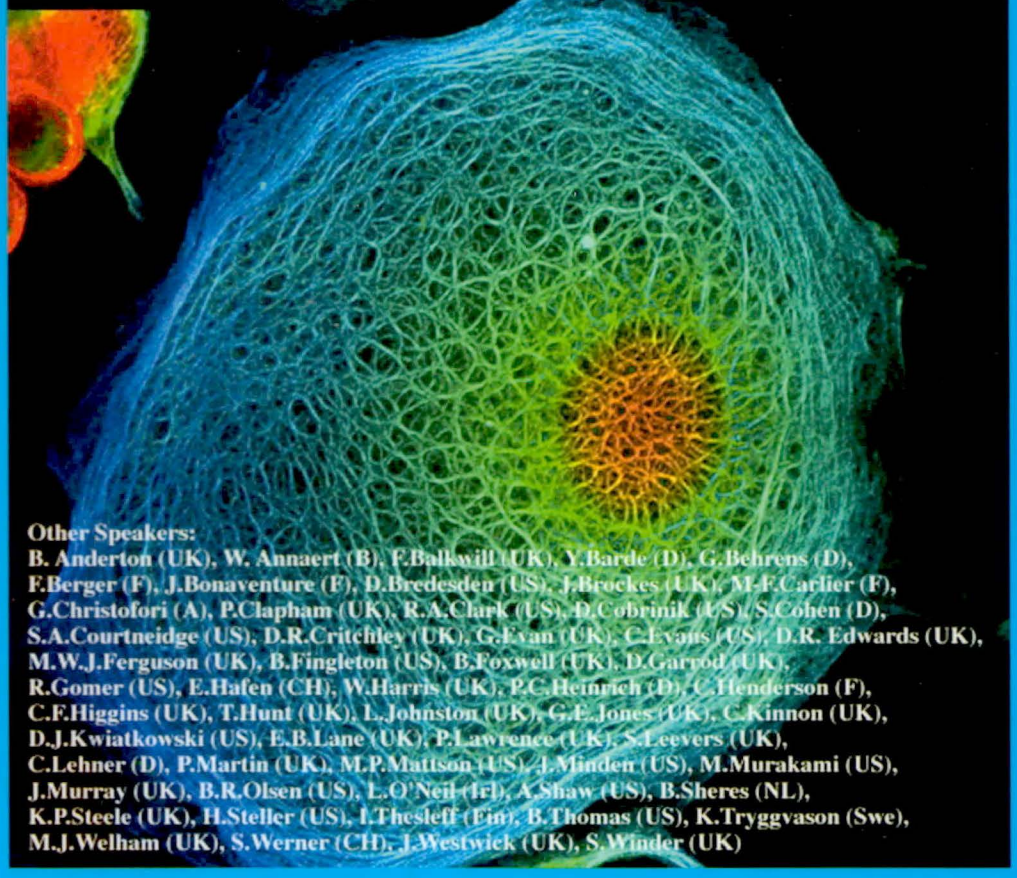
PATTERN FORMATION AND THE CONTROL OF CELL NUMBER

- Pattern formation and proliferation
- Cell size/number
- Pattern formation and cell death
- Proliferation and death in organogenesis

CELL BIOLOGY AND DISEASE

- Angiogenesis and tumour progression
- Cell adhesion and the cytoskeleton in disease
- Cellular basis of inherited disease
- Cytokines and disease
- Neurodegeneration
- Wound healing

Plenary Speakers: Judah FOLKMAN, Bruce EDGAR and Martin RAFF



Other Speakers:
B. Anderton (UK), W. Annaert (B), F.Balkwill (UK), Y.Barde (D), G.Behrens (D), F.Berger (F), J.Bonaventure (F), D.Bredesden (US), J.Brockes (UK), M-F.Carlier (F), G.Christofori (A), P.Clapham (UK), R.A.Clark (US), D.Cobrinik (US), S.Cohen (D), S.A.Courtneidge (US), D.R.Critchley (UK), G.Evan (UK), C.Evans (US), D.R. Edwards (UK), M.W.J.Ferguson (UK), B.Fingleton (US), B.Foxwell (UK), D.Garrod (UK), R.Gomer (US), E.Hafen (CH), W.Harris (UK), P.C.Heinrich (D), C.Henderson (F), C.F.Higgins (UK), T.Hunt (UK), L.Johnston (UK), G.E.Jones (UK), C.Kinnon (UK), D.J.Kwiatkowski (US), E.B.Lane (UK), P.Lawrence (UK), S.Leevers (UK), C.Lehner (D), P.Martin (UK), M.P.Mattson (US), J.Minden (US), M.Murakami (US), J.Murray (UK), B.R.Olsen (US), L.O'Neil (Ir), A.Shaw (US), B.Sheres (NL), K.P.Steele (UK), H.Steller (US), I.Thesleff (Fin), B.Thomas (US), K.Tryggvason (Swe), M.J.Welham (UK), S.Werner (CH), J.Westwick (UK), S.Winder (UK)

BRITISH SOCIETY FOR CELL BIOLOGY

The BSCB newsletter is published twice a year, June and December

Guidelines to Contributors
These guidelines apply to commissioned articles and images, to articles and images that members of the BSCB or interested parties would like to submit to the newsletter (see invitation below), and to material from members of the BSCB committee. The BSCB newsletter also accepts commercial advertisements – see advertising information.

Submission of text: Send the first version in the body of a normal e-mail (not as an attachment). If you do not have access to e-mail, please contact Kathryn Ayscough (address below). Once this has been accepted, submit the final version including all editorial changes, on a floppy disk (preferably in Microsoft Word) and a printed hard copy. Write your name, title of the article, and contact address on the floppy disk. If possible please include one or more images to accompany the submitted text (for example, a picture of the author(s), a picture to illustrate part of the text). Note for members of the BSCB committee, any standard requirements for the newsletter need only be submitted by e-mail and the first/final version requirement is not applicable. For non-standard articles from the Committee, the full procedure applies as above.

Submission of images: submit on a floppy disc, or as a high quality print. For images submitted on disk a printed hard copy must also be supplied (this is for layout purposes only and need not be high quality). Write your name, title of the image, and contact address on the floppy disk and on the reverse of the printed hard copy. Indicate the top of the image. A figure legend should be supplied on a disk and as a hard copy. Electronic files may be JPEG, TIFF or photoshop (300dpi preferred). Line drawings may also be PICT or Adobe illustrator. Preference is given to colour images for the front cover. Images for inside pages may be supplied as grey scale or colour, but will be printed as greyscale.

An Invitation to Submit Articles and Images
If you have an idea for an article please e-mail a brief outline first. Images for consideration for the front cover and inside pages are very welcome. Please submit as above. Please also state whether the image is for consideration for: front cover only, inside pages only or front cover first choice, with automatic consideration for inside pages second choice. Suggestions for images are those that highlight the research in your laboratory, a recent publication from your group, or a review of recent progress in a field.

Advertising Information
Single advertisement:
Back cover Black and White £275; Colour £425
Inside front cover Black and White: £275
Full inside page, black and white only £220
$^1/_2$ Inside page, black and white only £110
$^1/_4$ Inside page, black and white only £55

Four advertisements, to cover two years. The costs are reduced by 30%.
Mailing List (Peel-off Labels) – £225.00 + p&p

Supply either on a floppy or zip disk for Macintosh (Quark version 4, Quark version 3.32, JPEG, tiff or photoshop) with margins: top 26mm, left/right/bottom 20mm. Page size 218x280mm. Alternatively, supply film: single/four colour positive, right reading, emulsion down, screen 133x150. **Please note, there is only one colour advert slot per newsletter.**

For further information on commercial advertising contact: Margaret Clements, BSCB assistant, Department of Zoology, Cambridge University, Downing Street, Cambridge CB2 3EJ. Tel: +44 (0)1223 336655 Fax: +44 (0)1223 353980, E-mail: zoo-jeb01@lists.cam.ac.uk

There is no charge to advertise a scientific or educational meeting. Submit as for guidelines for contributors, above.

Submit all articles, images, committee items, and adverts, as per instructions to:
Dr Kathryn Ayscough, Division of Biochemistry and Molecular Biology, Davidson Building, University of Glasgow, Glasgow, G12 8QQ. Tel: 0141 330 3595. Fax: 0141 330 5027. E-mail: kathryn.ayscough@bio.gla.ac.uk

Deadlines for receipt of the *final* accepted version of articles and all other materials, and adverts:
[Note, the first version of articles from any contributor and any unformatted meetings information from the Committee should arrive two weeks before these dates].
April 7 for publication in June issue, or 6 weeks after the commission of an article, which ever is the earliest.
October 1 for publication in December issue, or 6 weeks after the commission of an article, which ever is the earliest.

Subscription information
Regular member, direct debit £20
Student or teacher member, direct debit £8
Regular member, bankers draft £25
Student or teacher member, bankers draft £12

Pay by direct debit (form on p30). If you are still paying by standing order, please cancel it and set-up direct debit. Those members who do not have a UK bank account should pay by bankers draft in pounds sterling payable to 'the British Society for Cell Biology'.

New members should also complete an application form to join the BSCB (form on p29) and include it with their subscription dues.
Send direct debit forms, bankers drafts and any membership application forms to Margaret Clements, Department of Zoology, Downing Street, Cambridge, CB2 3EJ.

BSCB members benefit from discounted journal subscription rates. Where prices are given, the full price is listed first, followed by the discounted member price (see also pages 8 and 12).

Journal of Cell Science	£142/106 (paper **or** online); £163/122 (paper **and** online)
Development	£231/173 (paper **or** online); £266/199 (paper **and** online)
Journal of Experimental Biology	£165/123 (paper **or** online); £190/142 (paper **and** online)
Trends in Plant Science	£89/71
Current Opinion in Plant Biology	£131/105
Trends in Cell Biology	£89/71
Current Opinion in Cell Biology	£131/105
Current Biology	£105/84

Postmaster and General Inquiries
Send changes of address, amendments, and general queries to:
Margaret Clements, BSCB assistant, Department of Zoology, Cambridge University, Downing Street, Cambridge CB2 3EJ. Tel: +44 (0)1223 336655 Fax: +44 (0)1223 353980, E-mail: zoo-jeb01@lists.cam.ac.uk

Invoices: send to Stuart Kellie, BSCB treasurer, Yamanouchi Research Institute, Littlemore Hospital, Oxford OX4 4XN.

Front cover
This human epithelial cell (MCF7) has been stained with anti-HSP27 antibodies. Note the cytoskeletal pattern of the staining which from other studies (Perng *et al.* 1999 *J Cell Sci*, 112:2099–2112) we know corresponds to the keratin intermediate filament network. These data indicate that small heat shock proteins are normally associated with intermediate filaments in growing, unstressed cells. The fluorescence signal has been colour coded for depth, so that the signal furthest away and therefore nearest to the substratum is blue whilst that closest is red, making the filaments above the nucleus appear yellow.

BSCB Newsletter
Winter 1999

Editorial

First, a welcome to all the new BSCB members who have joined this year – I hope you will enjoy reading the newsletter and also being a member of this growing society. Cell biology is truly developing as a central focus of many areas of research and many of us are realising the importance of undertaking complementary in vivo studies in order to understand physiological roles of the processes and molecules that have thus far been studied at a biochemical or structural level.

Nowhere is the understanding of biological processes more relevant at a cellular level than in our understanding of disease and next year's Spring meeting is devoted to recent advances in this area. The meeting 'Cell Biology in Disease' will be held in the University of Warwick from 28–31 March, 2000 and it promises to be a particularly exciting meeting. General information, registration forms and the scientific programme can be found later in the newsletter.

Also in this issue we have a profile of one of the BSCB past treasurers – John Pitts – who is just about to retire after a very active and productive career. A new feature we have is 'Cell Biology in Europe' which in this edition will focus on Finland. However, anyone from other European countries who would like to contribute to this section please feel free to contact me at the email address below.

This past year has also seen some excellent meetings and we have reports here from the BSCB autumn meeting on Endoplasmic Reticulum and also from the European Cytoskeleton Forum held this year in Portugal.

As ever I am extremely grateful to people who have written a report or feature for this newsletter. And as usual I would like to invite any member to submit any articles, meeting reports, comments or suggestions regarding the newsletter to the editor.
The Editor

Newsletter editor: Kathryn Ayscough
Publications editor: Louise Cramer
Design/layout: Giles Newton
Printers: Cambridge University Press
Website: maintained by Simon Hughes
http://www.kcl.ac.uk/links/bscb.html

Contents

Cell Biology Titles
from Portland Press

Protein Targeting and Translocation

Edited by DA Phoenix, University of Central Lancashire, UK

1 85578 121 2 Hardback 1998 304 pages £75.00

Protein Targeting and Translocation provides an overview of protein targeting, with experts reviewing developments in their area. By offering coverage of all the major eukaryotic and prokaryotic systems plus background information on protein membrane interaction, the book not only provides readers with coverage of their main areas of interest, but also affords them the opportunity to compare and contrast targeting to different organelles.

The book is aimed at researchers and postgraduates with an interest in protein targeting but is also a useful overview of targeting for undergraduates.

Mitochondria and Cell Death

Edited by GC Brown, University of Cambridge, UK, D Nicholls, University of Dundee, UK, and C Cooper, University of Essex, UK

1 85578 125 5 Hardback 1999 240 pages £65.00

This book reviews the involvement of mitochondria in cell death and disease. This is a rapidly expanding field of major importance to both basic biology and medical science. Clear and comprehensive chapters from some of the key researchers in the field cover most aspects of the subject from the molecular to the *in vivo* level. The role of mitochondria in neurodegenerative, inflammatory and ischaemic diseases, as well as necrosis, apoptosis and ageing is reviewed. Each chapter is by a different set of authors describing one aspect of the field. Researchers and postgraduates throughout biology and medical science will want to read this book.

Landmarks in Intracellular Signalling

Edited by RD Burgoyne and OH Petersen, University of Liverpool, UK

1 85578 101 8 Paperback 1997 278 pages £20.00

This book brings together sets of seminal papers which have contributed to key advances in intracellular signalling. Experts provide concise commentaries, which stand as mini-reviews in their own right.

For undergraduates, postgraduates, senior researchers. and lecturers, *Landmarks in Intracellular Signalling* will provide you with...

- Easy available access to a set of classic papers from one source at an affordable price
- A new appreciation of the quality of the original work that led to our current state of knowledge on the subject
- A novel introduction to the field
- Insight into the processes of scientific discovery (i.e. problem-solving, how one piece of research is built upon another)

Techniques in Apoptosis – A User's Guide

Edited by TG Cotter, University College, Cork, Ireland and SJ Martin, La Jolla Institute for Allergy and Immunology, USA

1 85578 076 3 Hardback Spiral 1996 344 pages £75.00
1 85578 129 8 Paperback 1996 344 pages £39.50

Cell death/apoptosis is currently an exponentially expanding field of broad research interest. The success of research projects depend on having access to detailed and clearly written accounts of techniques and methodologies used. This is particularly important in a new field. *Techniques in Apoptosis* aims to fill this niche, it differs from recent volumes on apoptosis since its focus will be purely technical in nature. Each chapter begins with an overview of the methodology to be employed, and goes on to provide detailed experimental protocols for the identification of apoptotic cells from plants to *Drosophila* to man in an easy-to-use, step-by-step format.

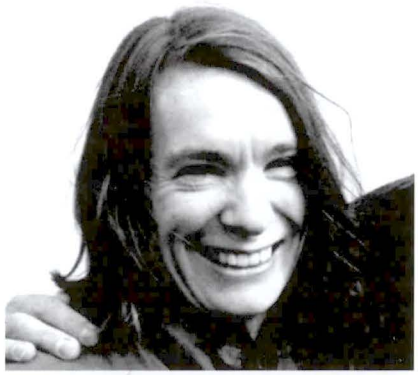
Written by key people in the field, this book is essential reading for all researchers in cell death, from PhD students to senior scientists.

NEWS

First BSCB Hooke Medal

We are extremely pleased to announce that Anne Ridley has accepted the first BSCB Hooke Medal. Anne will receive the medal at the Spring 2000 Meeting in Warwick where she will also be giving a lecture. A brief resume of Anne's career thus far is given below and was kindly written, at very short notice by Gareth Jones.

Nominations for the next BSCB Hooke medal can be sent to Michael Whitaker of the BSCB Committee at any time. Nominations should include a brief resume of the nominee and a list of their recent, relevant publications. Nominations received by the beginning of September 2000 will be considered for the 2001 award.



Anne Ridley: A personal resumé

Gareth Jones

All those that know her will not be at all surprised to learn that Anne has been awarded the BSCB Hooke medal in honour of her contributions to cell biology and to the good standing of British scientific endeavour in general. Born in 1963, Anne progressed smoothly through Cambridge University and ICRF, following a path familiar to many in the establishment of British science. In 1990, after a short but fruitful stay in the laboratory of David Page (Whitehead Institute, Cambridge MA), Anne took up the post that was to determine her future career; a postdoctoral fellowship in the laboratory of Alan Hall (then at The Chester Beatty Laboratories, London).

Initially under his tutelage, Anne rapidly developed her work on the Ras – related proteins Rho and Rac, using cell microinjection of bacterially expressed proteins to show a clear effect on the organisation of the actin cytoskeleton of cells. Towards the end of 1992 two seminal papers appeared back-to-back in *Cell* which described how Rho regulates the assembly of focal adhesions and stress fibres in cells while Rac regulates growth factor induced membrane ruffling and lamellipodial extension.

It would be hard to quantify the impact these two reports had at the time: I met Anne a few months before the papers were published; she was standing in front of two posters describing her work at a cell surface symposium at Cold Spring Harbor. People were milling around the poster and I was but one of many participants who recognised the huge significance of what I was seeing. After many years of describing how cells moved in culture, here I was reading about the control of the molecular machinery involved! Whatever my own feelings at that time, once the *Cell* papers had been assimilated and the results verified, pretty nearly every group working on cell movement had to get involved in the intricacies of these now classic members of the small GTPases.

About a year later, Anne left Alan Hall's laboratory to have her first daughter, and then to set up her own lab at the Ludwig Institute for Cancer Research, London. Within a few months, her laboratory began expanding as a result of external grant awards, firstly in collaboration with me on the regulation of macrophage motility, but rapidly followed by many projects of her own. By 1996 Anne had established a thriving group at the Ludwig, produced a second daughter, enhanced her reputation as a formidable intellect and become an international star in the field of Rho protein cell biology. These attributes have been subsequently recognised by her appointment to the editorial boards of at least 5 journals including *Current Biology* and *Molecular Biology of the Cell*, plus numerous calls to give talks at prestigious symposia.

Within ten years Anne has progressed from a promising postdoctoral assistant to an international figure who is read not only for her insights into cell biology, but as members of the ASCB will know, for her clear exposition on the themes of gender, family and professional commitment. I consider myself truly fortunate to know her both as a friend and collaborator and have little doubt in suggesting that the next decade will see a further substantial development of her career.

News of BSCB members

We would like to congratulate one of our members Tom Misteli, who has been awarded the annual Gian-Toendury Prize from the Union of Swiss Societies for Experimental Biology for his contributions to the study of nuclear architecture using live-cell microscopy. Tom is currently working at the National Cancer Institute, NIH, Bethesda, MD.

Tom Misteli

We would also like to congratulate Stephen Moss, who has been appointed to the Ashton Chair of Biomedical Research and Head of Cell Biology at the Institute of Ophthalmology in London. Perhaps significantly for the future of cell biologists in Britain, the initiative to create a cell biology department came in part from the clinicians at Moorfields Eye Hospital, who saw a clear need for a Dept of Cell Biology that could play a role in developing their own research needs. There are also up to 5 new lectureship positions associated with this new department which will be advertised in the usual sources in due course.

Changes on the BSCB committee

We would like to thank Birgit Lane for the tremendous amount of time and effort she has given to the BSCB during her time as Secretary. Birgit is stepping down from her position as BSCB secretary after 4 years (1995–99). She will be succeeded by Michael Whitaker (University of Newcastle) who is currently a member of the BSCB committee. We also are extremely grateful to Clare Isacke, the BSCB Committee representative on the UK Life Sciences Committee who is retiring from the committee after 7 years.

Membership information

In the last newsletter we appealed to all of you to help us trace our missing members, that is those people who are still paying their subscriptions but who have changed address and we can't get the newsletter or any other BSCB material to them. While some people have been traced there are still many for whom their newsletter is returned to us. The list of our missing members is on page 29/30 of the June 1999 Newsletter which can also be downloaded as a PDF file from the web site, *www.kcl.ac.uk/links/bscb.html* If you recognise any of these people, please email a current address to *zoojeb01@lists.cam.ac.uk*

Also, if any of you are still paying your membership subscription by Standing Order please cancel this and set up a direct debit. This makes it considerably easier for us to deal with membership accounts.

Awards

Bursaries for young scientists from central and eastern Europe

Six bursaries are available for young scientists from, and currently working in, Bulgaria, Commonwealth of Independent States, Czech Republic, Slovakia, Estonia, Hungary, Latvia, Lithuania, Poland, Romania, and the former states of Yugoslavia to attend the BSCB/BSDB Joint Spring Meeting at Warwick 28–31 March, 2000.

These bursaries, sponsored by the BSCB and the *Journal of Cell Science* will cover the cost of registration, accommodation and meals and a travel award of up to £250.

Applications, including a brief CV and reasons for wishing to attend the meeting should be sent by 28 January 2000 to the BSCB secretary, Professor Michael Whitaker (address on page 31).

BSCB members – if you know of any young scientists from central or eastern Europe who would benefit from attending this meeting, please send them this information.

Honor Fell Travel Awards

These awards are available up to a limit of £250, to provide financial support for young BSCB members to attend scientific meetings and conferences. Applications are considered for any meetings relevant to cell biology, although the applicant must be presenting a poster or talk. For more details and an application form, see page 28.

Young Cell Biologist of the Year Poster Prize 2000

All research students are invited to enter the next poster competition at our Spring 2000 meeting to be held at Warwick University, 28–31 March 2000. The first prize is a trip to the USA to attend the ASCB 2000 meeting to be held in San Francisco 9–13 December 2000 as a guest of the American Society for Cell Biology, with an opportunity to present their winning poster. The poster will be judged by a panel of internationally renowned scientists based on scientific merit and presentation.

BSCB Member's Profile: *John Pitt – A change of coats?*

After a little over 40 years in active research, John Pitts is hanging up his lab coat and retiring. Perhaps moving trades would be a better description. For the last ten years John has been running a small farm with a flock of a hundred or so sheep, several horses, hens and dogs as well leading a small research team with me at the Beatson Institute in Glasgow. His farm is situated in the foothills of Ben Lomond on the 'reiving' path of Rob Roy MacGregor and overlooking Flanders Moss where many invading armies came to grief. He and his wife Netta chose the spot for its beauty, history and, equally important, distance from the glare of Glasgow's street lights. So they have an unfettered view of the night sky, and this view is a key ingredient in John's approach to science.

John moved from Yorkshire to Scotland to do his first degree in St Andrews. He studied chemistry and not being one for pomp and circumstance, described himself modestly as a 'biochemist'. A foretaste of what was to follow came at St. Andrews through his climbing exploits. He fell a few hundred feet whilst leading one of the then harder ice routes in Glencoe. Such experiences probably gave John a more candid approach to the science enterprise. It's not worth dying for!

John jointly led a trip to explore Greenland with fellow mountaineering students gaining his first grant from the Mount Everest Foundation. This award held pride of place in John's cv being first in the list of the many grants he received. One of the memorabilia of this trip was a large print of John standing in front of a magnificent rock formation in Greenland. This print for many years hung alongside his pioneering work on 'metabolic co-operation' in his office. An almost identical picture taken from the same spot was featured in a mountaineering magazine a couple of years ago and claimed to show undiscovered climbing territory, obviously unaware of John's visit in the late 50's. This was – and still is – a hallmark of John, to tread new ground which is re-discovered many years later.

The last thing John would want is a list of his achievements. So perhaps a better way to describe his career would be to look at the approach he took. John went into any area of research with a 'wanting-to-know' attitude. He did not see any topic out of bounds so he became equally fluent in embryology as he was in his early research on the molecular biology of polyoma virus and bacteriophage which he carried out at Glasgow and Caltech in Bob Sinsheimer's lab. That wide spectrum of knowledge was coupled to John wanting to know how equipment and techniques worked. Few bother these days but John's approach meant he knew the limitations and pitfalls and led him to a better interpretation of his and other's data.

He also had the knack of exploiting serendipity and perhaps it was the chance discovery of metabolic co-operation in the mid 60's with John Subak-Sharpe and Bobby Burk for which he will be best known (see an article by John in *Bioessays* 1998, 20:1047–51). A simple discovery which caused him to switch fields. This is an attribute he has fostered with those who have come through his lab, to be imaginative and follow your nose.

I joined his lab in the early 70's as a PhD student and by lab I mean John, a technician and me. A far cry from the setups of today and even competitors' labs at the time in America. I arrived in the midst of controversy of what went through gap junctions. At one point it seemed it was everyone else versus John. But John's knowledge of nucleic acid metabolism allowed him and my predecessor to do some incisive experiments and demonstrate once and for all only small molecules and not large ones went through gap junctions. It earned him much respect in the international cell biology community.

John knew he was on the right track before doing the experiments because he took account of general principles. He knew it would make no sense for cells to share large molecules, otherwise they would never be able to differentiate. It was this common sense approach that underpinned much of his thinking.

The rest of the field, including myself, went chasing the proteins which made up gap junctions, but John asked the more imaginative question of what they might do. He had already realised the significance of this form of cell-cell communication and had talked far and wide about his ideas of gap junctions as a way of producing homeostasis and novel tissue phenotypes. This was around the time he moved from Biochemistry in Glasgow University to the Beatson Institute at the invitation of John Paul. John was a little over halfway through his research career and for the first time had something bearing resemblance to a sizeable research group. His high standing before then had been built up with a handful of graduate students, occasional postdocs and his own intellectual resources.

John turned his attention away from the limitations of model systems to map out cell-cell communication in a complex tissue so he could find out what actually might be happening. Few in any field really tackle the important issues. The approach he used was not unique but on the surface appeared difficult. But it was simplicity itself and revealed patterns of communication which could not have been guessed at by any other approach. This work is still ongoing and I suspect that in the years to come it will be seen as some of the most illuminating research in gap junctions.

The controversy on what went through gap junctions was followed hard on the heels by what they were made of. That was my area in his lab and my isolated position on the ductin story as a component of gap junctions would have gone down had it not been for John's support and his experimental approaches. He cajoled me to do the key experiments and John's enthusiasm was not confined to his office. He led by getting stuck in at the bench whenever he could, often with experiments of remarkable simplicity. When I returned after a couple of weeks in the States downhearted at what our rivals had done in the previous year, John had gone into action and in those two weeks he and a couple of his postdocs had the same data.

The ductin story was taken note of because John put his reputation behind it; a reputation built on well crafted and repeatable research papers. Perhaps if John had a weakness in terms of the modern era, it would be sitting on data. In these days of publish or perish, it might seem John would not survive. But he most certainly would. His papers may have been fewer in number and long in the making, but when they were published, they were highly regarded because everyone knew the data were correct and interpretation sound. They invariably challenged dogma and gave a broader picture with new ideas. The beneficiaries of his reputation were those people who passed through his lab.

His unfettered view had an uncanny way of getting the best out people. John would barrage anyone with questions about their work, what it meant and what were the weak points and how it could be strengthened. He did this with papers or grant proposals as well and at times there seemed more red ink on your work than anything else when he handed it back to you. At first some took this as a personal attack but then you realised that it wasn't you he was getting at. It was your ideas.

John became the Secretary/Treasurer of the BSCB in the late 1970's and alongside Colin Hopkins helped transform the society in the early 1980's into the

creature we have today. Under Colin's and John's leadership the idea of the main meeting being modelled around the American cell biology meeting took shape and the Society's meeting grew in number and quality. He moved with the times because he recognised the workings of the science community.

But at the end of the day what made life fun in John's lab, or in the Beatson, or serving on committees with him, as others will testify, was his approach to individuals. John listened to people and understood where they were coming from, whether they were Nobel prize winners or the people that emptied his office bin.

The picture of a youthful John in Greenland in his office was replaced several years ago by a small cutting from The Glasgow Herald "Suffolk Cross- £47.80". This was the top price for this breed of lamb at the Stirling auction that year and they came from John and Netta's farm. Long may it continue.

I know John would hate it being said that he was irreplaceable. Perhaps a better way of saying that we will miss you is to say that science would do an awful lot better if more of us had your attitude.

Malcolm E. Finbow
Department of Biological Sciences
Glasgow Caledonian University
Cowcaddens
Glasgow G4 0BA

Cell biology in Finland

Pekka Lappalainen and Marja Makarow

Finland has a long tradition in cell biology research. Many of the well known Finnish cell biologists such as Kai Simons, Ari Helenius and Erkki Ruoslahti started their careers at the Medical School of University of Helsinki in the Seventies. Since the 1970's cell biology research has spread out from the Medical School of Helsinki, and the majority of cell biological studies are currently being carried out in a number of new research institutes, the Biocenters.

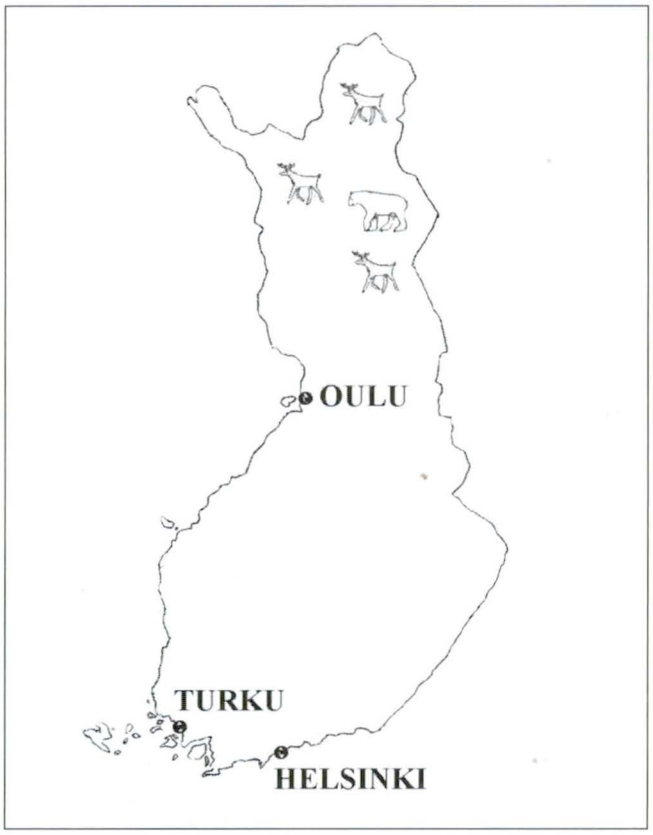


Figure 1. The large biocenters with cell biology research are located in the cities of Oulu, Turku and Helsinki.

Biocenters

During the past few years the funding for research has increased dramatically in Finland. At the moment the expenditure in research and development is 3 % of the gross national product. This is the second highest figure in the OECD countries after Sweden. The program of our current government includes a statement on development of research environments to further the international competitiveness of Finnish science, and the Council of Science and Technology of Finland has planned to increase the expenditure in research and development to 3.3 % over the years 2000–2004.

The good funding situation is already reflected in the quality of research, and at the same time new research institutes have been built in different parts of the country. Most of the cell biology research is currently concentrated in these new research institutes, biocenters. They are university institutes whose main responsibility is to carry out basic research, and in many cases they are not heavily involved in undergraduate teaching. However, all biocenters have graduate schools and are therefore actively organizing various courses and seminar series. At the moment cell biology is well represented in four of the large biocenters (see map in Figure 1).

Biocenter Oulu (www.oulu.fi/faculties/resea/biocente/homepag2.html) is an umbrella organization of the University of Oulu (see Figure 1). It was founded in 1986 to promote basic research in biosciences, medicine and biotechnology. Currently Biocenter Oulu consists of 11 research groups with a total staff of approximately 270. The most well known cell biology groups are those studying molecular biology of collagen (Kari Kivirikko and Taina Pihlajaniemi) and peroxisomes (Kalervo Hiltunen).

The Turku Centre for Biotechnology (www.btk.utu.fi/) belongs to the University of Turku and Abo Academi University. It is located in the city center of Turku (Figure 1). Turku Centre for Biotechnology was established in 1992 to facilitate biotechnology and biomedical research and scientific interactions between research groups across departments and

within two universities. The research activities of Biocenter Turku cover a wide range of cell biology from protein phosphorylation (John Eriksson), molecular biology of syndecans (Markku Jalkanen), molecular immunology (Riitta Lahesmaa) to regulation of transcription (Lea Sistonen).

In Helsinki there are two large biocenters, the Haartman Institute and the Viikki Biocenter. Haartman Institute is a biomedical research institute of the Medical School and is located in Meilahti campus close to the city center. The main research topics are cancer biology, human genetics, immunology and virology. The cell biology research in this institute includes studies on angiogenesis (Kari Alitalo), signal transduction (Leif Andersson and Erkki Hölttä), cytoskeleton/matrix interactions (Antti Vaheri), regulation of cell cycle (Tomi Mäkelä), formation and activation of cytokines (Jorma Keski-Oja) and membrane-cytoskeleton interactions (Olli Carpén). The number of research groups is around 35.

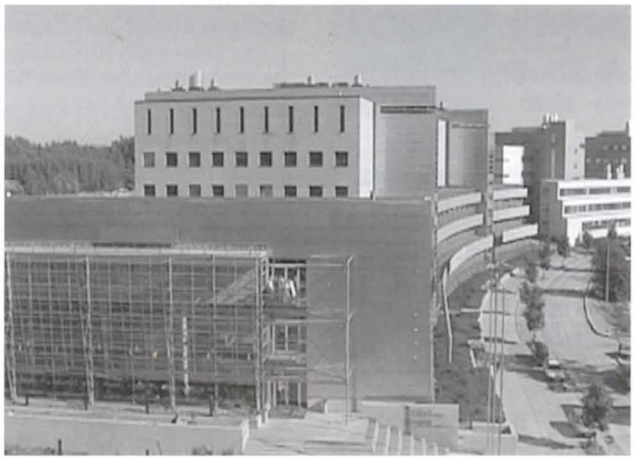


Figure 2: The Viikki Biocentre at the University of Helsinki

Viikki Biocenter

The Viikki Biocenter (www.biocenter.helsinki.fi) of the University of Helsinki is located in the Viikki campus. It was built in 1995 and it is the core of the Helsinki Science Park, which also includes the buildings of the Faculty of Agriculture and Forestry, the student village, a biobusiness incubator building and the residence for international scholars. The Viikki Biocenter consists of four major teaching and research institutes of the University of Helsinki. These are Departments of Biosciences and Pharmacy and part of the Department of Applied Chemistry and Microbiology, and the Institute of Biotechnology (www.helsinki.fi/biotek/10frame.html). The latter is an independent research institute, which was founded 10 years ago to promote molecular biology and biotechnology research in the University of Helsinki.

The Viikki Biocenter harbors some 70 research groups and many of these groups are carrying out studies in cell biology. Five of the cell biology groups are located in the Institute of Biotechnology. They are studying protein folding in yeast (Marja Makarow), virus replication (Leevi Kääriäinen), cell morphogenesis and migration (Johan Peränen), glycobiology (Ossi Renkonen) and the actin cytoskeleton (Pekka Lappalainen). The research activities of other cell biology groups at the Viikki Biocenter include neuronal development and plasticity (Heikki Rauvala), leukocyte adhesion (Carl Gahmberg), intracellular membrane traffic (Esa Kuismanen), and signal transduction (Mathias Bergman, Merja Auvinen, Ragna Rönnholm).

Due to the good research funding situation in Finland, the Viikki Biocenter has modern instruments including an up to date electron microscopy facility and two modern confocal light microscopes. It also harbors excellent technical core facilities. These include a protein chemistry laboratory with mass spectrometers (MALDI-TOF and Q-Tof electrospray mass spectrometers) and protein sequencing instruments, as well as a DNA laboratory with expertise to synthesize various specialized DNA fragments and capillary electrophoresis sequencing instruments for high output sequencing. Furthermore, Viikki Biocenter has a modern structural biology facility with one 800 MHz and two 600 MHz NMR-spectrometers as well as an X-ray crystallography unit.

Graduate schools in Finland

The Ministry of Education decided to found a graduate school system in Finland in 1995. It established 1300 four-year positions for graduate students, and allocated 30 million Euros yearly for this purpose. The aim was to enhance the quality of

supervision and to offer first class formal education in the form of practical and lecture courses. The graduate studies should preferentially be completed in four years, and the average age of dissertation should decrease. The Ph.D. theses in modern biosciences have previously consisted of four or more publications in international journals plus a literature survey. In addition, the student has to take about 32 credits (48 ECTS credits) of formal courses. The long post-graduate studies had put the Finnish new Ph.Ds in a non-competitive position relative to their European peers.

One hundred graduate schools are now in operation in the 20 universities of Finland, which has a population of 5 million. Some 15 are dedicated to biosciences and related disciplines, and many operate in the new biocenters. The Viikki Graduate School in Biosciences (www.biocenter.helsinki.fi/viikkigs/) and the Graduate School in Biotechnology and Molecular Biology (www.biocenter.helsinki.fi/biotechgs/) function in the Viikki Biocenter in Helsinki. They have altogether 92 students enjoying the 4-year salary from the Ministry of Education, and 13 matching funds students. The students have been invited to apply by an open call, and selected according to performance in basic studies, quality of research proposal and motivation. Each student has a support group consisting of the supervisor and two outside experts, who revise the work yearly and support the student if problems arise. Both schools have an excellent educational program, which is financed by the Academy of Finland, the Biocentrum Helsinki organization and the Institute of Biotechnology. All education is offered in English. It aims at preparing the students not only for basic research, but also for other jobs in the society, like biobusiness and science administration.

The graduate schools invite students from other European countries to apply. If you are interested, you should first identify a potential supervisor. You can find their names and research subjects from our web sites. The co-ordinators of the schools Dr. Ritva Niemelä (ritva.nimela@helsinki.fi) and Dr. Erkki Raulo (erkki.raulo@helsinki.fi) are happy to assist you.

Living in Finland

Because of a relatively large number of foreign post-docs and graduate students in Finland (for example at the Institute of Biotechnology, 25% of the researchers are from abroad) the working language in most laboratories in English. Furthermore, all seminars as well as teaching in graduate schools are in English, making it easy for foreigners to carry out research and adapt oneself to life in Finland. Also outside the laboratory, most Finns speak good English and therefore one can take care of everyday life, such as shopping, without any knowledge of the Finnish language.

Finland is also an excellent place to live for those interested in winter sports, water sports, hiking or fishing. Furthermore, larger cities such as Helsinki, Turku and Oulu have an active cultural life with theatres, art exhibitions and concerts. Because these cities also have universities with a large number of undergraduate and graduate students, they have an active night life with many parties and student events.

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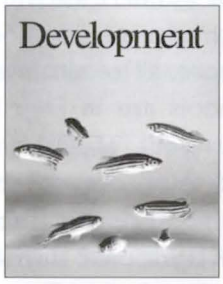
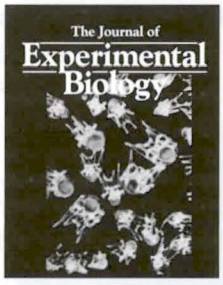
Scientific journals for today's researchers



The leading journal in developmental biology. It focuses on major experimental studies of genetic, molecular and cellular aspects of animal and plant development, that give insights into mechanisms, and also publishes reviews by the world's leading authors in the field. Its readership includes animal and plant developmental biologists, geneticists, molecular biologists, cell biologists, paediatricians, developmental neurobiologists and human embryologists.

The Journal of Cell Science covers the complete range of topics in cell biology. It is a leading journal in the field and its impact factor has risen steadily throughout the 1990s. The Journal is of key interest to all cell biologists, molecular biologists and geneticists



A leading journal in comparative animal physiology. It plays a major role in increasing cross-fertilisation of techniques and knowledge across specialisation boundaries, offering an approach to integrative biology at all levels, from the molecular and sub-cellular to the whole animal. Its principal readership is among scientists who study molecular, cellular and organismal physiology in the contexts of evolution and/or environment. Topics featured include cellular and molecular physiology, neuroscience, transport physiology, biomechanics and biochemistry.

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The Endoplasmic Reticulum – Structure and Function

Mark Larman and Jack Lucy

The autumn Workshop meeting was held in Bristol in late September, organized by George Banting, Mark Larman and Michael Whitaker. The aim of the meeting was to bring together work on three aspects of ER function: protein folding, membrane traffic and calcium uptake and release.

Patricia Camacho (Texas) started by reviewing her previous work that demonstrated how over-expression of calreticulin in *Xenopus* eggs alters the calcium release profile (Ca^{2+} is released but no oscillations). Calreticulin seems to exert this effect by interaction with Ca^{2+}-ATPase pumps (SERCAs). Using deletion mutants it has been shown that the C-domain of calreticulin is essential for this inhibition and could recognise the carboxy tale of SERCA2b. Two other proteins found in the membrane of the ER (calnexin and calmegin) possess protein kinase C phosphorylation sites on the cytosolic side. Mutations in these regions and dominant negative experiments prevented SERCA pump regulated calcium oscillations.

Mohinder Singh Bhogal (Leeds) discussed further possible regulatory mechanisms of SERCAs. Depletion of sarcoplasmic reticulum (SR) Ca^{2+} (in myocytes) results in phosphorylation of phospholamban, which promoted ER refilling. A protein kinase activity associated with the SR is low in <3 μM Ca^{2+} and high in the presence of >30 μM Ca2+. Monitoring ER lumenal Ca^{2+} changes (with compartmentalised Ca^{2+} indicators) and phospholamban phosphorylation revealed an increase as the ER Ca^{2+} concentration was dropped <5 μM with addition of ionomycin. This represents a novel way for ER lumenal Ca^{2+} concentration to control re-uptake into the ER.

Marek Michalak (University) discussed the physiological consequence of abnormal calreticulin expression. Calreticulin functions in Ca^{2+} storage/ homeostasis and as a lectin-like chaperone in protein synthesis, folding, and post-translational modification. The calreticulin gene was highly activated during the early stages of cardiac development, and embryos of a knockout mouse died by day 12 from umbilical hernia and other cardiovascular abnormalities. Cell lines derived from these embryos exhibited a 50% higher level of Ca^{2+} in the cytoplasm, and a low release of Ca^{2+} by bradykinin from IP3 stores. It appears that functions in calreticulin-deficient cells that depend on a sustained release of Ca^{2+} are adversely affected.

Earlier talks reported how the pump can be highly regulated. **Malcolm East** (Southampton) introduced regulation by lipid composition. Although the lipid-protein interface (the annular sites) of SERCA pumps do not select for the lipid composition that provides optimum activity there are, nonetheless, a number of SERCA isoforms and spliced variants that display differential targeting. It is clear that each subtype is extremely important and defects in expression can lead in to specific muscular disease phenotypes (e.g. Brody and Darier disease).

Gerrit Van Meer (Amsterdam) expanded on the potentially important roles of membrane lipids in the functioning of the ER and Golgi. In an interesting overview, he described how the thin membrane of the ER matures into the thicker membrane of the Golgi, as the concentrations of cholesterol and sphingolipids increase. He also raised many interesting points on which little information is available. For example, how is the lipid composition of the ER membrane regulated? Also, bearing in mind the high proportion of non-bilayer-forming lipids present (e.g. di-unsaturated phosphatidylserine), is the ER membrane a bilayer? Are domains of lipids present – and, if so, in which leaflet?

Patrizia Paterlini-Bréchot (Pasteur Institute) further discussed the role of SERCA pumps in disease. Explaining how *in vivo* Hepatitis B virus (HBV) DNA integration into the SERCA 1 gene leads to apoptosis

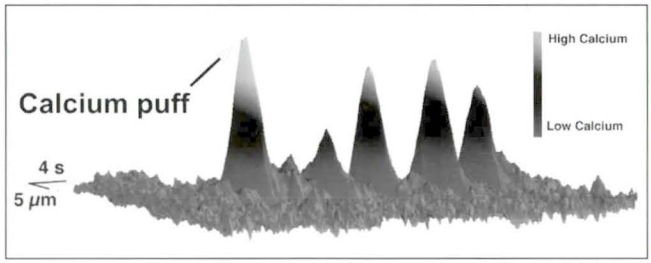
hyperexpression of HBV-SERCA I was only observed in tissues with tumours. Analysis of the HBV-SERCA I sequence showed that the N-terminal was replaced by almost a complete HBV X protein and that the C-terminal was truncated and lacked five of the six Ca^{2+} binding domains. GFP-SERCA chimeras allowed observation of its location in the ER. On viral infection, the exchangeable pool of Ca^{2+} was reduced by ~30% and cells became arrested in Go-G1 leading to a higher rate of apoptosis, suggesting a role for mutated Ca^{2+} pumps in cell transformation.

The ER membrane itself was hijacked by another virus: African swine fever virus described by **C. Netherton** (Pirbright). ASFV is a large double-stranded DNA virus that causes a lethal haemorrhagic disease in pigs. Interestingly, electron microscopy revealed the presence of two internal membranes in mature intracellular virions and in all structural intermediates, which is acquired by wrapping of the virus in membranes derived from the ER cisternae. Two viral genes, XP124L and Y118L were found to encode for proteins with KDEL-like motifs for retention in the ER. Retention of the protein product of XP124L and its packaging into the virion from the ER was thus consistent with the presence of two membranes in the viral envelope.

The role of ER as the main Ca^{2+} releasing organelle was highlighted in a series of talks. **Vincenzo Sorrentino** (San Raffaele) described the ryanodine receptor and the effect of ryanodine isoform knockouts in neonatal muscle and central nervous system of mice. The evidence provided showed clearly that differential isoform expression in tissue is very important for function. However, it is not clear how they bring about their phenotypes. **Colin Taylor** (Cambridge) presented interesting work investigating the question of how the IP3 receptor (IP3R) can be regulated by Ca^{2+}. Using a rapid perfusion set up he demonstrated how the IP3R becomes rapidly, partially activated by IP3 so that the channel has a lower conductance for Ca^{2+} and a higher affinity for IP3. In the absence of Ca^{2+} the latency in channel opening is around 300 ms. However, introduction of Ca^{2+} shortens the latency, suggesting that IP3 binding reveals a Ca^{2+} binding site which must be occupied to open the channel. If instead Ca^{2+} is applied before IP3, the receptor is inhibited. This mechanism generates lateral inhibition, since Ca^{2+} released from a single channel will not be able to inhibit the channel itself, but will block its immediate neighbours. This gives rise to a possible explanation for the well known and mysterious phenomenon of quantal calcium release.

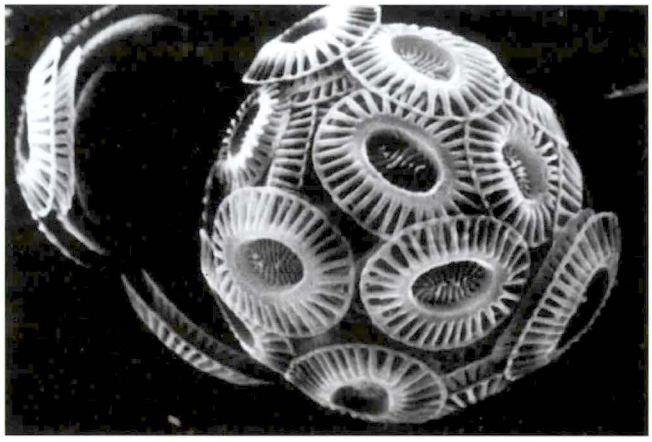
Martin Bootman (Babraham) further emphasied the idea of quantal release with a description of Ca^{2+} puffs in HeLa cells which comprise a ~100 nM Ca^{2+} increase with a diameter of ~6 μM. This pacemaker release can lead to recruitment of other sites and propagation of a wave. ~ 60–70% of the pacemaker sites are localised to the nucleus and sometimes observed on the nuclear membrane itself. Even more interestingly ~50% of the time these pacemaker sites remain fixed and independent of the agonist used to evoke release. **Sophie Brind** (UCL) gave a clear account of a study on the regulation of the expression of IP3 receptors at fertilisation and in early development of mouse oocytes. In experiments to find the trigger for the downregulation of type I IP3R which occurs within 4 hours of fertilization, when pronuclei form and Ca^{2+} oscillations cease, a number of factors were ruled out, viz. activation of Ca^{2+}-dependent enzymes, inhibition of protein synthesis, and egg activation itself. Ca^{2+} transients were apparently also unnecessary. However, cysteine proteinases may be involved. It was suggested that the binding of IP3 to the receptor might trigger downregulation.

The importance of other ion channels in intracellular membranes (e.g. K^+/Cl^- channels) was highlighted by



A series of elementary calcium release signals known as 'calcium puffs' recorded using confocal microscopy of a fluo-3-loaded human bronchial epithelial cell. Each calcium puff is a localised increase in calcium arising from the opening of a few clustered channels on the endoplasmic reticulum. The change in calcium concentration is denoted by the shade (light grey denotes high calcium, whilst mid and light grey denotes low calcium concentrations), and the height of the surface of the image. Courtesy of Martin Bootman.

Adam Szeweczyk (M Nencki Institute), who reminded everyone of their abundance. Primary functions for these channels include charge compensation, formation of pH gradients and regulation of organelle volume. **Andrew Thomas** (New Jersey) detailed the use of compartmentalised Ca^{2+} indicator dyes to investigate the intimate relationship between ER and mitochondria, which acts as a Ca^{2+} sink as the ER releases Ca^{2+}. The close proximity was exemplified by the fact that buffering the cytosolic Ca^{2+} released by the ER does not prevent mitochondrial uptake. This coupling of the Ca^{2+} release channels of the ER and mitochondria has the potential to provide a feedback mechanism on the receptors.



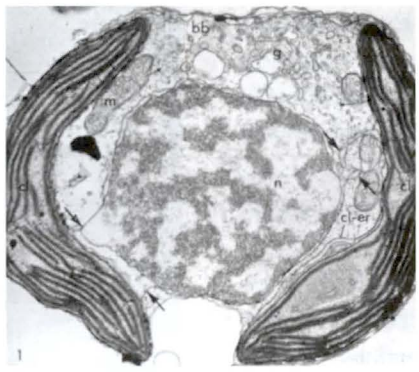
SEM of a coccolithophore (*Emiliania huxleyi*) showing the calcite scales that are produced internally (in a Golgi-derived vesicle) and then exocytosed to the cell surface.

The essential role of Ca^{2+} for polarised growth and osmoregulation in *Fucus* was detailed by **Colin Brownlee** (Marine Biological Association). Within the growing rhizoid tip there is a close correlation of Ca^{2+} release hotspots with actin focal points. Hypo-osmotic shock induces spatial Ca^{2+} release, which spreads from the apex to around the nucleus in response to severe osmotic shock. However, moderate shock displays a delay followed by an increase restricted to the nucleus. Spatial Ca^{2+} release induced by caged IP3 have different effects on the cell cycle with only Ca^{2+} release near the nucleus inducing a delay. The talk ended with a description of an intriguing animal (see photo), a coccolithophore which sustain extremely high rates of transcellular Ca^{2+} transport. This process of calcification plays an important role in the global $C0_2$ cycle since they are the dominant phytoplankton in the worlds oceans. **Jurgen Denecke** (Leeds) continued the focus on plants. Before describing work on complex formation between calreticulin and BiP, he enthusiastically expounded the virtues of plant systems for molecular biology. Although calreticulin may be secreted from mammalian cells under stress, it is not secreted at all in plants where it occurs in the ER as a stable complex with BiP, even though the calreticulin is apparently neither unfolded nor misfolded. This complex differs structurally from that between BiP and misfolded proteins. The calreticulin-BiP complex may act as a store from which BiP is released to interact with misfolded proteins during ER stress, after which it forms a complex with CRT again.

Work on the structure of the ER in different kinds of eggs (*Xenopus*, star fish, sea urchin, and mouse) and their response to Ca^{2+} release was reported by **Mark Terasaki** (Connecticut), who employed a fluorescent lipophilic dye, which stains all cellular organelles locally, but spreads only through the continuous network of the ER. Various kinds of structural changes in the ER were always seen during oocyte maturation. By contrast, two types of behaviour were seen on fertilization. In some species, the ER was fragmented on fertilization, and the dye then failed to spread. This behaviour was associated with the induction of a single Ca^{2+} wave. However, in other species, which exhibit multiple Ca^{2+} waves on fertilization, there was no accompanying change in the structure of the ER.

Martin Latterich (Salk Institute) proposed how the organelle membranes might rejoin. Although much is known about the SNARE complex mechanism for vesicle-membrane fusion little is known about how membranes of organelles fuse. Using *in vitro* membrane fusion assays to reconstitute ER membrane fusion, Cdc48p was shown to be required for ER and nuclear envelope fusion in yeast. Evidence for the role of other proteins (Ufe1p, Shp1P and Ufd3p) was presented which form a t-SNARE complex, which is required for the priming or initiating event of ER membrane fusion. Work on the regulation of vesicle movement, with real time videos of ER membrane tubule movement in extracts of *Xenopus* eggs, was shown by **Viki Allan** (Manchester). Observations indicated that dynein is

involved in movement towards the minus-end of microtubules near the centre of cells (inhibited by vanadate), while kinesin participates in movement to the plus-end located at the cell periphery. It was suggested that such movements might contribute to the establishment of different ER domains.



SEM of a coccolithophore showing cortical ER around the cell periphery and cholorplast ER which is contiguous with the nuclear envelope (arrows). Courtesy of J.Green, MBA

Rainer Pepperkok (EMBL) discussed the movement of membranes between ER and the cis-side of the Golgi complex. Striking videos of fluorescently-labelled vesicles showed the transit between the two sites. He described experiments designed to elucidate the number of steps involved in the trafficking process, and presented new information on the direction taken by COP I coated vesicles. The movement of COP I labeled vesicles was observed to be approximately 55% anterograde, 8% retrograde, 10% looping, and 30% resting. This was consistent with COP I replacing COP II in vesicles, arriving from the ER, at an intermediate compartment (transport complex) prior to their onward movement to the Golgi. Formation of the transitional ER and adjacent golgi stacks was described by **Ben Glick** (Chicago). The main question was why the Golgi is present in stacks in *Pichia pastoris* but dispersed in *Saccharomyces cerevisiae*? In *P. pastoris*, Golgi stacks are adjacent to discrete sites of transitional ER, which are associated with the plasma and nuclear membranes and which contain both COP II coat proteins and the guanine nucleotide exchange factor, Sec12p, that is essential for vesicle budding. This distribution may be responsible for the generation of coherent Golgi stacks. By contrast, in *S. cerevisiae*, COP II vesicles appear to be present throughout the cytoplasm and Sec12p is distributed throughout the ER, thus giving rise to a dispersed Golgi.

Taina Suntio and colleagues (Helsinki) described the role of a COP I-independent pathway in secretion of heat shock protein Hsp150, and invertase in yeast. The ER form of Hsp150 consists of distinct domains. By fusing heterologous proteins to these domains and expressing them in sec21-1 and sec21-3 mutants with temperature-sensitive mutations in the COP I subunit, they showed that a repetitive region sorts HSp150 for COP I-independent exit.

Sanford Simon (Rockefeller) gave a rapid-fire overview of the biogenesis of membrane proteins with particular reference to opsin and P-glycoprotein, which have their N-terminus on the outside and inside of the ER respectively. True intermediate structures are formed during the synthesis of opsin which are attached to ribosomes and to tRNA but have also been glycosylated in the ER. With P-glycoprotein, each latent transmembrane segment sequentially translocates across the ER membrane as it emerges from ribosomes. However, >90% of the protein appears to be held in the membrane only by salt-sensitive, electrostatic bonds in an aqueous-accessible compartment until it is fully released from ribosomes when it then becomes integrated into the bilayer. The attention of the talks then shifted to how the formation of a disulphide bond by members of the protein disulphide isomerase (PDI) family is essential for the maturation of newly synthesised proteins within the ER.

Maurzio Molinari (Swiss Federal Institute of Technology) reported the isolation of transient intermediates produced during the process. In this work, maturation in the ER of radiolabelled SE1 and p62 surface proteins of the Semliki virus was halted by alkyation with NEM. Products were separated on non-reducing 2D gels, and then immunoprecipated with specific antibodies to lectins. Observations were reported on the transient, mixed disulphides that were obtained which variously contained calnexin, calreticulin, and Erp57. PDI, calnexin and calreticulin, as chaperones, was mentioned by **Stephen High** (Manchester). He provided a comprehensive and very accessible account of work on the maturation of newly synthesised glycoproteins within the ER, in which a cross-linking approach was used to identify the chaperones involved. Data were presented to show that Erp57, which has 40% homology with protein disulphide isomerase (PDI), interacts with calnexin and calreticulin in the absence of their glycoprotein

substrate to form a complex that specifically modulates glycoprotein folding. Binding of the complex to the substrate is transient, and the substrate is released following trimming by glucosidase II. This is the favoured pathway for N-glycosylated proteins in the ER.

Benjamin Kaminer (Boston) described in sharp focus the PDI found in the ER of sea urchin eggs, termed calcistorin/protein disulphide isomerase (PDI). It has a 55% sequence identity with mammalian PDI, has PDI activity, and a high capacity and low affinity for Ca2+ which is apparently dependent on the number of pairs of carboxyl groups in the molecule. Experiments with transfected Chinese hamster ovary cells showed that the protein functions as a Ca^{2+} storage protein in living cells. 5 mM Ca^{2+} increased the PDI activity of both egg calcistorin/PDI and a C-terminal truncated mutant, and Dr Kaminer suggested that Ca^{2+} may be needed to activate mammalian PDI.

The importance of correctly folding protein in the ER was emphasised by **Randall Kaufman** (Michigan). He described the complex way in which cells respond to ER stress not only by up-regulating the synthesis of chaperone molecules in order to increase folding capacity, but also by activating pathways to induce apoptosis. Ire1p, which has kinase and endoribonuclease activities and occurs both in the ER and in the nuclear envelope near the pore complex, is apparently the sensor for the transcriptional response to unfolded proteins. He also suggested that 'unfolded protein' response is a misnomer for a glucose-deprivation response since glycosylation fails in the absence of glucose, and this results in the accumulation of unfolded proteins as a downstream event. The role of calreticulin in abnormal human neutrophils, which is upregulated in rheumatoid neutrophils was alluded to by **David Llewellyn** (Cardiff). By over expressing GFP-calreticulin it was reported that there was an increase in store capacity, a reduction in Ca^{2+} influx and lumenal ER Ca^{2+} and no effect on prolonged agonist induced Ca^{2+} release. However, transient Ca^{2+} release was significantly reduced.

An investigation to determine whether the procollagen-binding stress protein, HSP47, plays a role in protein folding, in the retention of trimeric molecules of unhydroxylated collagen in the ER, or in helix stabilisation was described by **Mohammed Tasab** (Manchester). Since hydroxyproline is not evenly distributed along the polypeptide helix, there is potential for unfolding particularly as the melting temperature of collagen is only a few degrees above body temperature. Data were presented which indicate that Hsp47 interacts specifically with trimeric molecules to stabilise them against local unfolding.

Richard Steinhardt (Berkeley) delivered the plenary lecture on mechanism of cell membrane repair at the close of the meeting. This was a fascinating talk that was delivered in a deceptively low-key fashion in terms of simple experiments which "a full-professor, who is not very accustomed to doing experiments, could undertake!" Resealing of a damaged plasma membrane was shown to require fast exocytosis, and the entry of Ca^{2+} at the wound site that results in vesicle recruitment, transport, docking and fusion. Phorbol esters do not inhibit the initial resealing. However, facilitated resealing after a second wounding is mediated by protein kinase C, which is activated by Ca^{2+} that entered in the first wounding, and involves the formation of new vesicles from the Golgi. A take-home message from this talk was that the fusion of intracellular vesicles with the plasma membrane for repair purposes probably arose early in cellular evolution and this mechanism was later developed for more sophisticated purposes, such as the release of neurotransmitter molecules.

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14th Meeting of the European Cytoskeleton Forum

Roy Quinlan and Alan Prescott

The 14th meeting of the European Cytoskeleton Forum was organised by Claudina Rodrigues-Pousada, Helena Soares and Luisa Cyrne and held in Oeiras, a coastal resort north of Lisbon, Portugal. Sun, sea and sangria – who said science isn't fun?

The meeting included a special symposium to celebrate Klaus Weber's 60th birthday and to mark the extensive contribution he has made to cell biology and particularly to all aspects of the cytoskeleton. It was the pioneering work of Klaus and Mary Osborn that demonstrated the potential of immuno fluorescence microscopy to cell biology and allowed us to visualise the cytoskeleton in cells for the first time. Immunofluorescence microscopy is still one of the most important tools to modern cell biology, especially now with the advent of GFP-tagged proteins to follow protein dynamics and localisations in living cells, a theme that was strongly represented in the meeting.

Another dominant theme was the interaction between the different cytoskeletal elements, intermediate filaments, microtubules and actin. Although recently the Weber laboratory has concentrated on the evolution of cytoplasmic intermediate filament proteins from their lamin ancestors, the integration of the different cytoskeletal elements is a well-versed theme in the Weber lab as seen by the breadth of publications in all things cytoskeletal . The identification of lamin-like members of the cytoplasmic intermediate filament family in early chordates was the theme of Klaus's opening talk to the Forum. This 'explosive' beginning was appropriately followed by a party at the Oeiras Gunpowder Factory!

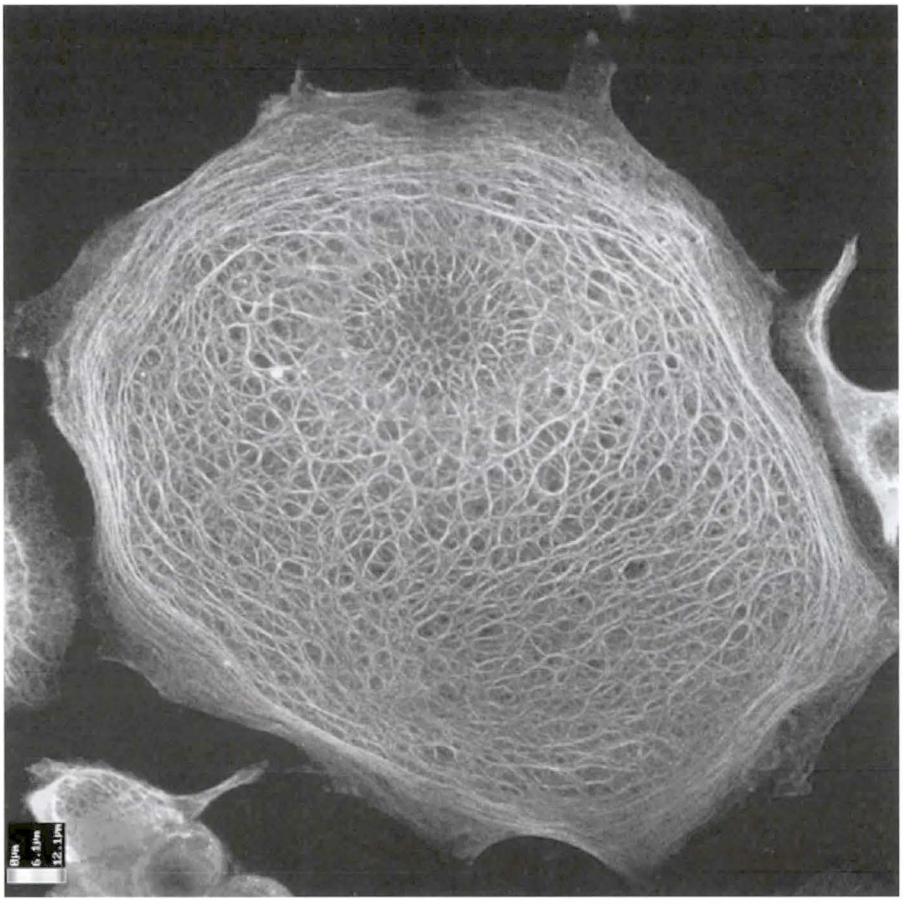
The first session had a 'Weber' theme in that the speakers were associated with Klaus's laboratory and ranged in topics from nuclear compartments and splicing events (**Joan Steitz**; Yale) to ERM proteins (**Tony Bretscher**; Cornell). **Paul Matsudaira** (MIT) discussed results that implicated the actin-binding protein fimbrin in an association with vimentin at focal adhesions. These examples illustrate other dominant themes of the meeting: the importance of the cytoskeleton in signal transduction and the developing interest in the cross-talk between different elements of the cytoskeleton.

From a technical point of view it is clear that the use of GFP fusion proteins in living cells are revealing the way the cytoskeleton integrates into the machinery of the whole cell. A pioneer in this area has been **Bob Goldman** (Chicago) and his talk focussed on the dynamic nature of the nuclear lamins and their association with sites of DNA replication. **Harald Herrmann** (Heidelberg) has used a novel approach to probe nuclear structure and internal compartments, by engineering the expression of vimentin, a cytoplasmic intermediate filament protein, to the nucleus. With this approach, he has begun to examine the nature of the 'interchromosomal domain'. Surprisingly, whether as filaments or as aggregates, vimentin in the nucleus had little effect on the organisation of other nuclear compartments and also failed to disrupt normal mitosis.

Roland Foisner (Vienna) described the different roles of LAP2α and β in the nuclear lamina assembly and their associations with lamins and chromatin. It was one of four talks from the Biocentre in Vienna (**Wiche**, **Propst** and **Eger**) that explored the interacting partners of cytoskeletal proteins (plectin, MAP1A and B and β-catenin respectively) and their role in determining function.

A number of talks described experiments aimed at dissecting the β-catenin signalling pathway: **Hans Clevers** (Utrecht) showed that members of the

TCF/LEF family, that are only expressed in lymphoid cells in the adult, are critical effectors of β-catenin signalling during early development. **Avri Ben-Ze'ev** (Israel) also showed that the β-catenin/LEF-1 complex could be important in colon cell transformation through an interaction with cyclin D1. **Jurgen Behrens** (Berlin) identified a new β-catenin binding protein, conductin, that promotes β-catenin degradation.



This human epithelial cell (MCF7) has been stained with anti-HSP27 antibodies. Note the cytoskeletal pattern of the staining which from other studies (Perng et al.1999 J Cell Sci, 112, 2099-2112) we know corresponds to the keratin intermediate filament network.These data indicate that small heat shock proteins are normally associated with intermediate filaments in growing, unstressed cells. The fluorescence signal has been coded for depth.

The role of the ERM (Ezrin-Radixin-Moesin) family of proteins in the regulation of the actin cytoskeleton and their association with tumour suppressor proteins were covered in talks by **Tony Bretscher** (Cornell), **Richard Lamb** (London) and **Alexis Gautreau** (Paris). Richard Lamb collaborating with Alan Hall described a new application of the Trojan horse principle to study protein function. This involved the localised inactivation of tagged proteins and their associated complexes in one region of the cell by the release of free-radicals generated by the laser-activation of the fluorochrome-tag on the protein ('Chromophore Assisted Laser Inactivation'). He used this technique to explore the role of hamartin (TSC1) in actin reorganisation. Hamartin was identified from 2-hybrid assays as a protein that interacts with merlin, the tumour suppressor protein in the ERM family.

The link between tumour suppressor activity and the cytoskeleton was a recurring theme and was addressed by talks from **Kathrin Schülter** (Braunschweig) and **Wolfgand Deppert** (Hamburg). **Anne Ridley** (London) covered how the signals coming from the Rac/Rho GTPases modulate the actin-plasma membrane interaction during such actin-driven processes as cell spreading and migration. Migration has traditionally been studied using the Bowden chamber (2 chambers separated by a filter); however,Anne described a novel chamber designed by Graham Dunn (London) that allows the migration of microinjected cells in response to cytokines to be video recorded. This has allowed Anne to inject dominant negative proteins of the Rho family of GTPases in order to determine which of the family members are important for chemotaxis. Cdc42, for instance, is not necessary for macrophage migration but is required for chemotaxis in response to CSF-1. This process also requires PI3K activity as shown by using both drug and neutralising antibodies.

More links between the cytoskeleton and cell signalling pathways are being discovered and was

reflected in the attention these areas received at the meeting. The Rho GTPase family's interaction with the actin cytoskeleton has already been mentioned and **Tomasek** (Oklahoma) presented data to suggest that Rho is involved in myofibroblast contraction through regulation of myosin light chain phosphatase activity rather than calcium acting on the kinase as is the case in smooth muscle cells. **Leterrier** (France) implicated cAMP and PIP2 in the regulation of the weak interactions between microtubules mediated by MAP2. **Clive Lloyd** (Norwich) gave a flowing talk on plant microtubules and their role in plant growth, cell wall deposition as well as plant MAPs and the features that they share with animal cell equivalents. The **Joël Vandekerckhove** (Ghent) presention was a tour de force in mass spectrometry and proteomics to identify those actin-binding proteins involved in *Listeria* motility. Clearly this is a rich vein of research of which we will hear more at the next meeting.

Two video-enhanced talks, one from **Andrew Matus** (Basel) and a second from **Vic Small** (Austria), confirmed (if you needed confirmation!) that using Green Fluorescent Protein fusions is definitely the method of choice when it comes to looking at dynamic cell processes. Using GFPs with two different fluorescent tags fused to actin and MAP2, the Matus lab looked at the cytoskeleton in dendritic spines of neurones. These spines undergo rapid shape changes that are actin-driven when unstimulated but become stabilised upon synaptic excitation. This suggests that these spines may be the molecular basis of neuronal plasticity and possibly the site of action of anaesthetics rather than the microtubule cytoskeleton of the dendrites which the MAP2 probe showed to be somewhat static. This idea is not difficult for cytoskeleton-minded people to accept and this talk was certainly one of the high points in the meeting.

The Small talk was anything but, and concentrated on cytoskeletal dynamics during cell movement using fish fibroblasts as the model system. These cells were recorded with fluorescently labelled tubulin and GFP-tagged zyxin to visualise the formation of focal contacts during progression. This dual labelling protocol demonstated the requirement for microtubules to 'mature' focal contacts. During the post-video discussions Paul Matsudaira pointed out that the inhibitor BDM which is sometimes touted as a specific inhibitor of the actin/myosin system is in fact a rather non-specific modifier of Arginine residues and should therefore be used with caution!

The GFP technology was also exploited by **Alison North** (Manchester) to demonstrate that both intermediate filaments and their associated cell–cell junctions, namely desmosomes, are dynamic structures. Using GFP-desmoglein and Cy3-conjugated desmoplakins Alison has shown that junction precursors are associated with the keratin intermediate filaments in sub-confluent or low calcium MDCK cells and these precursors migrate to the cell periphery to assemble the desmosomes. These junctions remain in a constant state of flux and are not at all static. There really is no substitute for 'seeing' in order to 'believe'!

The approach of using two fluorescently conjugated proteins to study protein dynamics has led to the use of fluorescence ratio imaging to demonstrate the close association of two proteins in the same structure. **Benny Geiger** (Israel) has used this technique to show that there are at least two types of focal contact in cells, classical focal adhesions rich in vinculin, paxillin and phosphotyrosine and tensin-rich fibrillar adhesions. The fibrillar adhesions are dynamically associated with the focal contacts and move towards the cell centre during maturation in an actomyosin dependent fashion.

The importance of substrate adhesion to cells was emphasised by two other talks by **Alan Horwitz** (Virginia) and **van de Water** (Leiden). Horwitz showed that the integrin ratio determined the decision to proliferate or differentiate in myoblasts, mediated via paxillin/MAPK/FAK signalling network. van de Water showed that loss of focal adhesion in kidney epithelial cells by treatment with the neurotoxin DCVC led to apoptosis via effects on the phosphorylation status of FAK, paxillin and the adducins. The implication from these experiments is that perturbation of the actin cytoskeleton during

apoptosis would reorganise the focal contacts and prevent cell adhesion and thus decrease the chance of the cell being able to reverse the apoptotic process.

Among the many cytoskeleton-associated proteins discussed at the meeting, the involvement of protein chaperones in the correct folding of the cytoskeletal polymer sub-units and in coordinating dynamic interactions of the three filament systems was timely. Talks by **Sally Lewis** (New York), **Julie Grantham** (London), **Katja Siegers** (Glasgow), **Helena Soares** (Oieras) and **Christophe Ampe** (Ghent) revealed the crucial role for chaperonins and their cofactors in actin and tubulin folding. Soares (Lisbon) showed that the chaperonin complex was present in the ciliate Tetrahymena suggesting that it has co-evolved with actin and tubulin even to the extent that chaperonin gene expression is regulated by anti-microtubule drugs such as colchicine and taxol. Christophe Ampe's group (Ghent) have identified actin and tubulin residues necessary for interaction with the chaperonin complex and have suggested that one of the cofactors, prefoldin, exposes these residues during the formation of the folding complex.

Chaperones are not only important in folding cytoskeletal monomers but are also involved in the regulation of polymer interactions. This was demonstrated in a talk by **Paul van den IJssel** from our group in Dundee. He showed data from a recently discovered human mutation in αB crystallin, a member of the small heat shock family of chaperones, which causes DRM (desmin related myopathy) and cataract, both characterised by inappropriate intermediate filament bundling. *In vitro* the mutant chaperone is unable to prevent filament-filament interactions that lead to gel formation and induced bundling of the filaments when included in filament assembly assays.

This illustrates the diversity of topics covered at the Cytoskeleton Forum from animal cells to *Drosophila* to plant cells and from the cytoskeleton to the nucleoskeleton. This report has inevitably not been able to acknowledge everyone who took part in this successful meeting. It is commendable that many young scientists attended and contributed, but this has always been a feature of these meetings. The poster sessions were lively and are clearly an important aspect in the success of the meeting. This is the place to foster an interest in the cytoskeleton, because this meeting is not just for the aficianados of the cytoskeleton. After all, this topic touches all aspects of cell behaviour. See you next year in Ghent!

Roy Quinlan and Alan Prescott
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Dundee
DD1 5EH

BSCB/BSDB Joint Spring Meeting 2000

University of Warwick, 28-31 March

General Information

Dates

Arrive Tuesday 28 March in time for dinner (19.30)
Depart Friday 31 March after lunch (13.00)

Conference site and travel information:

The conference will be held entirely within the University of Warwick*. Further information about the site is available on: *www.warwick.ac.uk*. Full details of travel to the University of Warwick, and further instructions about the conference and site will be sent to registrants approximately 4 weeks before the conference. A booklet *Essential Information for Visitors* will be included. Information will also be available on the Warwick Conference website from 1 November.

**The conference site has been the winner of 'Best Academic Conference Venue' for 7 out of the last 10 years.*

Registration

The number of registrants is limited. In the event that the meeting is over-subscribed, priority will be given to those who present posters. The deadline for registration forms and abstracts is 28 January 2000; those registering after this date are subject to a strictly enforced **late registration penalty of £35**. The accompanying registration form is also available at: *www.kcl.ac.uk/links/bscb.html*

Meeting Charges

The all-inclusive fees are as follows:
Resident BSCB/BSDB member
standard accommodation – £282
ensuite accommodation – £338
Non-resident BSCB/BSDB member – £200
Non-BSCB/BSDB member ADD £35
BSCB/BSDB member PhD students DEDUCT £25
BSCB/BSDB members presenting a poster DEDUCT a further £25 (only one deduction per poster).

The fee for residents covers accommodation and all meals for the duration of the conference, including the conference dinner. The fee for non-residents includes all meals for the duration of the conference including the conference dinner, but accommodation must be arranged independently. Registrants who are not members of the BSCB or BSDB can apply to join well in advance of the 28 January deadline to take advantage of the member price. Application forms to join BSCB are available on page 29 or can be found at: *www.kcl.ac.uk/kis/schools/life_sciences/biomed/bscb/ bscbapply.html*

Honor Fell Travel Awards for BSCB Members

PhD students and postdocs should remember that Honor Fell awards are available to cover conference costs in part. An application form should be submitted **directly to the BSCB** independently of registration (see page 28 or *www.kcl.ac.uk/kis/schools/life_sciences/biomed/bscb/honorfell.html*

Posters and abstracts

There will be a joint poster session between the BSCB and BSDB. Poster presentations from students who are members of the BSCB or BSDB and who have not been awarded a higher degree at the time of registration will be eligible for special poster awards. The top prizes will be attendance at the ASCB or ASDB meeting in the USA. In addition, abstracts may be selected for oral presentation. Please note that students should state clearly when submitting their poster that it is for consideration for the poster prize (see instructions below).

Abstracts and instructions for abstract submission

Abstracts must be submitted by email as text-only messages (no attachments) to: *abstract@hosp.warwick.ac.uk*
The subject field should contain the name of the registrant and an indication of whether your abstract is associated with BSCB or BSDB as follows:
Subject: surname-firstname/BSC/DB
The text field should contain:
Top line: abstract title in capital letters
Next line: author names – **please indicate if you are a student presenter**
Next line: affiliation
Leave one line blank
Type abstract in one paragraph; maximum 300 words.
Abstract deadline is 28 January 2000.

All conference information can also be found on:
www.kcl.ac.uk/links/bscb.html

For queries, please contact the University of Warwick Hospitality on: 0247 652 3755

BSCB/BSDB Joint Spring Meeting 2000

Registration

The number of registrations is limited and in the event that the meeting is oversubscribed, priority will be given to those presenting posters.

Registration forms should be sent as detailed below and abstracts by e-mail as described in 'General Meeting Information'. Both must be received by 28th January 2000.

Registration is handled by The Conference Management Service at the University of Warwick and will not be processed without receipt of a cheque or money order in pounds sterling made payable to The University of Warwick, or appropriate credit/debit card details.

Registration checklist:

- registration must be made in writing; fax copies will not be accepted. The form should indicate your name, phone number, fax number, e-mail address and dietary requirements
- either enclose a Sterling cheque or money order for the relevant amount, payable to 'The University of Warwick, or enclose credit card details.

Name .....

Title Prof / Dr / Mr / Ms

Address .....

.....

.....

.....

Telephone .....

Fax .....

E-mail .....

Please indicate details:

Resident BSC/DB member £282 ❑

Resident BSC/DB (ensuite) £338 ❑

Non-resident BSC/DB member £200 ❑

Non-BSC/DB member ADD £35 ❑

Late fee after 28 Jan ADD £35 ❑

BSC/DB Student DEDUCT £25 ❑

BSC/DB Poster submission DEDUCT £25 ❑

please state...

Vegetarian ❑

Other dietary needs ❑

Any other requirements ❑

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Tick if Poster abstract emailed ❑

Payment Method

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Expiry date ☐☐☐☐

Switch issue number ☐☐

Signature:

Date:

This form should be sent to: Sarah Edwards, BSCB/BSDB Conference Management Service, Hospitality Services, Rootes Social Building, Gibbet Hill Road, Coventry, CV4 7AL Tel: 0247 652 3755

BSCB Symposium – *Cell Biology in Disease*

Tuesday March 28 *Registration*

Wednesday March 29

Morning sessions

Plenary Judah Folkman (Boston)

Session 1
Angiogenesis
B. Fingleton (Nashville)
B. Olsen (Boston)
S. Courtneidge (San Francisco)
D. Edwards (UEA)

Session 2
Cellular Basis of Inherited Disease I
C. Higgins (London)
K. Tryggvason (Stockholm)
A. Shaw (St Louis)
S. Tucker (Oxford)

Afternoon sessions

BSCB Hooke Medal Lecture Anne Ridley (London)

Session 3
Cytokine Signalling in Disease
B. Foxwell (London)
L. O'Neill (Dublin)
M. Welham (Bath)
P. Heinrich (Aachen)
plus speakers selected from abstracts

Session 4
Cytoskeleton and Disease
B. Lane (Dundee)
M.-F. Carlier (Gif S. Yvette)
K. Steele (Nottingham)
D. Kwiatkowski (Boston)
plus speakers selected from abstracts

Evening

Poster Session

Thursday March 30

Morning Sessions

Plenary Bruce Edgar (Seattle)

Session 5
Cellular Interactions in Cancer
J. Behrens (Berlin)
D. Garrod (Macnhester)
G. Christofori (Vienna)
D. Critchley (Leicester)

Session 6
Neurodegeneration
B. Anderton (London)
W. Annaert (Leuven)
M. Mattson (Kentucky)
D. Bredesen (California)

Afternoon sessions

Session 7
The Pathology of Cytokines
C. Evans (Pittsburgh)
P. Clapham (London)
J. Westwick (Horsham)
F. Balkwill (London)
plus speakers selected from abstracts

Session 8
Cellular Basis of Inherited Disease II
J. Bonaventure (Paris)
C. Kinnon (London)
S. Winder (Glasgow)
G. Jones (London)
plus speakers selected from abstracts

18:00:19:00 *BSCB and BSDB Annual General Meetings*

20:00 *Conference dinner*

Friday March 31

Morning sessions

Plenary Martin Raff (London)

Session 9 *Wound Healing*
M. Ferguson (Manchester)
P. Martin (London)
R. Clark (Stonybrook)
S. Werner (Zurich)

BSDB Symposium – *Pattern formation and control of cell number*

Tuesday March 28 *Registration*

Wednesday March 29

Plenary Judah Folkman (Boston)

Session 1 (morning)
Pattern formation and proliferation
Tim Hunt (ICRF South Mimms)
Christian Lehner (Bayreuth, Germany)
David Cobrinik (Columbia, NY)
Laura Johnston (Seattle)

Session 2 (afternoon)
Pattern formation and proliferation (cont.)
Steve Cohen (EMBL)
Jim Murray (Cambridge UK)
Barbara Thomas (NCI. Bethesda)
Fred Berger (Lyon)
Monica Murakami (NCI, Bethesda)
plus speakers selected from abstracts

Evening *Poster session*

Thursday March 30

Plenary Bruce Edgar (Seattle)

Session 3 (morning)
Cell size/numbers
Ernst Hafen (Zurich)
Sally Leevers (Ludwig, London)
Peter Lawrence (Cambridge UK)
plus speakers selected from abstracts

Session 4 (afternoon)
Cell size/numbers (cont.)
Ben Scheres (Utrecht)
Richard Gomer (Rice, Texas)

Proliferation and death in organogenesis and CNS development
Chris Henderson (Marseilles)
Irma Thesleff (Helsinki)
Yves Barde (Martinsried, Germany)
Bill Harris (Cambridge UK)

18:00:19:00 *BSCB and BSDB Annual General Meetings*
20:00 *Conference dinner*

Friday March 31

Plenary Martin Raff (London)

Session 5 *Pattern formation and cell death*
Hermann Steller (MIT, Massachussets)
Barbara Conradt (Martinsried, Germany)
Gerard Evans (UCSF, California)
Jon Minden (USA)
Jeremy Brockes (UCL, London)

Forthcoming meetings

BSCB Autumn meeting: The Cell Biology of Apoptosis
Heriot Watt University, Edinburgh
10–13 September 2000
Organisers: Paul Clarke, Bill Earnshaw, Anthony Metcalfe, Ted Hupp

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BSCB/BSDB Spring Meeting 2001
Sussex University
3–6 April, 2001
Organisers: David Garrod, Charles ffrench-Constant, Alan Hall

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10th TENOVUS-SCOTLAND SYMPOSIUM: GENE EXPRESSION AND DISEASE
Royal Scottish Academy of Music and Drama, Glasgow.
10–12 April 2000
The triennial 3-day Tenovus-Scotland Symposium covers a broad range of subjects relevant to the topic of *Gene Expression and Disease*. The main foci are the six scientific sessions:

- Genome Instability,
- Transcription Factors,
- Nucleic Acid Modification,
- Protein Processing,
- Genes and the Environment,
- Gene Therapy.

Final Programme, abstract form and registration information are available on the symposium webpage, www.gla.ac.uk/Acad/IBLS/molgen/tenovus/tenovus_2000.html

Alternatively, please contact the Symposium Secretariat: Dr. Uta Böger-Brown, BioMedEx, 22 Allan Rd, Killearn, Glasgow G63 9QE UK; Phone: +44 (0)1360 551 082; Fax: +44 (0)1360 551 083; E-mail: uta@biomedex.demon.co.uk

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The Biology of Thrombospondins and Other Modulatory Extracellular Matrix Proteins
University of Wisconsin, Madison, WI, USA
4–8 June 2000
Organiser : Deane Mosher, (Wisconsin), Josephine Adams, (UCL, London)
Registration Information available by Web from Winter 1999.
To join the mailing list contact dfmosher@facstaff.wisc.edu or dmcbjca@ucl.ac.uk

Session topics:

- Modulation of angiogenesis and tumor behavior
- Cellular and molecular mechanisms
- Protein structure, folding and post-translational processing
- Tenascin family proteins
- Modulatory proteins of the extracellular matrix (SPARC, osteopontin, vitronectin, bone sialoprotein, ADAMTS, spondins)
- Tissue functions in homeostasis and disease

There will also be short talks selected from abstracts, poster sessions and a trade exhibit

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European Life Sciences Organization (ELSO) Meeting
Geneva, Switzerland
2–6 September, 2000
Organising committee of ELSO 2000: Denis Duboule and Jean Gruenberg

More detailed information about ELSO and the meeting can be found on www.elso.org

Plenary symposia speakers: Craig Venter (Rockville), David Botstein (Stanford), Matthias Mann (Odense), Frank Grosveld (Rotterdam), Bruce Stillman (Cold Spring Harbor), Richard Treisman (London), Julian Downward (London), Hermann Steller (Cambridge, USA), Bill Earnshaw (Edinburgh), Christiane Nuesslein-Volhard (Tübingen), Peter Gruss (Göttingen), Margaret Buckingham (Paris), Erwin Neher (Göttingen), Martin Schwab (Zurich), Nigel Unwin (Cambridge), Kim Nasmyth (Vienna), Paul Nurse (London), Eric Karsenti (Heidelberg), Don Wiley (Cambridge, USA), Rolf Zinkernagel (Zürich), Antonio Lanzavecchia (Basel), Charles Weissmann (Zürich), Judah Folkman (Boston), Claude Bordignon (Milan).

Minisymposia include: Nuclear architecture and control of gene expression, the machinery for protein degradation, cell adhesion and signalling, structure and function of membrane proteins, angiogenesis, RNA processing, the structure and the function of Golgi complex, endocytosis, the cell biology of neuronal organisation, molecular clocks, regulating the cell cycle, apoptosis and growth control, host-pathogen interactions, developmental mechanisms of generating cell asymmetry, mammalian genetics in the21st century, plant morphogenesis, nuclear import and export, lipids in membrane trafficking and signalling, the cell biology of cancer, cytoskeletal motors and the evolution of developmental mechanisms.

The 4th UK-Japan Cell Cycle workshop on The Regulation of Cell Proliferation
Churchill College, Cambridge University
23 - 26 September 2000.
Organising committee: Ron Laskey (Cambridge), Mitsuhiro Yanagida (Kyoto), Jonathon Pines (Cambridge), Mark Carrington (Cambridge)

The workshop will be limited to 250 people.

Invited Speakers:
From Japan - Mitsuhiro Yanagida (Kyoto Univ.), Takeharu Nishimoto (Kyushu Univ.), Sigeki Mizuno (Tohoku Univ.), Masayuki Yamamoto (Tokyo Univ.), Hiroto Okayama (Tokyo Univ.), Akio Toh-e (Tokyo Univ.), Eisuke Nishida (Kyoto Univ.), Hisao Masai (Tokyo Univ.), Yoshihiro Yoneda (Osaka Univ.), Fumio Hanaoka (Osaka Univ.), Takeo Kishimoto (Tokyo Institute of Technology), Noriyuki Sagata (Kyushu Univ.), Tokiti Miyagawa (Hiroshima Univ.), Minoru Yoshida (Tokyo Univ.), Haruhiko Takizawa (Osaka Univ.), Kazuo Todokoro (RIKEN, Tsukuba), Yasushi Hiraoka (KANSAI, Kobe), Tsuneko Okazaki (Fujita Medical Univ.), Yoshiko Kikuchi (Tokyo Univ.), Yukiko Goto (Tokyo

Univ.), Yoshimi Takai (Osaka Univ.), Shigekazu Nagata (Osaka Univ.), Akio Sugino (Osaka Univ.), Nobutaka Hirokawa (Tokyo Univ.), Junya Kato (Nara Institute of Sci. & Tech.),

From UK- David Lane (Dundee), Bill Earnshaw (Edinburgh), John Diffley (London), Kim Nasmyth (Vienna), Paul Nurse (London), Tim Hunt (London), David Glover (Cambridge), Tony Hunter (La Jolla), Frank McCormack* (San Francisco), Fiona Watt (London), Jerry Hyams (London), Iain Hagan (Manchester), Tony Carr (Brighton), John Kilmartin (Cambridge), Nic Jones* (Manchester), * Not yet confirmed.
Please register your interest at : email ajccc4@mole.bio.cam.ac.uk
postal address The Wellcome/CRC Institute, Tennis Court Road, Cambridge CB2 1QR

The 7th International Congress on Cell Biology in conjunction with the 11th Meeting of the International Society for Differentiation
Gold Coast, Queensland, AUSTRALIA
24-28th September, 2000
The Convenor for the 2000 Congress is Dr Peter French. Inquiries can be directed to him at: p.french@cfi.unsw.edu.au; or fax (+61-2-9361-2391).

Details about the congress venue and a detailed programme can be found on the website for the meeting, www.celldiff.unsw.edu.au

Please note that there is time for more symposia themes to be added. If there are any suggestions for themes for the symposia, please contact the Program Co-ordinator, Prof. Keith Stanley (k.stanley@cfi.unsw.edu.au).

The Theme
The biannual meeting of the ISD will be held for the first time in the Southern Hemisphere. The meeting's theme is *'Cell and Developmental Biology: the next Millennium'*. The program combines basic, applied and clinical biological research, showcasing cutting edge science and highlighting the interface between molecular cell biology and differentiation/development. It is also the first time that the ISD will meet with another organisation. In this case it is the International Federation for Cell Biology. Both societies will be hosted by the Australian and New Zealand Society for Cell and Developmental Biology.

Registration Information
Electronic registration will be open on the congress web site (www.celldiff.unsw.edu.au) after September, 1999. Intending delegates are strongly advised to register and submit abstracts on the internet as this will streamline the registration process.

For those who do not have internet access, contact the convenor (details above).

Registration fees (in Australian dollars) are$495 (students $225), if registering before June 30, 2000 and$595 (students $275) after this date.

Speakers include: Sydney Brenner (Paradigms in molecular and cell biology), Peter Gruss (Development of the vertebrate visual system), Yossi Yarden (Tyrosine kinases in signalling and development), Scott Emr (Phosphatidylinositol lipids and cellular control), Graham Warren (Organelle origin and replication), David Jans (Regulation of nuclear protein import), John Gurdon (Plenary Jean Brachet Lecture), Brigid Hogan (Branching morphogenesis), Linda Buck (Olfactory and pheromone signalling), Pietro de Camilli (Molecules of vesicle formation), Tony Burgess (EGF receptor in cancer), Alan Hall (GTPase control of the cytoskeleton), Vishva Dixit (Cell machinery of apoptosis).
Symposia include: Lineage specification and differentiation, Complex networks of signal transduction, Tissue patterning and organogenesis, Breast cancer, Caveolae and cholesterol in cell signalling and development, Trafficking in disease, Cell interactions and morphogenesis, Cell biology of asthma, Stem cells and differentiation, Molecular profiling of cancer, Gap junctions and ion channels in disease, Membrane traffic and cell signalling.

TECHNIQUES IN MOLECULAR BIOLOGY
University Of Hertfordshire (U.K.)

Information for all courses can be viewed at the Website: www.herts.ac.uk/natsci/STC

Postal address for enquiries: Department of Biosciences, University of Hertfordshire College Lane, Hatfield, Herts AL10 9AB UK.

MOLECULAR PROBES IN DIAGNOSTICS
A one-day lecture course
13 April 2000
Details and application forms from Dr Ralph Rapley – tel:(01707) 285097; fax:286137; e-mail: R.Rapley@herts.ac.uk

MOLECULAR BIOLOGY UPDATE
A four-day laboratory course
17–20 April 2000
Details and application forms from Prof. John Walker – tel: (01707); 284546 fax: 284510; e-mail: J.M.Walker@herts.ac.uk

INTRODUCTION TO DNA BIOINFORMATICS
A one-day practical computer course
4 July 2000
Details and application forms from Dr Henry Brzeski – tel: (01707) 284554; fax:286137; e-mail:H.Brzeski@herts.ac.uk

INTRODUCTION TO PROTEIN BIOINFORMATICS
A one-day practical computer course
5 July 2000
Details and application forms from Dr Henry Brzeski – tel: (01707) 284554; fax:286137; e-mail:H.Brzeski@herts.ac.uk

RNA EXTRACTION AND ANALYSIS
A one-day laboratory/lecture course
6 July 2000
Details and application forms from Dr Ralph Rapley – tel: (01707) 285097; fax: 286137; e-mail:R.Rapley@herts.ac.uk

PCR METHODS AND APPLICATIONS
A one-day laboratory/lecture course
7 July 2000
Details and application forms from Dr Ralph Rapley – tel: (01707) 285097; fax: 286137; e-mail:R.Rapley@herts.ac.uk

TECHNIQUES IN MEDICAL MOLECULAR BIOLOGY
A three-day laboratory/lecture course
12–14 July 2000, Hatfield, Herts UK
Details and application forms from Dr Ralph Rapley – tel: (01707) 285097; fax: 286137; e-mail:R.Rapley@herts.ac.uk

Honor Fell Travel Awards

Honor Fell Travel Awards are made, up to a limit of £250, to provide financial support for young BSCB members to attend meetings. Applications are considered for any meetings relevant to cell biology.

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The following rules usually apply (at the discretion of the Committee):

- Awards are not normally made to applicants aged over 35
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- No applicant will receive more than one award per year or three *in toto.*
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Meeting for which application is made (Title, place, date): .....

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Subsistence: .....

Registration: .....

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Have you submitted any other applications for financial support?: YES NO

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Number of meetings attended last year: .....

Supporting statement by Head of Department:

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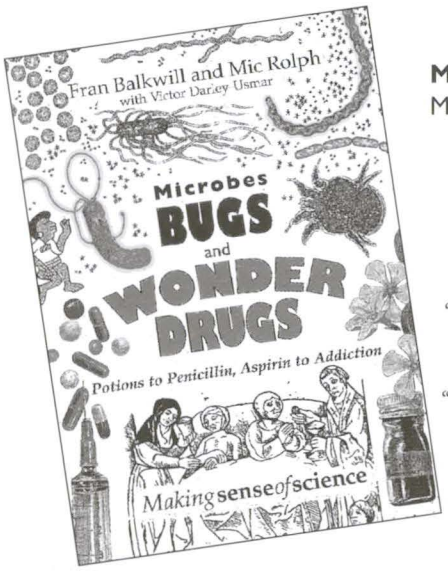

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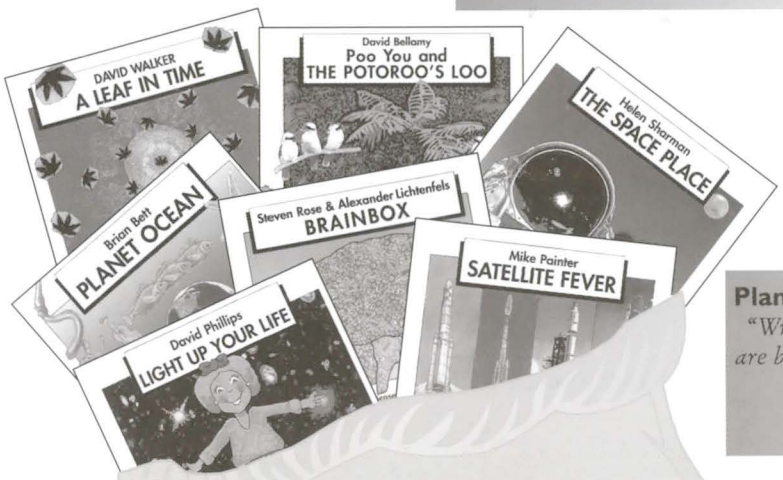

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