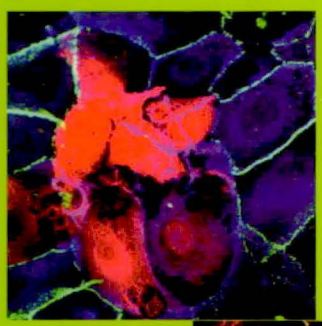
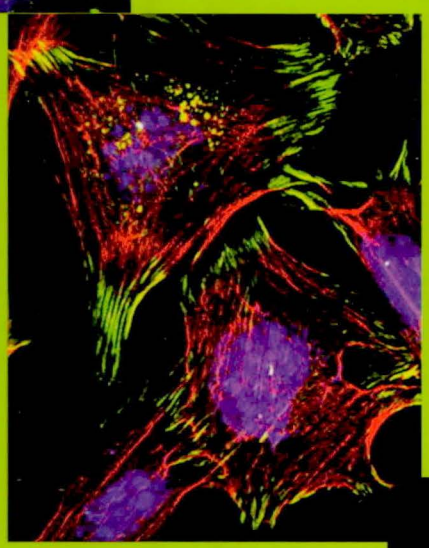
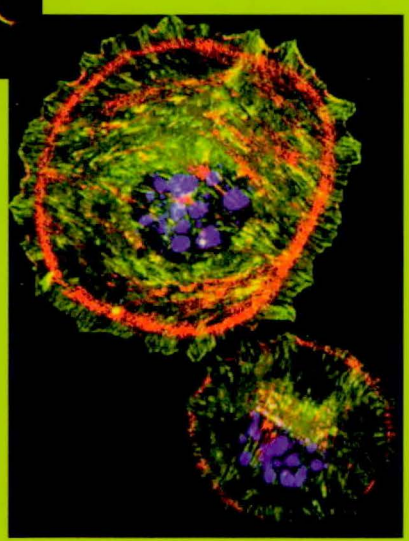
BSCB NEWSLETTER

Winter 1998



Epithelial Cell Biology '98





Summoned by cells to Lake Placid

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.

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Full inside page, black and white only £220
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Four advertisements, to cover two years. The costs are reduced by 30%. We are also happy to enclose flyers with the Newsletter. For a single page, the cost is £165; additional pages are £50.00. For booklets, we negotiate on weight.
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 1223 336655 Fax: +44 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, MSI/WTB Complex, Dept of Biochemistry, University of Dundee, Dundee DD1 5EH.
Tel 01382 345689 (office); fax 01382 322558
E-mail: kayscough@bad.dundee.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 Steve Winder, membership secretary, Institute of Cell and Molecular Biology, University of Edinburgh, Michael Swann Building, Kings Buildings, Mayfield Rd., Edinburgh EH9 3JR.

BSCB members benefit from discounted journal subscription rates. Where prices are given, the full price is listed first, followed by the discounted member price

Current Opinion in Cell Biology	20% discount
Journal of Cell Science	£142/106 (paper **or** online); £163/122 (paper **and** online)
Current Biology	20% discount
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)

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

BSCB Newsletter
Winter 1998

Editorial

Firstly, I'd like to thank all of the contributors to this newsletter for their patience and their prompt replies to frantic emails and for their final submissions; a big help for someone new to the job. Also, many thanks to the sponsors of the BSCB and to the Committee.

As in previous years, this is the time to start thinking about the BSCB Spring Meeting which is to be held at Owens Park, University of Manchester, 13–16 April, 1999. Information about the speakers for this symposium and regarding submission of abstracts, as well as the application forms are all detailed inside. It looks to be a particularly enticing meeting with sessions on motors, cell organization and mitosis.

This past year has also seen some excellent meetings and we have reports here from the BSCB autumn meeting on Epithelial Cell Biology and also from the Lake Placid Meeting on Cell Signalling and the Cytoskeleton. In addition, this newsletter features two articles which outline strategies that have been taken in the UK (Withnall and Raff) and in the USA (Marincola), to alert our respective governments to the importance of the life sciences. Finally, I would like to invite any member to submit any comments or suggestions regarding the 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

Cover pictures. Top left: Distribution of E-cadherin (green) and beta-1 integrin (blue) in human epidermal keratinocytes microinjected with recombinant H-Ras protein (red); courtesy of V. Braga. Centre and bottom right: normal and $FAK^{-/-}$ mouse embryo fibroblasts plated on fibronectin for 90 minutes, then fixed and stained for vinculin (green), actin (red) and DNA (blue); note the accumulation of focal adhesions in the FAK null cells; courtesy of Martin Schwartz.

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Robert Hooke Medal
As indicated in the minutes of the 1998 AGM held in Lancaster (see Spring 1998 newsletter), the BSCB is establishing an annual medal for achievement and excellence in cell biology. Called the Robert Hooke Medal, it is intended that the medal will be awarded to a scientist up to the age of 45 for research contributions to cell biology. The research should have been undertaken in the UK. Nominations for the medal should be sent to the BSCB secretary (Birgit Lane) and should be accompanied by a half page proposing statement, and the CV and a list of relevant publications of the nominee. The closing date for nominations is February 28th 1999 and the winner of the first medal will be announced at the BSCB annual spring meeting 13–16 April 1999 in Manchester.

Science Education
Promoting cell biology through interactions with teachers and children can be fun and rewarding. It is also critical if we wish to widen public awareness and understanding of the importance of this area of science.

The BSCB supports an annual lecture at the Association for Science Education (ASE) meeting, next to be held in January 1999 at the University of Reading. The ASE is the umbrella organization for science teachers in secondary schools. Previous lectures have been on programmed cell death (Martin Raff, 1996), the human genome project (Kay Davies, 1997) and the cell cycle (Paul Nurse, 1998). In addition to the lecture, the BSCB produces an accompanying leaflet which can then be used by schools in their teaching. This leaflet is also reproduced in the spring newsletter and is available on the BSCB website. This year's lecture is to be given by Robin Irvine (Babraham) and is called *'Inositol? No, but he sure knows a hell of a lot'*. So, that promises to be an informative and entertaining lecture about a small and very busy molecule in cells.

If you are interested in presenting your research topic at an ASE meeting, please contact the BSCB secretary (Birgit Lane). Nominations for speakers are also welcome. Please provide name, address and contact details.

Help wanted: BSCB and the WWW
The BSCB Website (http://www.kcl.ac.uk/links/bscb.html) currently carries information about BSCB, its Officers, Membership, and Meetings. Links can then lead the user to a variety of other sites, including those with further information about meetings and other related societies. Back issues of the Newsletter and the leaflet to accompany the BSCB lecture at the annual meeting of the Association for Science and Education are also available.

The Society needs help from members to improve our Website, particularly the sections explaining our subject to those not directly involved in research. We welcome offers of help. Anyone interested in creating a region of linked pages with images and text is encouraged to contact Simon Hughes (simon.hughes@kcl.ac.uk) with suggestions. We would like pages on cell biology for the general public, or for interested school-aged students. Input from schoolteachers would be particularly welcome.

Web editing skills are an advantage, but not essential. What we need are people with ideas of what to present, and how to do it in an exciting and accessible way. One thing that is needed is good quality colour images of cells doing exciting things, so if you have a good slide (without copyright) from a recent talk or lab meeting, just scan it and send it along as a jpeg file, with a brief description of what it is, how it was made and who you are. We will endeavour to get your name in lights (though we do reserve the right to vet submissions).

BSCB Young Cell Biologist of the Year 1998

We would like to congratulate Rita Abranches of the Department of Cell Biology at the John Innes Centre, Norwich who was awarded the BSCB Young Cell Biologist of the Year 1998 for her poster *'Transcription Sites and Chromosome Domains'* which was presented at the BSCB Spring meeting at Lancaster. The prize for this award is a trip to the ASCB meeting in San Francisco in December of this year – we look forward to hearing about her time at the meeting in the next newsletter.

A second prize of a years subscription to Trends in Cell Biology plus £100 was awarded to Janet Cronshaw (Paterson Institute for Cancer Research, Manchester) and the third prize of a years subscription to Current Biology was won by Joanne Shearer (The Medical School, University of Newcastle Upon Tyne).

For more details on how to enter for next year's award see page 27.

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 Manchester 13–16th April 1999.

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, in duplicate, including a brief CV and concise reasons for wishing to attend the meeting should be sent to: *Birgit Lane, CRC Cell Structure Group, Dept. of Anatomy and Physiology, MSI/WTB Complex, University of Dundee, Dundee, DD1 5EH.* Applications should be received by February 28th, 1999.

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.

Young Cell Biologist of the Year Poster Prize 1999

All research students are invited to enter the next poster competition at our Spring 1999 meeting to be held at Owens Park, Manchester, 13–16 April 1999.

The first prize is a trip to the USA to attend the ASCB meeting to be held in Washington D.C. December 11–15, 1999, as a guest of the American Society for Cell Biology, with an opportunity to present the winning poster. For an application form to enter your poster at this years's spring meeting, see page 27.

Honor Fell Travel Awards

Honor Fell Travel 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.

Thomas Kreis (1952–98)

The international cell biology community is just coming to terms with the loss of one of its leading figures. Thomas Kreis had made major contributions both to the membrane traffic and microtubule motor fields, and was one of the few people to recognise early on the intimate links between the two. Indeed, he was one of the organisers of an ASCB/EMBO meeting (to be held in June next year) specifically designed to bring people working in these two fields together. As well as organising many other specialised meetings, he also played a key role in establishing the European Life Scientist Organization (ELSO), which will run European meetings on the scale of the annual ASCB congress.

Thomas Kreis did his Ph.D. in Zurich and then went on to do post-doctoral work at the Weizmann Institute in Israel and at MIT in the USA before being offered a post as a Group Leader at EMBL, Heidelberg. From there, he took up a position in the University of Geneva, where he had just been appointed chairman of the cell biology department.

Early on in his career, Thomas was one of the first to use fluorescence microscopy for studying the cytoskeleton and organelles in both living and fixed cells. These techniques played a key role in his recent work with green fluorescent protein-vesicular stomatitis virus G protein chimeras to visualise membrane traffic between the ER and the Golgi apparatus. This study, at one stroke, pulled together many years of work on fixed cells by both light and electron microscopy. He and his co-workers also identified and characterised a number of key molecules, including a family of proteins – CLIPs – which are involved in linking organelles to microtubules, and beta-COP, a component of the coatomer complex of proteins which are vital for membrane traffic between the ER and the Golgi apparatus.

I spent three years as a post-doc in Thomas' lab. at EMBL. An inspiring teacher and role model, he was

Thomas Kreis (1952–98)

also great fun to work for. He regularly invited the lab round for dinner, where he demonstrated his cooking skills. He was adamant that any decent scientist ought to be a good cook, and needless to say, he excelled at both. Thomas will also be remembered as an expert skier (although as a beginner, I have to say that I never once saw him on the slopes – he was probably moving too quickly) and he even found the time to grow an extraordinary collection of cacti.

Thomas Kreis was killed when Swissair flight 111 crashed on 3rd September 1998 off the Nova Scotia coast, claiming 229 lives. He leaves a wife, son and step-daughter. He will be sorely missed.

Viki Allan
University of Manchester

The UK Life Sciences Committee

Mike Withnall and Martin Raff

The UK Life Sciences Committee (UKLSC) was formed in 1997 by 12 learned societies, including the BSCB (see below). It represents about 30,000 UK scientists working in molecular, cell, and physiological life sciences. By coming together in this way, the member societies hope to be able to speak with a unified voice to Government and other policy makers concerning these areas of the life sciences, which have become so important in today's world. The Committee co-ordinates the activities of its member societies in areas such as science policy and funding, career development, education at all levels, and communication with the public.

It is funded by a subscription fee that is levied on each society according to the number of its members, giving the UKLSC a budget of about £12,000 a year. It has an Executive that consists of a chairman (MR, BSCB), a secretary (Robert Freedman, Biochemical Society), and a treasurer (Pam Riches, British Society of Immunology). The subscription supports a committee secretary (MW, Biochemistry Society), who conducts research and drafts policy papers and responses to consultations from Government and other bodies. The Executive meets four times a year to consider policy, and the full Committee, which consists of representatives from each society, meets twice a year.

The UK Life Sciences Committee

Physiological Society
British Society for Immunology
British Biophysical Society
British Society for Cell Biology
Society for Endocrinology
British Pharmacological Society
Biochemical Society
British Electrophoresis Society
Anatomical Society
Genetical Society
British Society for Developmental Biology
Nutrition Society
Society for Experimental Biology (joined in 1998)

The UKLSC has been functioning for just over a year and, in some respects, is still feeling its way as to how to be most effective. How can it best collate the opinions of its member societies on various policy issues, for example, and how can it best co-ordinate its efforts with those of the Institute of Biology? Nevertheless, it has already been active in various areas. It submitted views to the Commons Science and Technology Committee on research aspects of the Dearing Report and on Government use of scientific advice. It responded to consultations by HEFCE on the future of the Research Assessment Exercise and by the Office of Science and Technology on the next round of Foresight. It has worked closely with the Higher Education Quality Council on its Graduate Standards programme and played an important part in formulating the response of the biosciences community as a whole to the recent Quality Assurance Agency consultation. It has provided information to the four MPs who have a biosciences background, two of whom are members of the Commons Science and Technology Committee. It is currently conducting a Forward Look among its member societies to identify important emerging areas of research; this information will help guide its future interactions with funding agencies and its collaborations with other professional societies.

The UKLSC helps co-ordinate activities of its member societies. It organises the Intersociety Liaison Group of meetings secretaries, which meets twice a year to co-ordinate the planning of scientific meetings. It is assembling a Meetings Database and a Directory of Life Scientists, which can be accessed on the internet (http://www.lifesci.org.uk). It helps its

member societies collaborate in the provision of educational material for schools and in the running of annual careers conferences to university students.

The UKLSC's most innovative (and expensive) endeavour so far has been to run a highly successful symposium on Postgraduate Training in the Life Sciences, which drew more that a hundred delegates from OST, Research Councils, QAA, pharmaceutical industry, research institutes, and universities. There was a surprising degree of agreement on some of the changes that are needed, such as a four year PhD programme with laboratory rotations in the first year and an increased stipend, even if it means fewer PhD students. The UKLSC is committed to making this meeting the first step in rationalising postgraduate education in the life sciences in the UK, which is currently something of a dog's breakfast.

The UKLSC is forging links with other organisations in order to collaborate on matters of common interest. It has worked with the Institute of Biology on quality assurance matters, and it is in close contact with the UK National Committee for Microbiology, which has similar aims to UKLSC. It is working with the Royal Society of Chemistry to try to bring chemists and biomedical scientists into closer contact, through joint symposia and postgraduate training schemes.

Advances in the areas of life science covered by the UKLSC are occurring faster than in any other area of science, and they are likely to have an increasingly large impact on people's lives. The challenge for the UKLSC is to help ensure that the UK is both maximally effective in contributing to these advances and optimally placed to benefit from them.

Biomedical research advocacy: We're in this together

Elizabeth Marincola

The US National Institutes of Health has enjoyed the strong support of Congress over the past several years, generosity which is directly attributable for the acceleration of biomedical research in that period of time. Surely the US effort is a work in progress; there is still much to be done and one should be cautious not to assume that a few good years means that one can relax. Indeed, advocacy is like a marriage: it needs constant nurturing, in good times and bad, to survive and thrive. But as research has become increasingly global and interactive, researchers in other countries often look to the US and wonder how they can replicate support enjoyed by the US biomedical research enterprise in their own countries. To that end, the US experience may be helpful, so I was asked to provide this perspective on basic biomedical research advocacy and to suggest how scientists in other countries may be able to influence their respective governments to increase support for biomedical research.

1. KEEP IT SIMPLE AND BE CLEAR ABOUT WHAT YOU WANT AND WHY

It is essential to be clear in your own mind about what you're working for and why: biomedical research fuels medical discovery, which advances health for all people. Improved health and disease prevention both reduce suffering, and, in the long run, reduce economic costs to society. Basic, untargeted research is the beginning of a process that produces insights, often unexpected, that can be applied to clinical care.

The role of government is critical because it is often not cost effective for pharmaceutical or biotechnology companies to invest in prolonged or high risk basic research: market pressure often requires that they concentrate on applied and clinical research. Sustained support is essential because personnel and

physical infrastructure cannot be efficiently ramped up and down each year if funding fluctuates significantly.

2. DISTINGUISH YOUR INTERESTS FROM THOSE OF YOUR FRIENDS

In the late 1980s and the early 1990s, the US suffered a precipitous decline in the number of grants being funded by the NIH. An analysis of the political infrastructure revealed that while there were established organizations advocating for disease-related research (e.g. the American Cancer Society, the American Heart Association, the Juvenile Diabetes Foundation) and also established organizations advocating for the interests of medical schools and universities, there was no significant advocacy on behalf of the basic biomedical scientist working at the research bench.

This distinction was not critical (indeed it was probably not even noticed) during the era when NIH funding was prospering. But when R01s (individual investigator-initiated research grants) started to decline, it became clear that disease advocacy groups were channeling money into directed research, and institutions were channeling money into indirect (overhead) cost pools which subsidized other activities: nobody was protecting the needs of the individual investigator performing basic research. The disciplinary societies existed of course, individually and in coalitions, but their traditional concerns were mostly limited to publishing journals and organizing scientific meetings. Advocacy for federal funding of basic biomedical research was largely left to the health and institutional interest groups whose funding priorities were not always equivalent to those of the bench scientist.

3. DON'T BE AFRAID TO GET DIRTY

Faced with this dilemma, the leadership of the American Society for Cell Biology determined to organize the basic science societies to advocate for federal funding of basic biomedical research by banding together to hire a full time representative on Capitol Hill to educate Members of Congress about the importance of untargeted (that is, not directed toward a particular disease) basic bench research. This may sound uncontroversial today, but at the time it was considered by many to be undignified for researchers (nay, professors) to amble through the halls of Congress 'selling' basic research to legislators. But while biomedical researchers may rightfully claim a higher moral plane than other interest groups, scientists were wise to learn that in Washington all legislators, regardless of their virtue and sympathy, are subject to the interests of their constituents, as measured by what the constituents tell them, and how they vote.

4. RECRUIT YOUR SCIENTIFIC LEADERS TO BE YOUR ADVOCATES

To overcome the concern that it's unrespectable to assertively encourage biomedical research funding, a core group of scientific leaders agreed to tackle the challenge. This was critical to the success of the movement for at least two reasons: first, it may have required the most respected scientist leaders to procure the moral and financial support of the scientific societies, and second, to convince the larger scientific community that biomedical research advocacy was important, worthwhile... and not undignified. The value of the participation of respected scientists is similar to the value of an eminent corporate Board of Directors: the more respected, the more confidence it inspires and the more investment it attracts. In science, perhaps even more than in general, much faith, for good or ill, is invested in those who are already respected in the scientific 'marketplace.'

5. BE INCLUSIVE

This reasoning may suggest that the eminence of those involved is also critical in actual interactions with Members of Congress. This is true in certain contexts, but not in others. A 'famous' scientist, one, for example, who has won the Nobel Prize, may carry more cache in publishing an opinion piece in the New York Times or in testifying to a Congressional committee. But when it comes to person-to-person interaction between a scientist and a Congressperson, a less-distinguished constituent scientist is often at least as effective in getting the attention of a Member of Congress than is an eminent scientist who is not a constituent.

Important, also, is that any organization that does not

encourage and nourish its young is bound to fail. Involve students, post-docs and young investigators in the effort. A letter from an Assistant Professor is barely distinguished by Congressional staff from one from a Full Professor at the same university. Including young people also has the obvious advantage of serving as a breeding ground for the next generation of scientific leaders who will take up the cause.

6. SEEK PROFESSIONAL HELP

As well intentioned and committed as scientific leaders may be, they are very unlikely to be able to devote sufficient time to the effort to launch and successfully maintain it. Scientific leadership should act as an oversight body, but day-to-day management must be delegated to a person or persons who can be held accountable for following through on activities. This may not necessarily mean hiring a new person for the effort. What talent already exists within the staff of disciplinary or national scientific societies? You may wish to designate someone who is already working in biomedical research to take on the responsibilities of the advocacy effort. Make sure that that person's primary organization will value and reward their advocacy efforts.

7. INFORMATION EQUALS POWER

The most basic first step is to learn legislatively how federal funding is appropriated in your own country. Which agencies fund research? What government entity is responsible for appropriations to those agencies? Who are the 'players' in the process? What are their political interests? Do they have personal health concerns? Following the workings of government can be at times as riveting as watching a test pattern on the television, but mastering the basic mechanism is critical to understanding where influence can be most effectively applied. A paid consultant may be necessary to both educate scientists and to keep them informed and advised as events unfold, which can be daily when the legislature is in session.

8. THE DEVIL IS IN THE DETAILS

You may be successful in recruiting a roomful of illustrious scientists to promote your case, but at the end of the day, they will disperse and return to their labs. The success of an advocacy program depends on the involvement of staff that is willing to ensure that what everyone agrees to do is in fact then done. This will probably require that all the work that can be performed by staff is performed by staff. For example, twenty prominent scientists may agree to write to their legislators and promptly go home and not get around to it. There must be staff attention to ensure actual implementation, which may mean the drafting of letters for the review, approval and signature of scientists. Horses may not be forced to drink, but the chances are increased if they are led right up to the shore.

9. KEEP YOUR EYE ON THE BALL

As with any start-up venture, it is tempting for well-intentioned people with a mission to want to embark on several paths simultaneously, but this approach risks failure at all of them. Establish attainable initial goals and make sure you reach them. For example, you may wish to develop a target list of legislators to receive a letter in support of biomedical research from at least one scientist by a date certain. This would be a respectable and realistic first step.

10. NO POINTS FOR ORIGINALITY

Influencing the legislative process can be dishearteningly uncreative. There is a limited bag of tricks and most advocacy organizations freely imitate others' tactics. Legislators are influenced by their constituents' opinions, by the real or perceived benefit of an activity to their district or constituency, by the cost-effectiveness of an investment, by public opinion and by (believe it or not) their own sense of the right thing to do. Demonstrate why investing in biomedical research is worthwhile by documenting the money that it brings into a district. Give an example of how basic research has led (serendipitously or by design) to preventing or treating a disease. Cite the health care savings that result from scientific discovery.

Influence public sentiment by submitting opinion pieces to national or local newspapers about the value of investing in biomedical research. Document the number of people (voters) in a district who are directly and indirectly effected by biomedical research funding.

11. ENCOURAGE LEGISLATORS TO BECOME ADVOCATES THEMSELVES

One very successful effort in the U.S. has been the formation of the Congressional Biomedical Research Caucus in 1990. A core group of sympathetic legislators was encouraged by scientists to host a series of briefings on research topics of public interest for their fellow legislators and their staffs. The researchers provide guidance on speakers and topics, but the Caucus itself (which, importantly, is non-partisan, bi-cameral and does not ask that members pay dues) is sponsored by Members of Congress themselves. Having publicly associated themselves with biomedical research by their leadership of or membership in the Caucus, they become motivated to encourage others to join the movement. More important, once this critical mass of sympathetic legislators is identified, it becomes a resource for sponsoring or endorsing bills that are supportive of biomedical research. In the U.S. over 150 Members of Congress are members of the Congressional Biomedical Research Caucus, and the briefings it sponsors on Capitol Hill routinely attract over a hundred Congressional staffers and several Representatives.

12. ALL POLITICS ARE LOCAL

... according to the late, great Speaker of the House of Representatives Thomas P. 'Tip' O'Neill. If a dialog can be established between a legislator and a scientist-constituent, this is an important step. The legislator may not blindly defend biomedical research funding, but s/he will at least have someone to turn to for advice about scientific issues should the need arise down the road. Cumulatively these relationships can have great effect, whether or not a particular politician is in a direct position to influence biomedical research funding.

A legislator who is on the Transportation Committee may be reassigned to a key Appropriations Committee; a Member of the House may be elected to the Senate; one Member may encourage another on a relevant committee. In politics as in life, it pays off in unanticipated ways to selectively cultivate friendships. Recently a delegation of North Carolina scientists visited their Members of Congress in Washington, including one Congresswoman representing a rural part of the state with no significant biomedical research activity in her district. She was visibly delighted (even honored) by the visit, noting that no one had ever come to talk to her about biomedical research before.

13. SAY THANK YOU AND KEEP GOING BACK FOR MORE

Legislators, like all of us, appreciate being recognized for their efforts, publicly and privately. If a legislator takes a stand in favor of biomedical research, thank him or her directly by letter and ask others to do the same. In exceptional circumstances more dramatic gestures may be appropriate, such as a scientist hosting a private fundraising event for champions of biomedical research funding (these are usually receptions in homes where everyone attending gives a modest ($50–$100) donation to the legislator for his or her next re-election campaign) or advertise the person's actions in the community (this latter action can backfire if the stand taken is controversial, so be cautious and seek endorsement for any such plans.)

Follow a visit or letter with another one later. Consistency is key to keeping biomedical research on the radar screen of busy legislators. There is also constant turnover among elected politicians. New legislators should be contacted early in their terms.

14. YOU'RE ON THE HIGH ROAD: STAY THERE

In contrast to the gun lobby or the tobacco lobby, biomedical research has the distinct advantage of having many friends and no significant enemies (animal 'rights' activists may oppose certain aspects of biomedical research but most credible movements don't oppose research in general). Avoid opposing other discretionary programs, even if they compete with biomedical research for limited federal funds. Biomedical research promotes health for the benefit of all mankind. Be proud to be an advocate for the support that it deserves.

Elizabeth Marincola, Executive Director of The American Society for Cell Biology, and Executive Director of The Joint Steering Committee for Public Policy. 9650 Rockville Pike, Bethesda, MD 20814-3992. Tel: 301-530-7153; Fax: 301-530-7139; E-mail: emarincola@ascb.org
www.ascb.org/ascb; www.jscpp.org/jscpp

BSCB Autumn Meeting: Epithelial Cell Biology '98

University of Oxford 13–16 September 1998

Neil Hotchin, David Hudson and Vania Braga

Once again the organizers chose St Catherine's College for the Epithelial Cell Biology 98 meeting and must be applauded for their choice. Although the college has little history to offer it does have comfortable rooms, good food, wide open green spaces and lots of room in the bar for late night socialising. The organisers – Charles Streuli (Manchester) and Paul Edwards (Cambridge) – assembled a truly excellent cast of speakers addressing questions as diverse as patterning in Drosophila *to mechanisms of toxicity in the human gut. In a meeting report such as this, justice cannot be done to all speakers and this report, by necessity, is both brief and selective. We apologise to those whose talks are not covered – omission in no way reflects content, but rather the particular bias of the meeting report authors.*

Growth factors and epithelial cell fate

The meeting started with an elegant illustration of the power of genetics from **Matthew Freeman** (Cambridge), in which he described how signalling through the *Drosophila* EGF receptor regulates cell patterning in the follicular epithelium of the fly egg. In the 'Twin Peaks' model he demonstrated a complex interplay between the receptor, its TGFα-like ligands *gurken* and *spitz*, and the secreted inhibitor *argos*. This leads to the splitting of an initial single peak of MAP kinase activation into two, thereby specifying the correct positions of two anterior dorsal appendages.

The roles of KGF-1 (FGF-7) and KGF-2 (FGF-10) in skin development and wound repair were discussed by **Sabine Werner** (Martinsreid). Use of differential display resulted in the identification of a diverse set of KGF-regulated genes. These included activin, which the same laboratory had previously observed was expressed at high levels during wound healing. Normal skin morphology is perturbed in transgenic mice expressing activin linked to the keratin 14 promoter with hyper-thickening of the dermis and epidermis. This may be a consequence of the increased rates of proliferation and impaired differentiation observed in these mice.

The ICRF Lecture introduced a tissue new to many at the meeting as an experimental model – the prostate. Using an *in vivo* tumour assay, **Gerry Cunha** (San Francisco) described how transformed epithelial cells are more likely to form tumours when cultured with stromal cells from prostate tumours compared to when cultured with cells from normal tissue. This control of cell proliferation by epithelial-mesenchymal interactions illustrates the complex nature of tumour progression *in vivo*.

Epithelial morphogenesis

In another elegant study **Helen Skaer** (Sheffield) showed that cell proliferation in the developing excretory system (Malpighian tubules) of *Drosophila* is driven by activation of the EGF receptor by a growth factor produced by a specific cell, the tip cell, unique to each Malpighian tubule. Tip cells do not themselves proliferate, but instead direct proliferation in neighbouring cells of the tubule. Removal of these tip cells results in the tubules failing to develop. Tip cell specification is complex and results from lateral inhibition mediated by the neurogenic genes *Notch* and *Delta*, patterning by *Wingless* and the asymmetric activity of *numb*. Together these act to maintain tip cell expression of the proneural gene, *achaete*.

The *Drosophila* theme continued with the Wellcome Trust Lecture. **Mark Krasnow** (Stanford) described the role of FGFs and FGFr in development of the tracheal system. The tracheal system which arises from two sets of 10 sacs, each of which contain 80 cells. Formation of the tracheal system involves

co-ordinated, but distinct cellular events. Formation of primary and secondary branches during embryogenesis involves epithelial cell migration from the tracheal sacs. The formation of fine terminal branches, occurs during larval development, is triggered by hypoxia and involves cytoplasmic extension. Paradoxically, these two distinct cellular events are regulated by interaction of the same secreted FGF (*branchless*) with its receptor (*breathless*). This raises the obvious question of how interaction of *branchless* with *breathless* control two distinct tracheal responses. One hypothesis put forward is that the first time a tracheal cell is exposed to branchless a migratory response is triggered along with the transcriptional induction of various genes. One of the induced genes (the *pruned* Serum Response Factor) is a downstream transcription factor in the FGF signalling pathway and is required for cytoplasmic extension. On subsequent exposure to FGF, the SRF transcription complex is activated and the tracheal cells grow by cytoplasmic extension rather than epithelial cell migration.

Signalling through ECM and Rho proteins

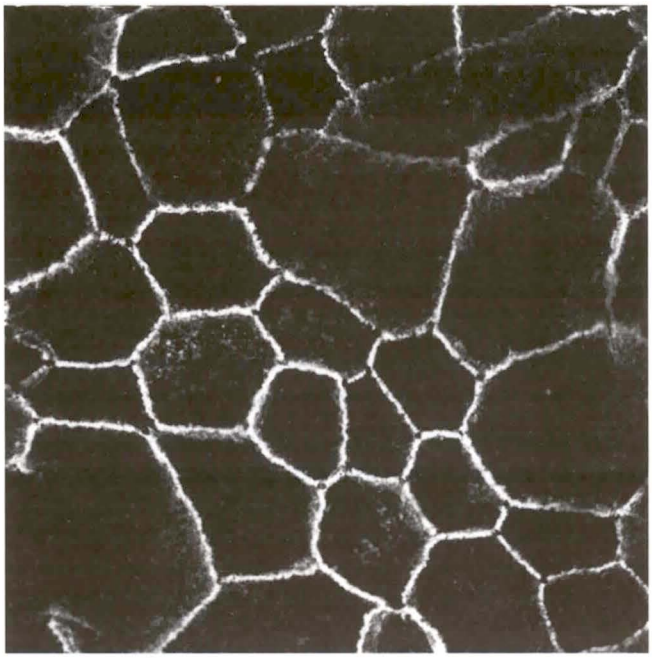
In recent years much interest has centred on analysing how integrins, via their interaction with extracellular matrix, interact with intracellular signal transduction pathways and **Filippo Giancotti** (New York) presented exciting new data on the role of caveolin-1 in integrin-dependent cell cycle progression. Previous work had identified caveolin-1 as a potential link between integrins and the Ras-ERK pathway. Here, Giancotti presented evidence for caveolin-1 acting as an adapter, providing a physical and functional link between integrins and the tyrosine kinase Fyn. Ligation of integrins causes activation of Fyn, sequential recruitment of Shc and Grb2/mSOS, and activation of the Ras-ERK pathway.

The theme of adhesion-dependent signal transduction was continued by **Charles Streuli** (Manchester) in his talk on how signals from classical growth factor receptors synergise with those from integrins to regulate cell function in primary mouse mammary epithelial cells. In the absence of insulin these cells undergo apoptosis. Interestingly, this survival signal mediated via PI-3 kinase, requires that cells be plated on basement membrane matrix components. In the absence of contact with basement membrane cells die despite activation of the insulin receptor by insulin. The key event appears to involve IRS-1, in that IRS-1 is not phosphorylated in the absence of contact with basement membrane. This suggests that association of IRS-1 with PI-3 kinase, and subsequent downstream signalling, requires both insulin and interaction with basement membrane.

It is now well established that the Rho family of small GTPases play key roles in assembly of cell adhesion structures, reorganisation of the actin cytoskeleton, as well as regulating a number of intracellular signal transduction pathways. Further emphasising the concept of synergy between adhesion and growth factor signalling, **Neil Hotchin** (Birmingham) presented data from Swiss 3T3 fibroblasts, which indicates that cellular responses to activation of Rho GTPases by growth factors are regulated by interaction with specific domains within extracellular matrix molecules.

Continuing with Rho family GTPases, **Vania Braga** (London) presented data on the disassembly of cadherin-based adherens junctions in human epidermal keratinocytes following microinjection of oncogenic Ras (H-Ras). It has been previously shown that activation of the small GTPase Rac is required for cadherin function; however, here data was presented which suggests that Rac may play a role in Ras-mediated E-cadherin destabilization at cell-cell contacts. This apparent contradiction may be explained by the observation that high levels of Rac or sustained activation of Rac are not compatible with stable cell-cell contacts.

Paul Martin (London) described his studies of wound healing in embryos. In the embryo, re-epithelialisation of the wound is accomplished by contraction of an actinomyosin purse-string, rather than by lamellipodial crawling of cells as occurs at



Normal human epidermal keratinocytes stained with an antibody against E-cadherin receptors.

the adult wound edge. Assembly of this embryonic wound cable is dependent on activation of the small GTPase Rho. By contrast, closure of adult wounds involves cell crawling, an activity not blocked by inhibitors of Rho suggesting that this process may be regulated by other members of the Rho family.

Catenins, APC and wnts

Genetic analysis has clearly established that signal transduction mediated by *Wingless* in *Drosophila* and its vertebrate homologue (Wnt) control many developmental processes including axis formation in *Xenopus*. Upon appropriate signals from Wnt/*Wingless* receptors, β-catenin (vertebrates) and its *Drosophila* homologue *Armadillo* relocate from intercellular junctions and accumulate in the cytoplasm. This event is followed by translocation to the nucleus, where *Armadillo*/β-catenin binds to the TCF/LEF-1 family of transcriptional factors, relieves suppression of TCF resulting in transcription of specific genes. Two talks in this session centred around the discovery of repressors for the Wnt/*Wingless* pathways.

Mariann Bienz (Cambridge) identified CBP/p300 (CREB binding protein) as a repressor for *Armadillo* signalling in *Drosophila*. CBP does not bind directly to *Armadillo*, but interacts with TCF. The proposed mechanism of repression involves the acetylation of TCF by CBP, therefore preventing the association of TCF with *Armadillo*. Consistent with this role, flies with reduced CBP levels show a similar phenotype to those with an activated *Wingless* pathway.

Another repressor of the *Armadillo*/β-catenin signalling pathways in mammalian cells, TIS-7, was described by **Lukas Huber** (Vienna). TIS-7 (TPA-induced sequence-7) was identified using RT-PCR differential display to identify genes involved in epithelial-mesenchymal transition in mouse mammary epithelial cells and expression of TIS-7 is associated with a loss in polarity. TIS-7 is a 47 kDa peripheral membrane protein with a nuclear localisation signal and several putative phosphorylation sites. In polarised, confluent epithelial cells TIS-7 is found at adherens junctions and in both polarised and unpolarized cells can be co-immunoprecipitated with β-catenin. Interestingly, high levels of TIS-7 expression prevents β-catenin nuclear localisation and subsequent association with TCF transcription factor, suggesting that TIS-7 is a repressor of β-catenin-induced transcription. In addition, microinjection of TIS-7 into *Xenopus* embryos perturbs axis formation, suggesting that it can interfere in the Wnt pathway.

Epithelial proliferation and apoptosis

Martin Raff (London) addressed the role of caspases, which mediate programmed cell death (apoptosis), in three separate models of epithelial development. In the first, neural tube formation, closure of the epithelial sheet involves extensive cell death at the fusing edges. Treatment with caspase inhibitors, inhibits this cell death and prevents fusion of the epithelial sheets and pinching off of the neural tube. In the second model, lens development, epithelial cells lose their organelles and fill up with crystallins, a process which is blocked by caspase inhibitors. In the final model, terminal differentiation of human epidermal keratinocytes, the cells lose their nuclei

and fill up with keratin fibres. Caspase 3 is activated in the region of nuclear loss and this loss is blocked by treatment with caspase inhibitors. Thus in both lens and skin, organelle loss seems to depend on caspases.

An interesting talk from **John Hickman** (Manchester) made the important observation that drug-induced gut toxicity is not necessarily a direct consequence of apoptosis, but rather it results from both apoptosis and inhibition of proliferation. This clearly has important implications for treating the side-effects of chemotherapy.

In the AICR lecture, **Bill Muller** (Ontario) described work from his laboratory on the role of ErbB2 in mammary tumorigenesis. Following his earlier work expressing an activated mutant of ErbB2 in mammary gland, in transgenic mice , he had expressed wild-type ErbB2 to see whether this would give rise to mammary tumours. The transgenic mice bearing the wild-type gene did indeed develop mammary tumours, but examination of the tumours showed that they nearly all expressed a mutated ErbB2. Each tumour had a different in-frame deletion frequently resulting in loss of a cysteine. This loss of a cysteine resulted in an odd number of extracellular cysteines suggesting that the free cysteine might be involved in stabilising receptor dimers. In elucidating the role of ErbB2, knockout mice have not been very informative, as the mice die before mammary gland development. In an attempt to get round this problem Bill Muller used the Cre/LoxP system to knockout ErbB2 in a tissue-specific manner. Unfortunately (or interestingly depending on your viewpoint), no ErbB2 knockout cells were detected, suggesting that ErbB2-negative cells are selected out in mammary epithelium.

The meeting ended as it had begun – with *Drosophila*. The Zeneca Lecture, delivered by **Peter Bryant** (Irvine), centred on the mechanisms which control cell proliferation within the *Drosophila* imaginal disc. He described the isolation of four novel, highly related, growth factors – imaginal disc growth factor (IDGF) 1–4. IDGFs on their own were unable to stimulate DNA synthesis in cultured imaginal disc cells, but were able to synergise with insulin. None have obvious homology to known growth factors, but interestingly share some homology with chitinases and may act as lectins.

Overall the meeting brought researchers from diverse fields together and provided a refreshing novelty of ideas. Most importantly, it showed that by using a combination of techniques from cell and developmental biology major problems in the control of normal cell function are gradually being solved. Epithelial are arguably the most important cells in the body and the study of their behaviour is most certainly alive and kicking.

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Summoned by Cells: Cell Signaling and the Cytoskeleton

Adirondack Biomedical Research Institute Symposium, September 24–27 1998, Lake Placid NY.

Gareth Jones

This meeting is the latest in a profusion of conferences that aim to keep us up to date with the rapid advances in the study of cell adhesion and motility. An outsider to the field might wonder why we need quite so many, but new results continue to flood into the journals, and only the few can hope to keep abreast of every idea as it is ceaselessly refined in the literature. Meetings are a quick fix for the likes of me, snowed under by teaching and administration in addition to keeping a lab going.

This meeting was divided into five sessions on actin skeleton, cell- substratum adhesion, cell–cell contact, motility, and a session of mixed talks on intracellular targeting and multimolecular organisation. Given the restraints of space, I will restrict my report largely to those talks that I found most interesting. In places I will also offer a personal interpretation of a speaker's findings: please don't take these as gospel.

Session 1: Actin Cytoskeleton

Tom Pollard described recent work from his laboratory that might alter the current general view of the regulation of actin filament assembly and disassembly at the leading edge of cells. Physiological concentrations of just five components (actin, profilin, an ADF/cofilin protein, capping protein and the ARP2/3 complex) can explain many features of actin filament turnover. With enough capping protein available to cap every filament in a cell at both ends, the ARP2/3 complex plays a major role in stimulating new barbed end growth while also inhibiting pointed end elongation. The ARP2/3 complex can also anchor new filaments to the sides of old filaments at an angle of approximately 70° via the pointed end, thus leaving the barbed end available for further elongation in the direction of the plasma membrane. Interestingly, just such a pattern of branched actin filaments has been observed by the Borisy laboratory in motile fish keratocytes.

A more contentious consequence of this new model of actin dynamics, termed 'dendritic nucleation' by Pollard, is that there appears to be no need to assume that actin filaments should be attached to the plasma membrane of the leading edge. **Alan Hall** used a standard *in vitro* wound-healing assay (Figure 1) to show that wound closure is abrogated by micro-injection of dominant-negative Rac into rat embryo fibroblasts. Closure is reduced by some 50% following

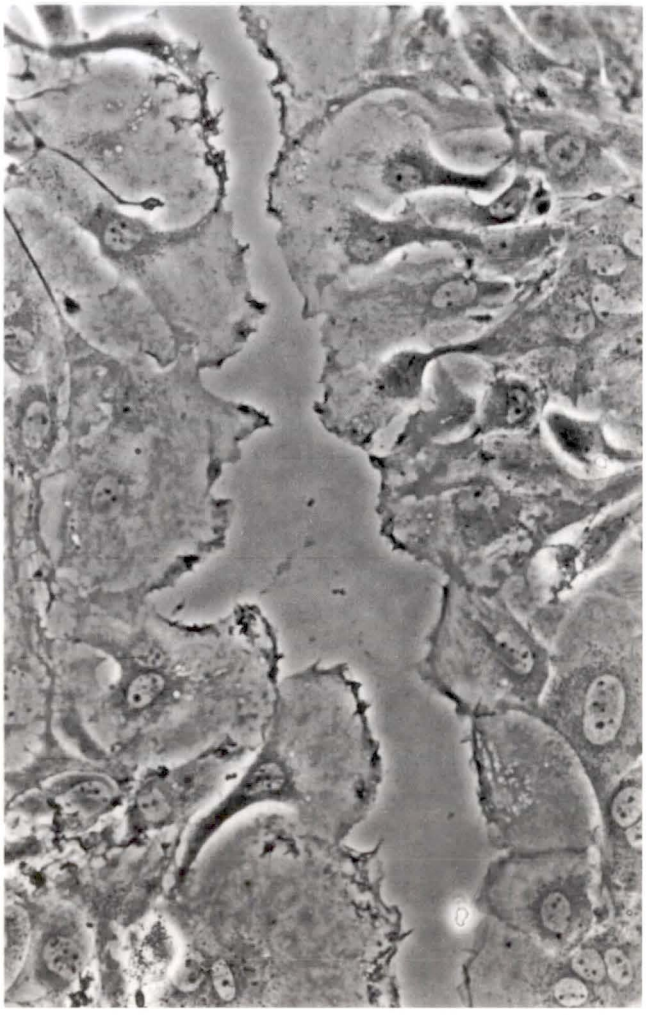

Figure 1. Rat embryo fibroblast cells crawling into a wound made by scratching a confluent monolayer. Courtesy of Catherine Nobes and Alan Hall.

injection of dominant-negative Cdc42, and largely unaffected by inhibition of Rho. These data can be interpreted to show that stress fibres (Rho action) are neither required for nor do they inhibit cell locomotion while lamellipodia (Rac action) are obligatory.

In the case of Cdc42 (generation of filopodia), things are a bit more complicated. Cells appear less polarised following injection of dominant-negative Cdc42, so it seems that filopodia are required to establish and/or maintain a fully polarised morphology. In a separate study on phagocytosis in macrophages, the actin cytoskeletal response was again shown to be under the control of separate members of the Rho family. Complement receptor (CR3) – induced phagocytosis of erythrocytes follows activation of Rho, while immunoglobulin receptors require Cdc42 and Rac activation.

The last speaker in this data-saturated session also described work on the crucial role played by Rho proteins in regulating microfilament structures within cells. **John Hartwig** discussed the signal transduction pathways controlling actin remodelling in blood platelets. In resting platelets, Rac and Cdc42 are in a GDP-bound (inactive) form, but following platelet PAR-1 receptor activation, the GDP is replaced by GTP. Rac now moves to the plasma membrane where it interacts with phosphoinositide kinases. The subsequent production of PIP2 leads to the inactivation and dissociation of capping proteins which decorate the barbed ends of actin filaments. Thus activation of Rac promotes filament uncapping with consequent filament elongation. However, it is now clear that the C-terminal region of Rac has a GTP-insensitive binding site for PI-5 kinase, and mutations of this site inhibits the ability of V12Rac to promote actin filament uncapping. Thus there seems to be a GTP-independent (PI-5K) and a GTP-dependant (PI-3K) pathway for Rac-induced filament uncapping. Cdc42 also promotes actin assembly, via the GTPase Ral1A, which itself binds to the last repeat of the actin crosslinking protein ABP-280. How this leads to filopodia production wasn't made clear (at least to me), since ABP-280 is known to promote orthogonal branching of actin filaments at the cell periphery. Quite a bit more needs to be done to see whether Cdc42 is really involved in platelet filopod formation.

Session 2: Cell–Substratum Adhesion

Keith Burridge discussed Rho and intracellular contractility. Classical tensegrity theories, like those of Ingber, suggest that tension generated by the actomyosin system is opposed by microtubules which are viewed as compression-resistant struts. Burridge takes another view following an observation that in quiescent cells, in which Rho is largely inactive, microtubule depolymerisation stimulated Rho-dependent contractility. He suggested that microtubule depolymerisation is affecting contractility not because of the loss of an antagonistic mechanical strut, but rather because depolymerisation stimulates the activation of Rho.

Signal transduction was more comprehensively reviewed by **Martin Schwartz**, who elegantly showed how signals from both occupied integrins and growth factor receptors could be coordinated, via the MAP1 kinase pathway and Rho. Growth factors induce activation of the MAP kinase ERK2 very poorly in suspended cells compared to adherent cells. This effect of adhesion occurs specifically at the step between Raf1 and MEK1, and is mediated by FAK. Using $FAK^{-/-}$ fibroblasts from a mouse transgenic, it was shown that MAP kinase activation became adhesion-independent. The laboratory has recently developed a novel, and currently the only, assay for Rho activity based on GTP loading, and exploitation of this method enabled the demonstration of a change in Rho activity as cells spread and become polarised. Rho activity is low in the rapid spreading phase, then high as the cells stop spreading and begin to elaborate stress fibres and focal adhesions. Cell adhesion to fibronectin also modulates growth factor activation of Rho.

Finally, it was demonstrated that FAK knockout cells have elevated Rho activity that is no longer regulated by adhesion to fibronectin. This result may account

for the over-abundance of focal adhesions in these cells (see issue cover). As these cells spread, every focal adhesion is retained, with little or no evidence for dissolution or disassembly as would be seen in normal cells.

Julian Downward was called upon to describe the linkage between cell adhesion, apoptosis and Ras signalling pathways. Of particular interest was a discussion on the protective effect of Ras from the apoptosis generated by epithelial cell detachment from an extracellular matrix. The effect of Ras is transduced through PI-3 kinase and Akt/PKB. Detachment-induced apoptosis involves the autocrine activation of Fas and related death receptors, leading to stimulation of caspase 8. Akt/PKB uncouples caspase 8 from death domain-containing receptors, thereby preventing initiation of the death signal. In normal cells matrix adhesion stimulates PI-3 kinase and hence Akt/PKB to provide a suppression of death receptor function.

Session 3: Cell–Cell Contact

James Nelson was unexpectedly unable to attend for family reasons, but **Charles Yeaman**, a senior postdoc. in his laboratory stepped in to give a polished presentation in his place. Mechanisms that specify delivery of vesicles to the appropriate membrane domain and retention of proteins in the membrane are important in the generation of cell surface polarity. Cell–cell adhesion initiates this polarisation by acting as a cue to the generation of structural and functional asymmetry. In budding yeast, the Sec6/8 complex is essential for generating cell polarity by specifying vesicle delivery to the bud tip. Now, Sec6/8 homologues have been found in MDCK cells. These components are in a cytosolic compartment in unpolarised cells, but upon initiation of calcium-dependent cell–cell contact, the complex is rapidly recruited to the sites of cell–cell adhesion. Further work was able to demonstrate that the lateral membrane recruitment of the Sc6/8 complex is a direct consequence of cell-cell adhesion and essential for the biogenesisof epithelial cell surface polarity.

Masatoshi Takeichi told us of two model systems he was exploiting in order to look at the regulation of cadherin function by associated proteins. Of these, the most engaging was a study of the synapse, where he showed that α-catenin and cadherins are important components of the synaptic cleft, lying around the zone in which neurotransmitter release and binding are found. To study the function of cadherin 11 at the synapse, a knockout mouse was generated. The null mutant mice grow to maturity and appear to have a normal phenotype apart from a truncated snout. There is no structural defect in the brain, but when subject to a battery of electrophysiological tests, the LTP mechanism was enhanced! Behavioural tests were then performed to show that the null mice were less anxious and had reduced innate fear characteristics. The precise role of cadherin 11 in LTP is not yet known as no direct improvement of memory could be shown in these mice.

Barry Gumbiner told us of work using a novel antibody, AA5 that activates C-cadherin-mediated adhesion. Using the *Xenopus* animal cap assay, it was shown that the normal activin-induced elongation of embryonic tissue is linked to a weakening of cadherin-mediated adhesion between blastomeres. Addition of mAb AA5 restores strong adhesions between the blastomeres and the animal caps no longer elongate. Since mesoderm-specific gene expression is unaffected, the decreased adhesiveness induced by activin appears to be essential for morphogenesis during gastrulation. Activation of C-cadherin by mAb AA5 seems to result primarily from changes in the organisational state of the cadherin in the plasma membrane. It was shown that oligomerisation and clustering of cadherins is important for full adhesive activity and that clustering is ligand-dependent.

Surprisingly, the classical catenin-binding domain of C-cadherin does not seem to be required for either clustering or basic adhesive activity (at least when expressed in CHO cells). Purification of proteins binding to the juxtamembrane region of cadherin revealed that the major associated protein is p120ctn,

raising the possibility that p120ctn is involved in clustering and thus regulation of cell adhesion.

Session 4: Motility

Daniel Jay described the use of CALI (chromophore assisted laser inactivation) as a tool for temporarily knocking out protein function. In this talk he used the technique to ablate myosin V within the growth cones of dorsal root ganglion cultures. This leads to filopodial collapse, though the lamellipodia remain intact. Closer analysis of the time course of filopodial collapse suggests that it is only the extension phase that is disrupted by destruction of myosin V, the retraction behaviour is maintained. Jay concluded that myosin V is involved in cargo transport of materials to the filopodial tips. In contrast, CALI of myosin Iβ had no effect on filopodial kinetics but there was lamellar expansion in those areas of the growth cone irradiated by the laser. This expansion is sufficient to generate turning into the direction of irradiation if the CALI is repeated 2 or 3 times. These findings support the hypothesis that lamellipodia, and not filopodia, function in the mechanics of motility.

Extending her earlier work on *Listeria* motility, **Julie Theriot** described how she had coupled purified ActA to beads, and then added them to cytoplasmic extracts of *Xenopus* eggs. Her videos of the actin comet tails so generated and the movement of the beads was stunning, as was the subsequent analyses of the kinetics of the phenomenon. With low concentrations of ActA, a diffuse cloud of f-actin distributed around each bead. Higher concentrations lead to the generation of comet tails. At even higher concentrations of ActA, the pattern reverts to clouds. Smaller spheres are more likely to make tails than larger beads, this is due to Brownian motion and the requirement for a bead to move over a critical fraction of its diameter.

Finally we were shown a method of making a more bacterium-shaped cylinder rather than using spherical beads, and how to deposit ActA asymmetrically on the cylinders; more-or-less mimicking the *Listeria* bacterium in basic design.

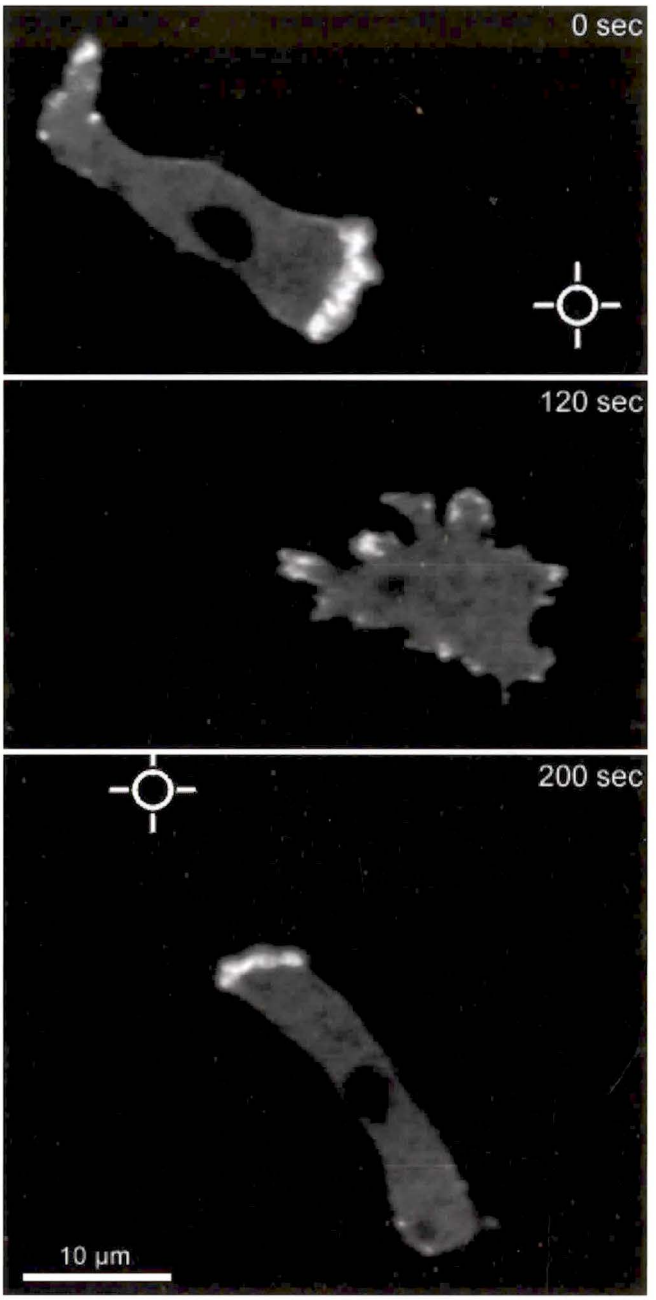

Figure 2. GFP–coronin accumulates at the leading edge of a *Dictyostelium* cell stimulated by the chemoattractant cAMP. The pipette tip that delivers the cAMP is indicated. Note the coronin accumulating at the tip of multiple extensions during re-orientation of the cell (middle panel). Courtesy of Mary Ecke and Günther Gerisch.

The views of comet tails and motility using these objects was a highlight of the meeting.

After this remarkable performance, it was just as well that the session was ended by **Günther Gerisch**

as most other presentations would surely have been an anti-climax. Yet again, a series of video sequences served to highlight the remarkable discoveries recently coming from his laboratory. Three parameters of *Dictyostelium* motility, chemotaxis, phagocytosis and cytokinesis, were shown to be consequential to the re-distribution of proteins from the bulk cytoplasm to cortical sites in the cell.

The talk focused on coronin, a member of the WD-repeat family of proteins, which regulates the disassembly of cortical actin networks. Fusions of coronin with GFP show that this protein assembles dynamically in the actin-rich protrusions of migrating cells (Figure 2) as well as in phagocytic cups induced by the attachment of particles. During cytokinesis, coronin accumulates in association with actin filaments at the two poles of a cell. Analysis of knockout mutants indicate that null cells have problems re-organising actin filaments with drastic effects on migration, phagocytosis and cytokinesis. A great deal was also said about the cortexillins, members of the spectrin superfamily. The 2 cortexillin isoforms of *Dictyostelium* are the only family members whose elimination strongly impairs cytokinesis. Their important role in mitotic cleavage is probably due to their ability to bundle actin filaments in an antiparallel fashion and their translocation to the cleavage furrow during mitosis.

Session 5: Multimolecular organisation

This session was planned for the morning after the conference dinner, during which we were regaled by a demonstration of the occasional ferocity of North American weather. A spectacular storm lasted for many hours, and next morning delegates awoke to find themselves in a hotel (indeed a whole County) with no electricity – even the reserve generators were not functional. After a basic cold breakfast in the gloom, the organisers managed somehow to lay on a 'chalk-talk' session in which four speakers displayed their lecturing skills at a blackboard. Never before have I seen coloured chalks used so imaginatively to mimic those fluorescent confocal images we all use now.

The most interesting of these presentations came from **Shoichiro Tsukita** in his exposition on molecular assemblies in tight junctions (TJ). Recent technical progress has enabled the identification of several TJ-associated peripheral membrane proteins such as ZO-1 and ZO-2. This laboratory also discovered the TJ-specific integral membrane protein occludin some five years ago, and evidence has since accumulated to show that occludin is directly involved in the formation and functions of TJs. To see if occludin is the only integral membrane protein in TJs, occludin-deficient ES cells were generated. Surprisingly, these cells can be differentiated into polarised epithelial cells that bear well-developed TJs.

This discovery led to a search for other integral proteins, and after much effort the laboratory isolated and characterised two proteins, claudin-1 and claudin-2. These related proteins are also 4-membrane spanning moieties, and when expressed in L-cells, can generate TJ-type junctions in areas of cell-cell contact. Many claudin forms have now been discovered (claudins 1–11), with interesting tissue-specific distributions. Much work is currently in progress to elucidate the structural and functional relationships of occludin with the claudins in TJs.

With every prospect of new discoveries to come within the next few months, get ready for another meeting along the same lines coming your way soon.

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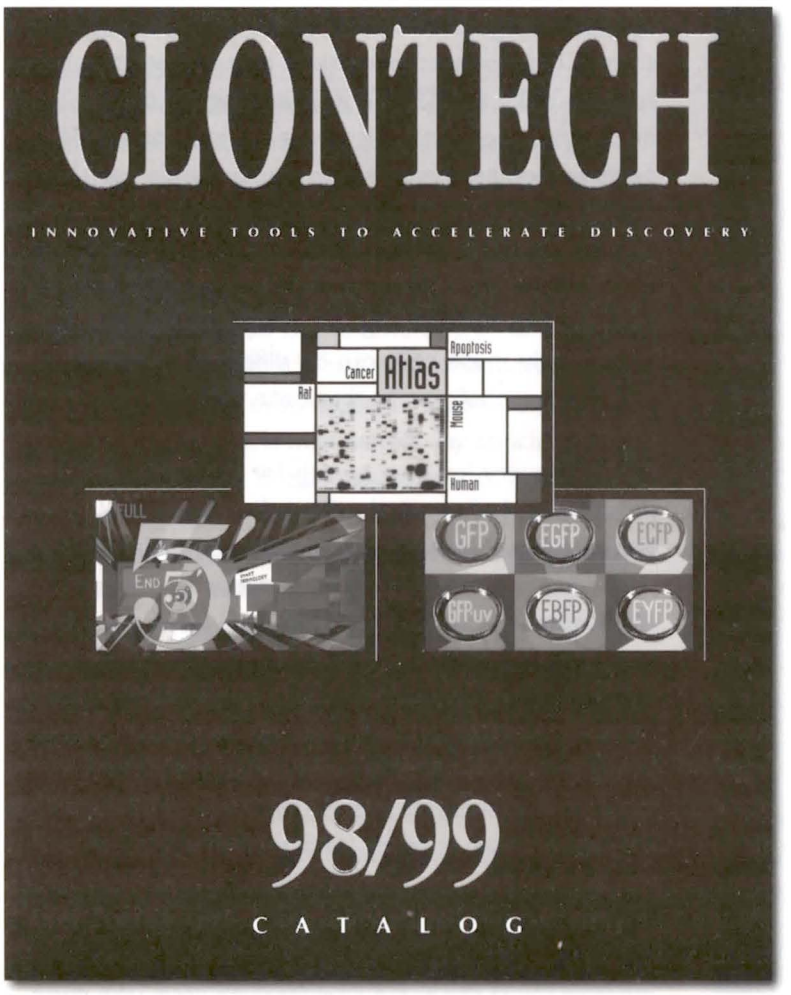

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BSCB/BSDB Joint Spring Meeting

Manchester University, 13–16 April 1999

General Information

Venue

The meeting will held at the University of Manchester Owens Park Campus, approximately 2 miles south of the City Centre. The Plenary lectures and most sessions will be held in the University Armitage Conference Centre. A few sessions and Workshops will also be held in the Owens Park Main Hall, approximately 5 minutes walk from the Armitage Centre. Tea and Coffee will be held at each centre. Posters will be displayed throughout the conference; however, a dedicated poster/wine session will be held on the Wednesday evening.

Accommodation

Accommodation will be in single rooms at three sites: Woolton Hall, Allan Hall and Dalton-Ellis Hall. Woolton Hall is at Owens Park adjacent to the Conference facilities; Allan Hall is approximately 5 minutes walk from Owens Park. Further single rooms and a small number of en suite rooms are available at Dalton-Ellis Hall, which is a 5–10 minute drive from Owens Park. A bus service will be provided for transport between the conference centre and Dalton-Ellis Hall,

Programme

The BSCB and BSDB symposia will run in parallel, and the scientific programme will begin at 9.00 am prompt in the Armitage centre on Wednesday 14th April. Registration for those from outside Manchester will take place on Tuesday afternoon and evening. The registration desk will also be open from 8.15 am on Wednesday 14th April for local participants.

Registration Details

Pre-registration is essential and must be completed by 1st February 1999 in order to avoid a **late registration penalty of £20.** Registrants must complete the official registration form (page 25) and must remit in full to cover all accommodation and meeting costs when submitting their form. Registrants will receive an acknowledgement and receipt and local travel information to assist arrival in Manchester.

Meeting Charges

An all-inclusive package for members of the BSCB and BSDB has been arranged. This includes accommodation, meals and registration for the meeting and represents a significant saving. Note however, that this does not include the conference dinner, for which booking is essential. For those who do not wish to attend the whole meeting, or who do not require the full meal and/or accommodation facilities offered, registration is payable and covers tea, coffee, abstract and programme booklet and social programme, plus the costs of conference organisation and hire of University Facilities. Registrants choosing this option can purchase additional accommodation and meals as required but note that all such requests should be made at the time of pre-registration, and cannot be arranged in Manchester at the last moment.

Registrants who are not members of either the BSCB or the BSDB can apply to join well in advance of the 1st February deadline in order to take advantage of the member package or else must pay the non-member registration fee plus accommodation and meals as required. Application forms to join the BSCB are available on page 29. Note that there are discounted rates available for students. Details of how to pay are given at the foot of the registration form. Young investigators who have been members of the BSCB for more than 12 months and who are presenting a poster or talk at the meeting are eligible to apply for an Honor Fell travel award.

Social Programme

There will be private bar facilities with a late extension on Tuesday and Wednesday night. On

Thursday night, a conference reception and meal will be held in the impressive Power Room of the Manchester Museum of Science and Technology, at which wine and food will be served with live musical accompaniment and demonstrations of Manchester's industrial history. The museum is in Manchester's Castlefield area which has an excellent choice of bars, cafe/bars and restaurants. Note that the conference dinner is restricted in numbers and its cost is not included in the member package.

Accompanying Persons

Accompanying persons must complete a separate registration form. All accommodation and meal costs must be paid at the time of pre-registration, although they are exempt from the registration fee. They may attend the social programme including the conference dinner (providing that this is paid for), but may not attend the scientific sessions and will not receive an abstract/programme booklet.

Car parking

There are car parking facilities at each accommodation venue. At the conference venue itself there is available parking at the Armitage Centre. However, please indicate on the application form whether you intend to drive so parking needs can be adequately met.

Posters and Abstracts

There will be a joint poster session between the BSCB and BSDB which will be accompanied by a wine reception and late bar. Although this will be the formal 'poster session', posters will be displayed throughout the conference and can be viewed at any time from Tuesday evening. All participants are encouraged to present a poster at the meeting, and those presenting posters will be given preference when allocating rooms closest to the conference centre. Poster presentations from students who are members of the BSDB or BSCB and who have not been awarded a higher degree at the time of registration for the meeting will be eligible for the special poster awards and prizes. The top prize will be attendance at the ASCB meeting in the USA; further prizes will include a case of wine and an Olympus Camera. To present a poster, please note the details below and send your abstract electronically to arrive no later than **1st February 1999.** If you have indicated on your form that you are a student society member you will automatically be considered for poster awards if you submit an abstract and present a poster.

How to submit an abstract

Abstracts should be sent electronically, preferably by email (as attachments or else in a text only message) to bscbbsdb@fs1.scg.man.ac.uk or on diskette to:

BSCB/BSDB Conference, 2.205 Stopford Building, School of Biological Sciences, University of Manchester, Oxford Road, Manchester, M13 9PT, UK.

Please identify your abstract in the following way. If sent as an email message, please write BSCB/BSDB followed by your surname (add initials if it is a common name) as the subject field of the message. If sent on diskette or as an attached file, please name the file as BSCB/BSDB-(your surname), or a suitable abbreviation of this if required by your word processor.

Deadline for receipt of abstracts is 1 February 1999. Abstracts should not be more than 300 words, to fit inside a rectangle (16 cm across by 8 cm deep). Figures and diagrams must be capable of being printed on a black and white laser printer and must fit within the allowed space. The abstracts will be made available to all registrants in booklet form at the meeting. The text will not be retyped, so authors are responsible for the quality of the presentation of the abstract. Any errors will appear in the reproduced text. Please draw our attention to any special characters or symbols as these sometimes differ when these are transmitted or converted electronically.

Indicate in the first line the title and authors in capital letters. The name of the author who is responsible for the poster should appear first. Then indicate the laboratory where the research was conducted, the city, the postcode and country.

Symposia and workshops

To view the latest programme plans, check out the BSCB web site:
http://www.kcl.ac.uk/kis/schools/life_sciences/biomed/bscb/bscbmeeting.html

BSCB Symposium

Cellular Machinery – the Dynamic Regulation of Cell Processes

Scientific organisers: Viki Allan and Iain Hagan

Motors and their cargoes

Plenary: Richard Vallee (U. Mass. Medical School) *Cytoplasmic dynein*

Manfred Schliwa (Munich) *Motors in fungi*
Vladimir Gelfand (Urbana) *Actin- and microtubule-based pigment granule movement*
Rainer Pepperkok (Heidelberg) *Visualisation of ER–Golgi traffic in living cells*
Marino Zerial (Heidelberg) *Endosome motility and its regulation*
Justin Molloy (York) *Single molecule myosin motility*
John Kendrick-Jones (Cambridge) *Unconventional Myosins*
Rob Singer (New York) *Spatial regulation of mRNA localization*
John Carson (Connecticut) *Myelin Basic Protein mRNA movement in oligodendrocytes*
Don Ingber (Boston) *Localisation of mRNA at adhesion complexes*

Cellular organisation and dynamics

Mark McNiven (Rochester) *Dynamin function*
Clare Waterman-Storer (North Carolina) *Microtubule dynamics and ER movement*
Mike Way (Heidelberg) *Viral manipulation of the actin cytoskeleton*
Jürgen Wehland (Braunschweig) *Bacterial manipulation of the actin cytoskeleton*
Paul Nurse (London) *Polarity and tip growth in fission yeast*
Kathryn Ayscough (Dundee) *Polarity of the actin cytoskeleton in budding yeast*

Mitosis

Plenary: Tim Mitchison (Boston) *Cytoskeletal dynamics (To be confirmed)*

Conly Rieder (New York) *Chromosome movement and spindle formation*
Andreas Merdes (Edinburgh) *Spindle assembly in vitro*
John Kilmartin (Cambridge) *Budding yeast spindle pole body*
Jeff Errington (Oxford) *Bacterial chromosome segregation*
Kim Nasmyth (IMP, Vienna)
Tetsuya Hirano (Cold Spring Harbor) *Chromosome condensation in Xenopus*
Iain Hagan (Manchester) *Co-ordinating spindle formation and the cell cycle*
Bill Earnshaw (Edinburgh) *INCENPs and cytokinesis*
Viesturs Simanis (Lausanne) *Cytokinesis in Fission Yeast*
Viki Allan (Manchester) *Cell cycle regulation of organelle movement*
Dave Shima (London) *Partitioning of the Golgi apparatus during mitosis*
Carl Smythe (Dundee) *Nuclear envelope dynamics*

BSCB workshops

Signalling and cellular motility

Scientific organizers: Anne Ridley and Robert Insall

Robert Insall (London) *Signalling in Dictyostelium*
Karl Saxe (Atlanta) *Signalling in Dictyostelium*
Pat Doherty (London) *Signalling in neuronal outgrowth*
Lena Claesson-Welsh (Uppsala) *Signalling in an in vitro model for angiogenesis*
John Collard (Amsterdam) *Signalling and invasion/ migration*
Anne Ridley (London) *Signalling in macrophage migration*

Paolo Comoglio (Turin) *Signalling and SF/HGF*
Ira Herman (Boston) *Calpain/Ca in migration*
John Cooper (St. Louis) *Control of motility of actin patches in yeast*

Dynamics of cell-adhesive contact structures
Scientific organizer: Jo Adams

Andrew Matus (Basle) *Dynamics of dendritic spines*
Rick Horwitz (Illinois) *Integrin dynamics; integrin/cadherin crosstalk*
Birgette Jockusch (Braunschweig)
Jo Adams (London) *Cell adhesion and microspike formation*
Nick Brown (Cambridge) *Hemidesmosomes*
Vic Small (Salzberg) *Title to be decided*

Mechanisms of establishing and maintaining epithelial/endothelial cell polarity
Scientific organizer: Tom Fleming

Mary Weiss (Paris) *HNF4 transcription factor control of hepatocyte differentiation*
Bruce Stevenson (Alberta) *Tight junction cytoplasmic domains*
Elisabetta Dejana (Milan) *Endothelial cell adhesion*
Enrique Rodriguez-Boulan (New York) *Epithelial membrane porarity*
Vania Braga (London) *Keratinocyte cadherin adhesion mechanisms*
Jerry Kidder (Ontario) *Na pumps and epithelial differentiation in early mouse embryo*
Charles Streuli (Manchester) *Control of polarity in breast epithelia*

BSDB Symposium

Cell Polarity and Development

Scientific organizer: Daniel St. Johnston

Asymmetric divisions and cell fate determination
Richard Losick (Harvard) *Sporulation in Bacillus*
Tony Hyman (EMBL) *Asymmetric spindle positioning in C. elegans*
Julie Ahringer (Cambridge) *Spindle orientation in C. elegans*
William Chia (Singapore) *Drosophila CNS*

Localised determinants and axis formation
Colin Brownlee (Plymouth) *Fucus axis formation*
Ken Kemphues (Cornell) *Germ cell determinants in C. elegans*
Anne Ephrussi (EMBL) *Pole plasm in Drosophila*
Lawrence Etkin (Texas) *Germ plasm in Xenopus*
Liz Robertson (Harvard) *Early mouse development*

Cell polarity, morphogenesis, and cell migration
Plenary: Eric Wieschaus (Princeton) *Gastrulation in Drosophila*
Martin Hüllskamp (Tübingen) *Arabidopsis trichome development*
John Chant (Harvard) *Yeast polarity*
Jeff Hardin (Wisconsin) *Gastrulation in C. elegans or sea urchins*
Elisabeth Knust (Dusseldorf) *Epithelial cell polarity in Drosophila*
Maria Leptin (Cologne) *Gastrulation in Drosophila*
Ruth Lehmann (NY) *Pole cell migration in Drosophila*
Mark Krasnow (Stanford) *Tracheal cell migration*
Andrea Brand (Cambridge) *Drosophila*

Axon guidance
Robert Pruitt (Harvard) *Pollen tubes*
Gian Garriga (Berkeley) *C. elegans*
Guy Tear (London) *Drosophila*
Friedrich Bonhoeffer (Tübingen) *Mammals*

BSCB/BSDB Spring Meeting

University of Manchester, 13–16 April 1999

Name ..... Prof / Dr / Mr / Ms

Address .....

.....

.....

Telephone ..... Fax ..... E-mail .....

Registration fee

Member	**£80**
Non-member	**£100**
Student member	**£25**
Student non-member	**£35**

Accommodation will be in the University of Manchester. The availability of on-site accommodation is limited; however, additional accommodation within a short walk (approximately 5 mins) is also available, as is a more distant facility from which a bus service will be provided (approximately 5–10 mins travel time to the conference venue). A limited number of rooms will have available en-suite facilities. Each accommodation venue has on-site breakfast facilities.

The registration fee includes programme/ abstract booklet, tea/coffee, attendance at the scientific sessions and the social programme, excluding the conference dinner. The conference dinner is limited, in the first instance, to 300. However, if there is sufficient interest this can be expanded to 500. Students have a reduced registration, **provided evidence of status is supplied with this form. There is a £25 reduction in registration fee for BSCB members who present a poster. To receive this discount a COPY of the poster abstract must accompany this form (the original abstract must be submitted electronically). Only one reduced registration per poster.**

	Tues 13	Wed 14	Thurs 15	Fri 16	Totals
Member Package Standard (£165)					£
Member Package En suite (£200)					£
Lunch (£8.50/day)		8.50	8.50	8.50	£
Dinner, B&B, Standard (no dinner Thurs)	31.50	31.50	31.50		£
Dinner, B&B, En suite (no dinner Thurs)	47.50	47.50	47.50		£
Registration (see above)					£
Conference dinner			20.00		£
Late booking fee*					£
Reduction for poster presentation (members only) – £25					£
				Total	£

Special dietary requirements .....

Please state if you intend to drive and require car parking .....

Payment should be by cheque, and these must be made out to "**BSCB Spring Meeting**" and must be in POUNDS STERLING. We regret that payment by credit card is not possible.* The late booking fee will apply to forms received after 1st February 1999.

Return this form to: BSCB/BSDB Conference, 2.205 Stopford Building, School of Biological Sciences, University of Manchester, Oxford Road, Manchester, M13 9PT. Queries Tel: 0161 7980707 or 0161 2755083

Forthcoming meetings

Sixth Joint American Society for Cell Biology/European Molecular Biology

Membrane Trafficking and the Cytoskeleton: An Integrated View
Mario Negri del Sud, Chieti, Italy. June 26–30, 1999

Scientific Organizers: Viki Allan (Manchester), George Bloom (Texas), Thomas Kreis (Geneva-posthumously), Jennifer Lippincott-Schwartz (NIH).

This meeting represents an opportunity to bring together two distinct groups whose research efforts have been aimed for many years at a common topic: membrane transport, particularly within the secretory and endocytic pathways. Membrane transport is being studied by traditional membrane 'traffickers' at the same time as it is being investigated by 'motorists.' The development of a comprehensive understanding of membrane transport will require integration of data obtained by both groups of researchers. The meeting goal is to bridge the gap of understanding and appreciation that may currently separate the two groups.

For additional information contact Dot Doyle at the American Society for Cell Biology.
Tel: 301-530-7153; fax: 301-530-7139;
e-mail: ddoyle@ascb.org
Further information is available on the ASCB website: http://www.ascb.org/ascb

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TFF-Meeting

Molecular medicine of TFF-peptides: from the gastro-intestinal mucosa to the central nervous system.
Magdeburg, Germany, Sept. 24–27 1999

Subject areas to be covered at this meeting include: TFF-peptides and mucins: biosynthesis, secretion and interactions, physiological and pathological expression of TFF-peptides, TFF-peptides and cell migration processes, TFF-peptides: binding sites and intracellular signaling, TFF-peptides and their functions in vivo medical aspects.

Further information and registration details are available from the organizer:
Prof. Werner Hoffmann
Institut fuer Molekularbiologie und Medizinische Chemie
Universitaetsklinikum
Leipziger Strasse 44
D-39120 Magdeburg, Germany
Tel.: +49-391-67-15895
Fax.: +49-391-67-13096
Information is also available on the website:
http://www.med.uni-magdeburg.de/fme/institute/immc/

BSCB Autumn 1999 Meeting

Endoplasmic Reticulum: Structure and Function

Organizer: Michael Whitaker (Newcastle)

Topics to be discussed include:

- ER structure, remodelling and dynamics
- receptors and channels
- ER and calcium

The programme and registration details will be available early next year on the BSCB website and also in the Spring Newsletter.

Young Cell Biologist of the Year Poster Prize 1998

WIN A TRIP TO WASHINGTON D.C.

Win a trip to the American Society for Cell Biology 39th Annual Meeting and show your work.

All research students are invited to enter the next poster competition at our Spring 1999 meeting at Owen's Park, Manchester, 13–16th April 1999 (main symposia: motors, mitosis and cell organization). The prize is a trip to the USA to attend the 1999 ASCB meeting to be held in Washington D.C., December 11–15 1999, as their guest, with an opportunity to present the winning poster.

The poster will be judged on scientific merit and presentation by a panel of British and American cell biologists.

You are eligible to enter if you are a full-time PhD student registered at a British Institution and a member of the BSCB.

Complete the form below and return it to the BSCB Secretary, Birgit Lane, CRC Cell Structure Laboratory, Department of Anatomy and Physiology, MSI/WTB Complex, University of Dundee, Dundee, DD1 5EH.

Name:

.....

University and Department

.....

.....

Year studies commenced

.....

Address of planned postdoctoral position,

if known:

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Date of commencement of BSCB membership:

.....

Present academic address for correspondence

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.....

E-mail address:

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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.

Applications (including a copy of the meeting registration form) should be sent to Alan Hall (MRC Laboratory for Molecular Cell Biology University College London, Gower Street, London WC1E 6BT)) using a copy of the form below.

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

- Awards are not normally made to applicants aged over 35
- Applicants must have been BSCB members for at least a year.
- No applicant will receive more than one award per year or three *in toto.*
- The applicant must be contributing a poster or talk.

Application for an Honor Fell travel award

Name: .....

Age: .....

Work address: .....

.....

.....

..... Postcode: .....

E-mail address: .....

Degrees (with dates):

.....

.....

Present position (graduate students give start year of PhD): .....

.....

.....

Date of joining BSCB: ....

Record the years of previous Honor Fell awards (if any): .....

Key publications (2) or research interests:

.....

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.....

Meeting for which application is made (Title, place, date): .....

.....

.....

Estimated expenses: Travel: .....

Subsistence: .....

Registration: .....

Other: .....

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

If yes, please give details: .....

.....

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

Supporting statement by Head of Department:

The applicant requires these funds and is worthy of support

Name: .....

Signature: .....

Applicant's signature: .....

Date: .....

Application to join the BSCB

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

Please 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'.

Please complete and return this form and the direct debit form to: Steve Winder, Institute of Cell and Molecular Biology, University of Edinburgh, Michael Swann Building, Kings Buildings, Mayfield Road, Edinburgh EH9 3JR.

Name: ..... Sex: .....

Position: .....

Academic qualifications: .....

Tel: ..... Fax: ..... E-mail: .....

Work address: .....

.....

..... Postcode: .....

Research interests (5 keywords): .....

.....

Membership of other scientific societies: .....

.....

BSCB member proposers (names and signatures):

1) .....

2) .....

Applicants without proposers should enclose a brief curriculum vitae.

Applicant's signature: ..... Date: .....

British Society for Cell Biology

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Please complete parts 1 to 6 to instruct your branch to make payments directly from your account. Then return the form to: **British Society for Cell Biology, c/o Dr Steve Winder, Institute of Cell and Molecular Biology, University of Edinburgh, Michael Swann Building, Kings Buildings, Mayfield Road, Edinburgh EH9 3JR.**

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- If an error is made by the BSCB or by your Bank/Building Society, you are guaranteed a full and immediate refund from your branch of the amount paid.
- You can cancel a Direct Debit at any time, by writing to your Bank or Building Society. Please also send a copy of the letter to the BSCB.

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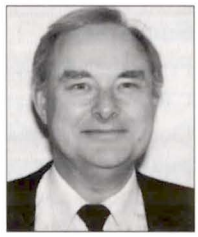

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or michael.whitaker@ncl.ac.uk

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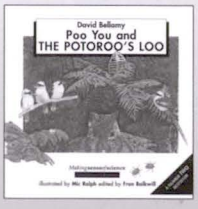

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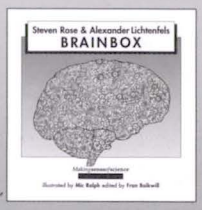

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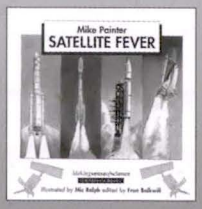

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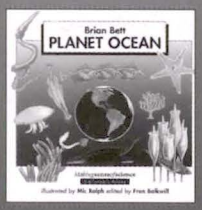

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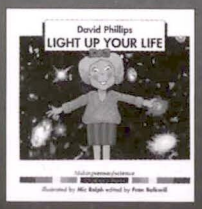

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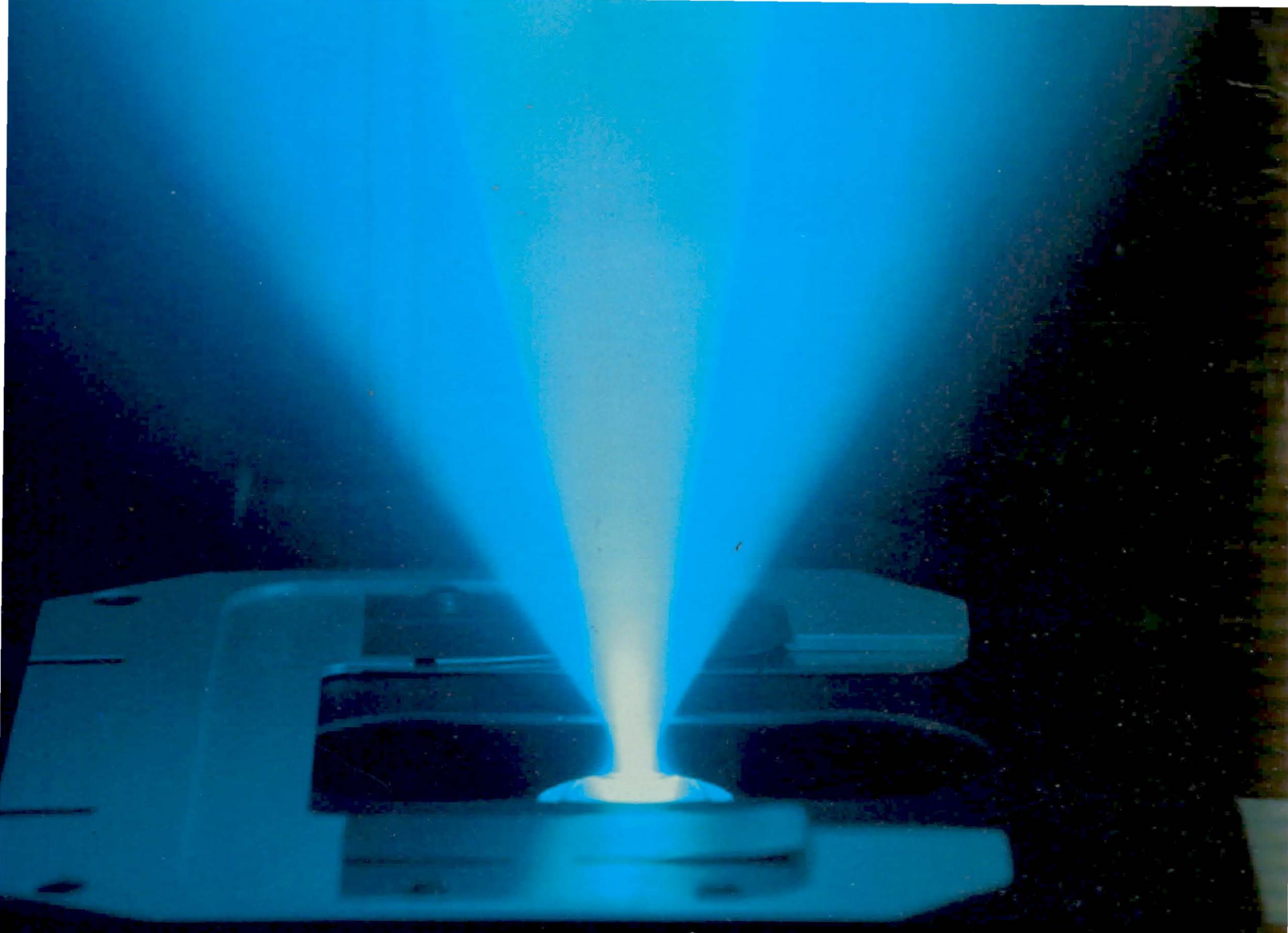

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