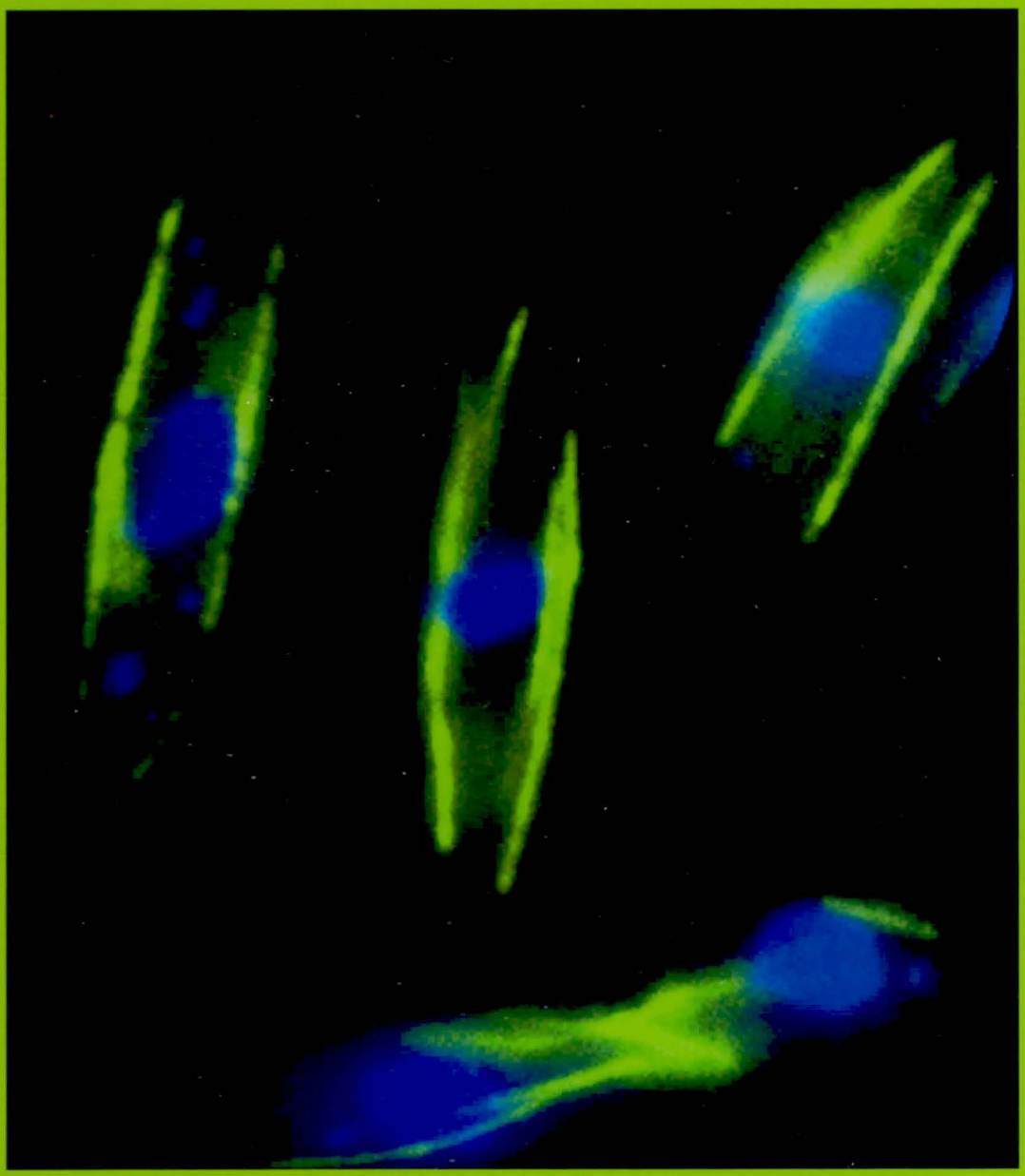
BSCB NEWSLETTER



BRITISH SOCIETY FOR CELL BIOLOGY

Summer 2000

NEWS
UK Life Sciences Committee

FEATURES
BSCB Young Cell Biologist 1999

Cell biology in Austria

MEETING REPORTS
BSCB/BSDB Joint Spring Meeting, Warwick

Dynamics of the Cytoskeleton, Keystone

Myology Meeting, Nice

FORTHCOMING MEETINGS
BSCB Autumn Meeting 2000 – Cell and Molecular Biology of Apoptosis

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

Submission:
If you have an idea for an article please email the editor a brief outline first. Appropriate colour images are welcomed for consideration for the front cover.

It is preferable to send all articles, reports and images by email (though alternatives can be arranged after contacting the editor). Attachments for text are best received in Microsoft Word and images as 200-300 dpi JPEG/TIFF or Photoshop files. Hard copy images can also be sent.

Submission of articles and images should be made to
Dr. Kathryn Ayscough, Institute of Biomedical and Life Sciences, Davidson Building, Glasgow University, Glasgow, G12 8QQ. Tel: 0141 330 3595 Fax: 0141 330 2707
Email: kayscough@bio.gla.ac.uk

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

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

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

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

BSCB Newsletter
Summer 2000

Editorial

Welcome to the first Newsletter of this Millennium. Like many other organisations, we have been spending time looking at where we have come from and where we now see ourselves heading in the future. Our new president, Fiona Watt gives her thoughts on the subject later in the newsletter.

Also in this issue, the BSCB Secretary Michael Whitaker informs us of the latest moves towards a single representative biosciences body in the UK that could speak authoritatively and speedily to the government and the press about issues of interest or concern. We hear from Mario Gimona about the current state of Cell Biology in Austria and from our Young Cell Biologist from 1999, Fanni Gergely and her trip to the ASCB meeting last December. We have meeting reports from the latest BSCB Spring Meeting in Warwick which had a wonderful programme focussing on *Cell Biology in Disease*, and also from meetings in Keystone and Nice.

We also have the programme and registration forms for this year's BSCB Autumn meeting '*Cell and Molecular Biology of Apoptosis*'. This meeting is being held at Heriot-Watt University on 10–13 September 2000. The line-up of speakers looks excellent so make sure you get your application forms in soon.

Finally, thanks to all those who have contributed to this Newsletter, to the BSCB Committee for their hard work throughout the year and to the sponsors of the BSCB.

The Editor

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Front cover
Schizosaccharomyces pombe cells stained with TAT1 anti-tubulin antibody (green) and co-stained for DNA using DAPI (courtesy of Alison Pidoux).

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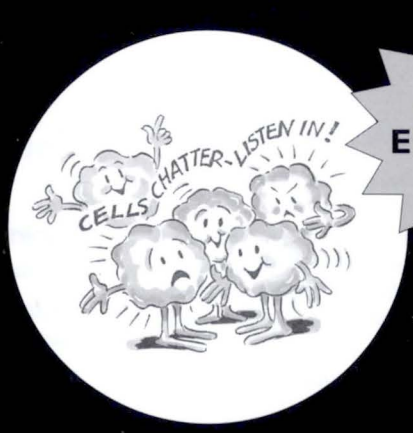

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NEWS

The British Society for Cell Biology – how are we now?

I am delighted to be the new President of the BSCB, and I am glad to have this opportunity to make some comments about the Society.

I joined the BSCB when I was a PhD student and the most valuable things about the society then are still the same today. It was at BSCB meetings that I first had the chance to hear talks by the famous Cell Biologists whose work I had only had the chance to read until then. It was tremendously exciting to hear about the latest results and to be able to go up and talk to the speakers. I was struck then (and I think it is still true now) how friendly and approachable most invited speakers were. It was interesting at this year's Spring meeting to hear one of them say that she was amazed by how young and enthusiastic the delegates were, and how favourably this compared with meetings of the ASCB.

Getting to watch the scientist megastars in action is only one reason to attend the BSCB meetings. Another important benefit is being able to present your own work, either in poster form or as a short talk. It is very stimulating to have the chance to explain what you are doing to a broad and knowledgeable audience, and it often leads to unsolicited job offers, especially if you happen to be nearing the end of your PhD. The social side of the meetings is just as important as the official program (OK, I admit it, I love to dance) and some of my oldest friends are Cell Biologists I first met at BSCB meetings – I think this is what Americans call 'networking', but it really is painless.

What are some of the other good things about the BSCB? Thanks to the generosity of the Company of Biologists there is an excellent scheme of travel awards (you'll read elsewhere in the Newsletter that the budget for travel awards has just doubled!) and PhD students are particularly encouraged to apply. You should also be aware that as a member of the BSCB you are entitled to reduced subscriptions to the *Journal of Cell Science* and a number of other journals. We enjoy a close relationship with the British Society for Developmental Biology and this certainly enriches the program of the Spring meetings.

We also have the opportunity to voice our opinions about Government science policy through the BSCB's membership of the UK Life Sciences Committee. Finally, the BSCB committee try to be very responsive to suggestions from Society members and if you have any ideas about ways in which the BSCB can serve Cell Biologists better just let us know!

I believe that job opportunities in Cell Biology are better now than at any other time since the mid-1970s. There are many different reasons for this (the massive expansion of the Wellcome Trust budget, the positive effect of the economy on other charities, such as the Cancer Research Campaign, growing opportunities in biotech and large pharmaceutical companies, to name but a few), but the bottom line is that it's a good time to be a Cell Biologist. So have fun and make the most of the BSCB!

Fiona Watt, ICRF, 4th April 2000.

BSCB Young Cell Biologist of the Year 2000

We would like to congratulate Matthew Howard, a PhD student in Clare Isacke's lab in the Department of Biology at Imperial College London, who was awarded BSCB Young Cell Biologist of the Year 2000 for the poster he presented at this year's Spring meeting in Warwick. He will now have an opportunity to present his poster 'ENDO180, a Novel Endocytic Receptor with a Role in ECM Remodelling' at this year's ASCB Meeting in San Francisco. We look forward to hearing about his time at the meeting in a future BSCB Newsletter.

A runner-up prize of a years' subscription to *Journal of Cell Science* donated by the Company of Cell Biologists, was awarded to Fanni Gergely. We are also trying to contact past winners of the Young Cell Biologist poster prize – so if there are any of you out there who are reading this, we would be grateful if you could contact the Newsletter editor.

A single national voice for biosciences

You may remember Martin Raff's article in the Winter 1998 newsletter in which he described the aims and workings of the UK Life Sciences Committee (UKLSC).

UKLSC has joined with the UK National Committee for Microbiology and the Institute of Biology to pursue the idea of a single UK body that could speak authoritative and speedily to the government and the press about issues of interest or concern (GM food is an obvious example) and set a strategy for the bioscience community. This trio has commissioned an independent consultant, Brian Jamieson Associates, to look into how effective the bioscience community was at coordinating its activities and to suggest various ways of setting up a UK body. The consultant's report can be read at: http://www.lifesci.org, the UKLSC's website.

In response to the report, a group (Professor R McNeill Alexander, Professor Charles A Fewson, Dr Brian A Jamieson, Dr Laurence H Smaje, Professor Sir David Weatherall, Professor John B. Whitaker and Dr Alan R Williamson) has been set up to consult Societies and others (for example, the Wellcome Trust and the Research Councils) about the value of the idea. This group will recommend in June whether or not to proceed to the next stage of determining exactly how a UK Federation of Bioscience societies might work. Putting together a framework for a UK Federation would then take another six months to one year.

The BSCB Committee supports the idea of going on to the next stage. Individual members may comment to the group by email to Brian-jamieson@compuserve.com.

We have not, of course, committed BSCB to joining a UK Bioscience Federation, should it be set up. We should be interested to hear your views. Please send them to me.

Michael Whitaker
michael.whitaker@ncl.ac.uk

The BSCB Hooke Medal

We were extremely pleased to announce Anne Ridley as the first winner of the BSCB Hooke Medal. The medal was presented at this year's annual spring meeting in Warwick. Anne gave a wonderful talk both on recent aspects of her lab's work and also on a review of her career at a more personal level – noting key interactions and decision points that led her to where she is now.

Anne was an ideal first winner of the BSCB Hooke Medal, as she epitomises the essence of what the society wishes to recognise. The medal is to be awarded annually to an emerging leader in cell biology. Usually, it would be expected that the award will be presented to someone with no more than 10 years of independent research which has largely been conducted within the UK.

The BSCB invites nominations for next year's Hooke Medal from any BSCB member and these should be sent, with a few lines outlining why the person nominated would be a suitable recipient of the Hooke medal, to the Secretary, Michael Whitaker by 14th July 2000.

The medal shows Robert Hooke's microscope and the cork cells he first described. It was designed by Dr Brad Amos.

Membership Information

As many of you are aware we are trying to update our membership databases to ensure that we have current information on all members – at present, a large number of Newsletters are sent back to us. We are also trying to include email addresses on the database.

So, if you think we do not have your current details please contact us by email on *zoo-jeb01@lists.cam.ac.uk* or by mail to Margaret Clements at the address at the back of the newsletter. If you did not receive email notification of the Spring Meeting in Warwick then we don't have your email address – so could you also send this to the address above.

We are also announcing, for the first time in 6 years, a rise in the membership subscription rate. The new rate, if paying by Direct Debit, is £25 for full members or £10 for students, school teachers and retired members. The rate is higher if not paying by direct debit because of the extra time involved in processing (details on inside cover, and the application form at the end of the newsletter).

Please note that if you are paying by Standing Order you will now be paying the wrong subscription amount. Notification of these changes should also have been received by all members by mail. Again, if you have not received this letter it is probably because we do not have your correct details.

The society has also introduced membership numbers for all members paying the appropriate subscription. If you do not receive a number, please update your subscription and it will be forwarded to you.

New address for the BSCB website

The BSCB has a new website address: http://www.bscb.org

The new website will provide the same information as the previous site, such as background about the BSCB, forms for joining and travel awards, PDF files for newsletters and links to BSCB Meetings sites and other Cell Biology organisations. However, we also hope to increase the number of links for educational purposes.

So, as a starter for those on the lookout for potentially useful sites, here are a just a few addresses that we have checked and seem to have useful ideas and information of interest to both children and their teachers.

Possible resources for teachers:
http://schmidel.com/bionet.cfm
Includes downloadable articles from various scientific sources, including *Scientific American* and *New Scientist*, as well as lists of other sources available for accessing material.

More for children:
Simple cell biology and concepts.
http://www.kapili.com/biology4kids/cell/index.html

There are also companion sites for chemistry, and ecology.

A Howard Hughes Medical Institute Initiative.
Cool Science for Curious Kids
http://www.hhmi.org/coolscience/

Neuroscience for kids
http://faculty.washington.edu/chudler/neurok.html

Changes on the BSCB committee

At the AGM this year we officially welcomed Fiona Watt (ICRF) as our new president. Fiona has been President elect of the Society for the last year. We also welcomed Michael Whitaker (University of Newcastle) from the BSCB Committee who is the new BSCB secretary after Birgit Lane stepped down at the end of last year.

We are also very grateful to Peter Shaw and Viki Allan, who have retired from the BSCB Committee, and greet new committee members Jonathon Pines (Cambridge), Roy Quinlan (Dundee), Jo Adams (UCL) and Angus Lamond (Dundee) onto the committee. Details of the current BSCB Committee are detailed at the back of the Newsletter.

Please note that any BSCB members can nominate themselves or fellow cell biologists for election to the committee, and nominations are welcomed throughout the year. Nominations should be sent to the BSCB secretary Michael Whitaker. New committee members are then elected at the AGM – which next year will be held during the Spring meeting in Sussex.

Honor Fell Travel Awards

We are also pleased to be able to announce that, through a generous donation from the Company of Biologists, we have doubled the funds we have available for travel awards through the Honor Fell Scheme. An application form is given at the end of the Newsletter, but the most important changes to the awards are that there is now a sliding scale of funding available. Awards of up to £250 can be given for meetings in the UK, up to £350 for European meetings and up to £450 for meetings in the rest of the world.

School News

David Archer. BSCB Schools Liaison Officer

Evolution of GCE 'A' level

From September 2000, the UK, except Scotland, will see changes in the structure of the post-16 General Certificate of Education (GCE) 'A' level courses and qualifications. The changes will be the most extensive since 'A' levels were introduced in the 1950s.

The changes are designed to: (1) retain the 'gold standard' of 'A' level; (2) broaden the number of subjects that can be studied post-16 and at the same time enable the choice of subjects for more specialist and advanced study to be deferred; and (3) give greater access to a post-16 qualification for those students who do not wish to pursue a full 'A' level course in chosen subjects or to those who might wish to study in an incremental way, taking a step at a time. In the spirit of life-long learning it might be possible for students, especially those in further education, to 'bank' units as they progress and take the advanced GCE course over a longer period of time.

From September 2000 the traditional 'A' level will be replaced by a GCE having two parts. One part will be the Advanced Subsidiary GCE (AS.GCE). The other part is called Advanced or A2. Students who qualify in a subject at AS and A2 will be awarded Advanced GCE

Advanced Subsidiary GCE in Biology

This option will normally be studied during one year as one subject within a group of three, four or perhaps five subjects. In biology, the first certification year will be 2001 when students who qualify will be awarded an AS GCE grade in biology. This will be a 'stand-alone' qualification and some students will finish their study of biology at this point. Biology at AS level will be presented in modular form and with three units to study. It is expected that AS GCE biology will be studied by rather more students than have elected to study the traditional two-year 'A' level subject in the past.

Advanced or A2 GCE in biology (the second part)

The second part of the 'A' level package is called Advanced or A2. In schools A2 will normally be taught in the second year of advanced level studies along with two or three other subjects. It is expected that AS GCE would be taken before embarking on advanced A2 work. There are three units of study in A2 advanced biology with some measure of choice. In the A2 examinations students have to answer a synoptic paper.

In a Further Education and life-long learning situation it may be possible to study both AS and A2 advanced in the same year. A break between AS and A2 would also be possible. There is no stand-alone qualification for success in the advanced A2 part, since it is intended that A2 should build on AS to form the full Advanced GCE qualification. The AS and A2 parts each contribute 50% of the total Advanced GCE mark. The first year of the new certification in Advanced GCE in biology will be 2002.

The Key Skills qualification

Work in biology at AS and Advanced A2 level can contribute to this qualification, which covers the transferable key skills of communication, application of number, information technology, problem solving, working with others and improving own learning and performance.

Please note the comments above are general in nature. Educational establishments may not be able to offer teaching in some of the ways described and the regulations of the appropriate examination boards should be consulted for detailed information. Members of the BSCB will be interested to know that cell biology is well represented in AS GCE biology. In time this should mean that a greater percentage of the general population will have a basic knowledge of cell and tissue biology.

New Universities and Colleges Admissions Service (UCAS) Tariffs

In parallel with the awarding of the first results of the new A2 Advanced examinations in 2002 will come a new tariff or points score system from UCAS. At present an 'A' grade at 'A' level has a numerical value five times as great as an 'E' grade. Under the revised tariff an 'A' grade will be three times the value of an 'E' grade. The main new values are summarized below.

LEVEL AS and A2 level	UCAS tariff (A*)	Scottish**
A2 grade 'A'	120 (10)	Advanced Higher 'A' grade
A2 grade 'B	100 (8)	Advanced Higher 'B' grade
A2 grade 'C'	80 (6)	Advanced Higher 'C' grade
	72	Higher 'A' grade
AS grade 'A'/ A2 grade 'D'	60 (4)	Higher 'B' grade
AS grade 'B'	50	
	48	Higher 'C' grade
AS grade 'C'/ A2 grade 'E'	40 (2)	
AS grade 'D'	30	
AS grade 'E	20	

*UCAS tariff. (Old 'A' level score in brackets)
**Scottish framework qualifications

More details, including the tariff rating of GNVQ results are available from the UCAS Website. http://www.ucas.ac.uk/new/press/tariff/html

The ASCB Meeting from a young biologist's point of view

Fanni Gergely

After the BSCB spring meeting, I soon forgot that in addition to the pretty sounding Young Cell Biologist of the Year title, my prize also included a free trip to the ASCB meeting in Washington. However, towards the end of November, my correspondence with Dr Kellie (the treasurer of the BSCB) got busier and busier and as I started getting bombarded with massive numbers of leaflets from various biotech companies, I started realising that I was really to go to Washington to attend one of the biggest scientific meetings.

Although I tried to get familiar with the abstract book while still at home, asking for advice on what to go to and what to miss, the first real opportunity to delve into the abstracts came only on the plane. After a few minutes of trying to adapt my eyes to the size of the characters in the abstract book, I concluded that it was a mistake not to take a magnifying glass with me. Even the stewardess could not pass me without looking horrified and wondered how the publisher could have expected anyone to read that.

I spent two hours in my room recovering from my jet lag and then headed to the Washington Convention Centre, where the meeting was held. My first impression was 'ohmygod', how can I find anything and anyone at such an immense place. However after having noticed a few familiar faces in the crowd, I started relaxing and relating to the conference as (I guess) one was supposed to. Huge but accessible, highly organised but informal.

The 'kick-off' plenary session was given by David Botstein followed by Gerald Rubin and Cornelia Bargmann, all describing the advances made in genome-wide studies by sequencing the genomes of model organisms. Exploration of the genome using DNA microarray techniques makes it possible to assess the mRNA profiles not only of individual cells but also of whole tissues. Therefore comparing profiles of stem to differentiated cells, cycling to quiescent cells, healthy to diseased tissues will provide us with all the candidate genes that could be involved in bringing about these fundamental changes.

My first thought after the plenary session was: that is all nice and well, the biotech companies will do all the array comparisons, isolate the genes, develop the drugs, so where is the need for me, for bioscientists outside industry? But when I took it further, I realised that although cDNA microarrays are very efficient means to characterise variation in human gene expression, no revolutionary (at least, not in the last year) means were developed to study the function of genes, so while genome-wide studies will undoubtedly accelerate the isolation of novel genes, their actual characterisation will take as much time and effort as it is taking now. Good news for young cell biologists, I guess.

The plenary sessions covered broad areas of cutting-edge cell biology, but unfortunately their size was a bit intimidating for open discussions. The minisymposia were on a much more tolerable scale, and could therefore cater for my taste for useful comments and heated debates. I especially liked the session on cytoskeleton assembly and dynamics (please, excuse me for my biased opinion), as it provided such an excellent overview of recent advancements in the understanding of the behaviour of microtubule and actin cytoskeleton from *Dictyostelium* to mammalian cells. Finally, I cannot fail to mention Dr Alsop's talk on cleavage furrow positioning in animal cells, which was not only very interesting but also I could not stop being amazed by the fine microsurgical technique they were using to manipulate mitotic spindles in living cells.

Although I mentioned plenary sessions and minisymposia already, it is only now that I reached my favourite part of the conference: poster sessions. Posters were big, and there were a great number of them, just like everything else at this meeting. However they were well presented and nicely distributed, so despite 600 posters being on display every day for four days in a row in a hall of 250,000 square feet, one could still find it enjoyable to read, discuss or contemplate on them. And when I grew tired, I walked up to one of the biotech companies, checked out their always colourful displays in exchange for some ever useful goodies, such as cookies and key-rings. Obviously, the conference was not only about science, a lot of talks concentrated on social aspects of today's science education and constant career advice was provided to lost PhD students and postdocs.

When science became a bit overwhelming, I sneaked out and indulged myself with a few visits to museums, restaurants and cafes in Washington. Another unforgettable experience involved a visit to an exhibition by the Hungarian photographer, Brassai, called The Eye of Paris which was a compelling portrait of cosmopolitan life in Paris from the beginning of the 20th century. I felt there was something common between us: we both left Budapest and went a long way before reaching Washington – Brassai through Paris, and myself through Cambridge.

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I would like to thank Kim Jeffers, Debbie Kidd and Jordan Raff for their contribution to the work I presented on the winning poster and the BSCB for the opportunity to attend the ASCB meeting.

Cell biology in Austria

Mario Gimona

Austrian science is best known for its contributions to medicine, physics, chemistry and mathematics. Cell biology research is not a traditionally strong area, but internationally recognized and competitive groups have emerged over the last two decades.

Austrian research is divided between universities and basic research institutions. The latter includes several units financed by the Austrian Academy of Sciences, or research institutes founded in collaboration with industry, like the Institute of Molecular Pathology (IMP) and the Institute of Molecular and Cellular Bioinformatics (IMBA). The University of Vienna also runs a Biocenter in close proximity to the IMP (see http://www.ac-info.ac.at/index-de.html for a complete overview of Austrian Universities and other research institutes). Close collaboration takes place between the universities and the associated basic research institutions. Research and student life at Austrian Universities is very different from that at UK universities. Most department heads have heavy teaching commitments and campus life is not really possible as there is rarely a campus. Nevertheless, there are a number of excellent and internationally recognised cell biology labs in Austria (see Figure 1). Recent recommendations for future developments, put forward by the ministry and the Austrian Biochemical Society, focus on the establishment of Biocenters in Graz, Innsbruck and Salzburg – which should lead to a strengthening of molecular and cell biological research in Austria in the new millennium.

Figure 1.
Universities and research institutes with active cell biology labs are found in the state capital Vienna, but also in Salzburg, Graz and Innsbruck.

Research sites in Vienna

The Institute of Molecular Pathology (IMP)

The IMP (www.imp.univie.ac.at) under the directorship of Kim Nasmyth and located next to the Vienna Biocenter is the top address in Austria for basic research at an international level. Due to its unique structure and funding system this institute has attracted top-quality group leaders and post docs.

The internationally best known groups are those of Hartmut Beug (developmental plasticity TGF receptor in tumorigenesis), Meinrad Busslinger (*Pax* gene function in brain development, hematopoiesis and disease, Gerhard Christofori (molecular mechanism of multistage tumor development), Barry Dickson (axon guidance in *Drosophila*), Michael Glotzer (mechanisms of cytokinesis in *C. elegans*), Lukas Huber (epithelial polarity, wnt signaling), Jürgen Knoblich (asymmetric cell division during *Drosophila* nervous system development), Kim Nasmyth (chromosome segregation during mitosis and meiosis) and Erwin Wagner (gene function in mammalian development and oncogenesis, Fos and Jun).

The Vienna University Biocenter

The Biocenter is split into several departments. In the Department of Biochemistry, a number of groups are working on signal transduction and cell cycle control in yeast (Gustav Ammerer) and the biogenesis of peroxisomes (Andreas Hartig). The Department of Molecular Cell Biology harbors active groups studying the molecular and cellular biology of cytoskeletal linker proteins in morphogenesis, cytoarchitecture and signal transduction (Gerhard Wiche), cell cycle and differentiation-dependent dynamics of cytoskeletal and matrix proteins (Roland Foisner), and MAP1B and other cytoskeletal components during differentiation (Friedrich Propst).

At the Institute of Medical Biochemistry, also situated at the Vienna Biocenter, the Department of Molecular Biology concentrates on terminal erythropoiesis (Ernst Müllner), the regulation of PP2A in normal and transformed cells (Egon Ogris), thymidine kinase as a marker for malignant state of cells (Edgar Wawra), and growth factor-dependent gene expression in mammalian cells (Christian Seiser). The Biocenter runs an (inter-)active seminar and lecture series, allowing Ph.D. students to invite up to four seminar speakers of their choice per year. Since 1993 the center offers an attractive and modern Ph.D. programme which is open for students from all over the world.

Other sites around Vienna

These include the General Hospital Vienna in which basic research is provided by five divisions within the Department of Pathophysiology, which is part of the Medical School of the University of Vienna. Cell biology groups here include those of of Renate Fuchs, working on transcytosis in polarized cells and the cellular pathophysiology lab (Jürg Graf) focusing on membrane and Ca^{2+} transport in hepatocytes.

At the Institute for Cancer Research (Vienna) research groups study the molecular mechanisms of cell division and actin isoforms in yeast (Ursula Wintersberger), nuclear proteins (Rolf Schulte-Herrmann) and cell-cycle regulation (Jozefa Gadek-Wesierski),

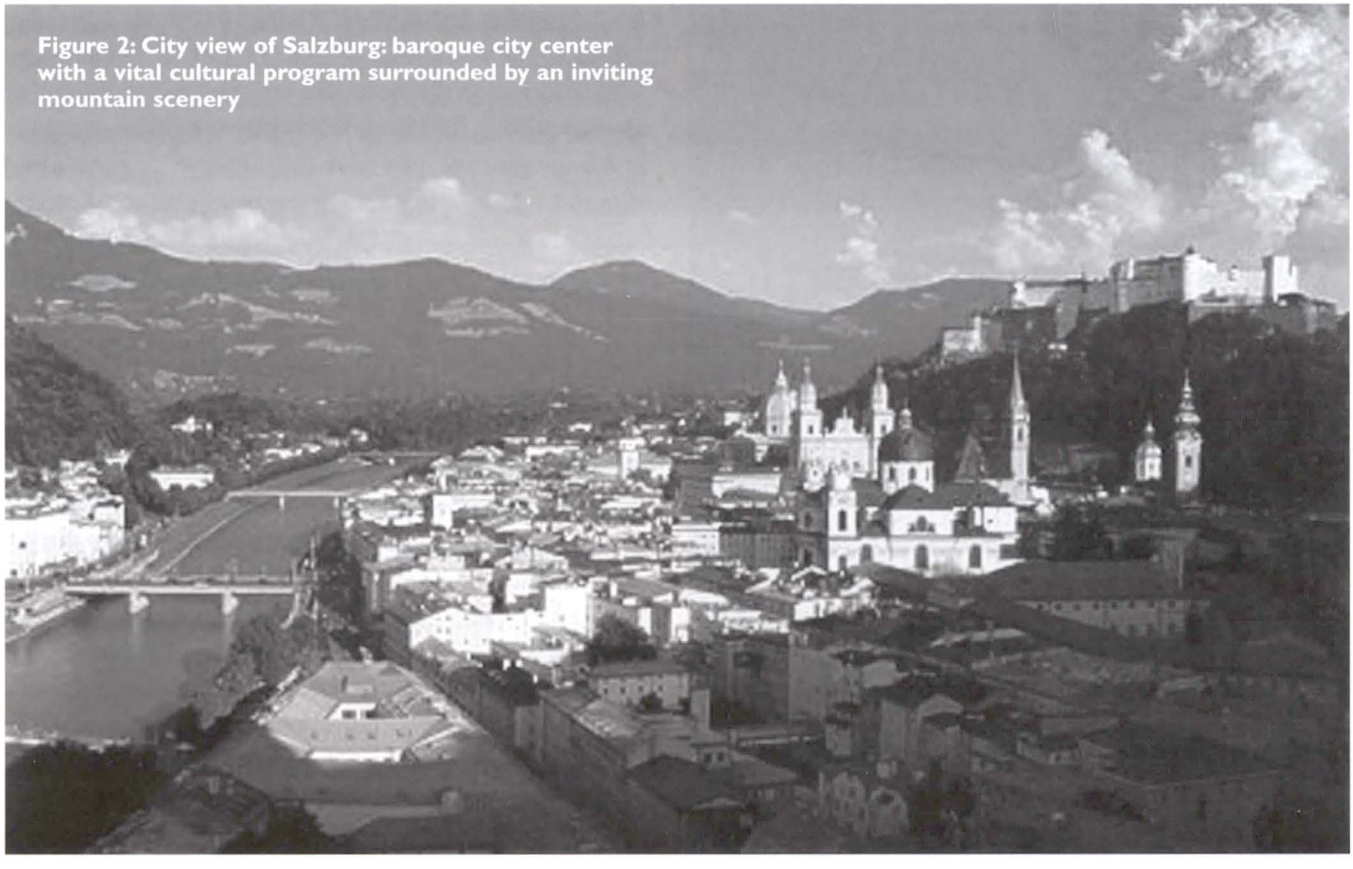

Figure 2: City view of Salzburg: baroque city center with a vital cultural program surrounded by an inviting mountain scenery

and at the Institute of Vascular Biology and Thrombosis Research, the cell biology focus is on the role of fibrinolytic systems in tumor biology (Bernhard Binder).

Universities outside Vienna

The faculty of natural sciences at the University of Salzburg is a large, multidisciplinary building containing the Institutes of Botany, Plant Physiology, Zoology, and Genetics, among others. At the Institute of Genetics and Molecular Genetics, research centers on the genetic and cellular mechanisms of growth control and aging in yeast (Michael Breitenbach).

Due to its strong medical and biochemical faculties, the University of Innsbruck is an excellent place to study and has a population of around 26,000 students. The University (www.uibk.ac.at) is divided into several campuses. Internationally competitive groups are found at the Institute of Medical Biology and Human Genetics, focusing on PKC isoform function in cellular signaling (Gottfried Baier), and at the Institute of Medical and Clinical Chemistry studying mitogenic signal transduction in mammalian cells (Hans Grunicke).

Internationally active research groups at the University of Graz can be found in the Department of Pathology and Medicine, studying cytokeratins CK8/18 (Kurt Zatloukal), and at the Institute of Medical Biochemistry interested in atherosklerosis, lipoproteins and tumor cells (Gert Kostner). Reach the University of Graz at www.kfunigraz.ac.at .

The Austrian Academy of Sciences (ÖAW)

Founded in 1847 (!), the general purpose of the Austrian Academy of Sciences (www.oeaw.ac.at) is the support of basic research in all fields with an emphasis on complementing the research activities at Austrian universities. Among the 56 different research institutions currently operated by the ÖAW, the Institute of Biomolecular Aging (IBA) in Innsbruck and the Institute of Molecular Biology (IMB) in Salzburg harbor a number of active research groups. The third noteworthy institute, the Institute of Molecular and Cellular Bioinformatics (IMBA) is currently in its construction and recruiting phase. This institute, a collaboration between the ÖAW, the IMP and the pharmaceutical company Boehringer Ingelheim, is a new approach of the ÖAW to focus research on molecular and cellular biology relevant to humans. This institute is anticipated to bridge the gap between basic and applied science and to serve as a platform for the development of new strategies for the production of therapeutics. The IMBA is expected to develop close collaboration with the Vienna General Hospital and the Vienna Biocenter. The groups will also serve to train diploma and Ph.D students in addition to hosting a number of post docs.

Work in the Department of Cell Biology at the IMB in Salzburg (www.imolbio.oeaw.ac.at) concentrates on the mechanisms of cell movement and regulation (Vic Small), and on the molecular cell biology of actin-modulating proteins (Mario Gimona). This late 70s-style, ÖAW financed unit also hosts groups interested in plant and developmental genetics, biochemistry and virology. The institute is internationally recognized for its contributions to cytoskeleton dynamics and operates a state-of-the-art real time imaging microscopy facility.

The IBA was founded in 1992 and harbors four independent departments under the roof of its bucolic historical, but well-equipped building. Work in the Department of Molecular Cell Biology centers around the mechanisms of cellular aging, and the roles of tumor viruses in senescence and immortalization (Pidder Jansen-Dürr) while the Department of Pathology concentrates on the involvement of heat shock proteins in atherosclerosis (Georg Wick).

Funding

Austria has been spending a mere 1.56% of its gross national product (GNP) for science, research and development (compared to 2.16% on average in the OECD) and has repeatedly been under political pressure by the European Community to comply with these standards. Both the current and previous governments have aired plans to increase the expenditure on science and research in the following years to above 2%.

The funding situation in Austria is competitive. Since the recent retraction of the Austrian National Bank

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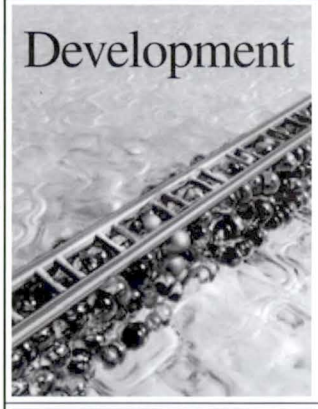

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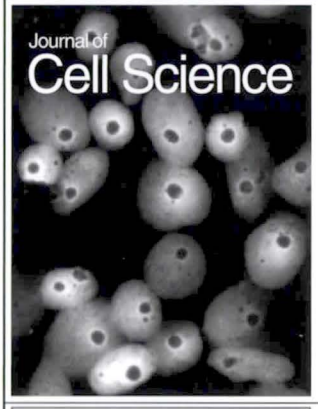

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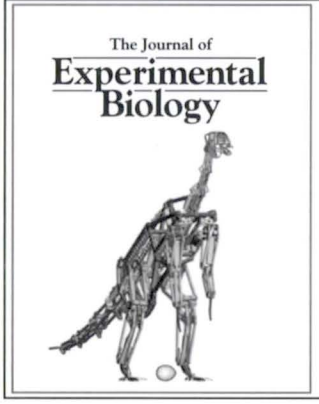

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Deadlines: 31 March and 30 September 2000

from science funding, the weight of research support lies entirely on the single national funding agency, the Austrian Science Foundation (FWF; www.fwf.ac.at). The mandate of the sister organization, the Industrial Research Promotion Fund (FFF) supports applied research and development projects. The FWF runs several programs to support Austrian research and researchers, in addition to providing fellowships for EU and non-EU graduate students and post docs. Approval rate for research grants is at around 44%. Total expenditure in 1998 was 952 million ATS (£42 million); 17% of the successful applications in 1998 have been submitted by female colleagues.

Living in Austria

In contrast to recent media releases, Austria is a liberal country hosting students and post docs from within Europe and abroad. The FWF, the Austrian Academy of Sciences and almost every Austrian university have published open letters to the new government and also to the outside community in order to encourage all scientists to maintain collaborations and contacts with scientists working in Austria.

Owing to the relatively small size of the country (Austria has roughly 7.5 million inhabitants), even the major cities lack a broad cultural diversity. However, knowledge of the English language is common in the Austrian population, due to the myriads of tourists who take over the country during the holiday seasons.

Living is relatively expensive, as is food, clothing and transportation. There is a gradient in living costs rising from Vienna in the east to the more western part of the country (somewhat peaking in Salzburg). Medical support is excellent and readily available. Basic insurance is affordable and includes in most cases medical prescriptions at reduced prices. Bureaucracy has its quirks, but is improving, and people are mostly helpful.

Austria is an El Dorado for outdoor fanatics. Top addresses for skiing, hiking, biking, climbing, swimming etc. are all within easy reach from the main cities (see Figure 1), and the best variety is found in the areas around Innsbruck and Salzburg. Vienna and Salzburg offer a large variety of theatre and music (from classical to jazz and pop) events throughout the year. Vienna is also famous for its museums and galleries, and Graz is a must for Jazz lovers.

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BSCB Spring Meeting 2000 – Cell Biology of Disease

Stuart Kellie, Ian Bird and Graham Craggs

This year's BSCB Spring Meeting was held at Warwick University on 28–31 March 200 and was centred on the theme 'Cell Biology in Disease'. Major sessions in the meeting were on angiogenesis and tumour progression; cytoskeleton in disease; cellular basis for inherited disease; cytokines in disease; neurodegeneration and wound healing. Due to the nature of the parallel sessions, all the sessions cannot be covered completely here and this report inevitably is biased towards the interests of the authors. So, apologies to those participants who have not been included in this review due to space pressures, and the inability of the authors to co-ordinate session attendance.

There were three plenary talks as well as the first BSCB Hooke Medal lecture: **Judah Folkman** (Boston) gave the opening talk in which he reviewed naturally occurring angiogenesis inhibitors. He gave a highly polished account of how tumour development was intimately associated with angiogenesis. Importantly, in order to generate energy mitochondria need to be within 100–200μm of oxygen (i.e. a capillary) and, in fast growing tumours only 2% of the cells are in cycle which is potentially a major problem with some anti-tumour therapies. There are many natural anti-endothelial fragments in the haemostatic system, and of these endostatin successfully inhibits tumour growth in mouse models, even after treatment is withdrawn. He feels that the controversy over the inability of other groups to show anti-angiogenic activity with endostatin can now be explained by deterioration of the samples during shipment, due to CO_2 from dry ice getting into the sample and significantly decreasing the pH. Therefore any acid-labile molecule it potentially at risk when shipped on dry ice without an adequate buffering system. Endostatin is now being tested in humans but it is too early for any results.

Bruce Edgar (Seattle) reviewed patterning and cell cycle in *Drosophila*. Cdc25 (stg) is a key enzyme in the cell cycle. There is evidence for nutritional control of the cell cycle and he produced a model of nutrition activating PI3K, then myc, then cyclin E leading to G to S transition. If stg and cyclin E are activated, the rate of cell growth is increased but the size of clones in the imaginal discs is unaffected. However activation of ras or myc increases clone size, but the doubling time is unchanged. Therefore there are independent controls for cell cycle transition and cell numbers.

Martin Raff (London) gave a typically thought-provoking lecture on control of numbers and timing in optic nerve development. Oligodendrocyte numbers are tightly controlled by migration and proliferation of precursors – in the developing optic nerve, 50% of oligodendrocytes die each day for four weeks. *In vitro* oligodendrocytes are dependent on survival factors from astrocytes, and *in vivo* oligodendrocyte proliferation is dependent on axons. Remarkably, oligodendrocyte proliferation and differentiation *in vitro* occur with an identical timescale to that found *in vivo*, and this is independent of the number of cell divisions. therefore the cells appear to be measuring time before differentiation. A factor involved in the time measurement of differentiation is thyroid hormone, which in turn regulates p27kip. Finally, he described culture conditions in which differentiation could be markedly altered. If oligodendrocyte precursors are cultured in FCS for three days, then switched to bFGF for 5 days, these cells go back to a more primitive state, then differentiate into neurones, i.e. have the characteristics of neural stem cells. This must have tremendous implications for neural repair after injury.

Anne Ridley was the recipient of the first BSCB Hooke Medal – given to acknowledge excellence in Cell Biology. She reviewed her personal history of how she ended up working in the field of rho and rac regulation of the cytoskeleton, and generously acknowledged the people who had influenced her. Her current interests are the control of HGF-mediated

cytoskeletal changes, and the role of rac and rho in macrophage movement and responses.

Julie Cherrington (Sugen) opened the session on *Angiogenesis and Metastasis in Cancer* with a talk on tyrosine kinase inhibitors in research and the clinic. Many positive regulators of autocrine and paracrine growth are receptor tyrosine kinases (RTKs) and RTK inhibitors could potentially inhibit both deregulated cell growth and metastasis seen in many cancers. Of around 100 RTKs, Sugen are mainly targeting receptors for PDGF, FGF and FLK. She reviewed research and clinical trials data on SU5416, a FLK selective inhibitor. This compound reduces vascularity, oedema, vascular leakage and tumour size in a variety of animal models. SU5416 is currently in phase III clinical trials in colorectal carcinoma and non-small cell lung carcinoma. **Bjorn Olsen** (Boston) talked about the roles of collagen XVIII/endostatin in angiogenesis. He detailed two methods for producing endostatin from collagen XVIII by proteolytic cleavage of the hinge region by matrix metalloproteinase (MMP) dependent and cysteine protease (cathepsin L) dependent processing. However, endostatin$^{-/-}$ mice have no phenotype., so there may be a compensating molecule.

Susan McDonnell (Dublin) described work on matrilysin (MMP-7). High levels of this protein are produced by many breast, prostate and GI tumours. It is secreted apically in early tumours, and both apically and basolaterally in later tumours. A model was presented of MMP-7 cleaving Fas ligand off normal gland cells thus allowing its interaction with Fas and resultant apoptosis of the cell. Development of resistance to apoptosis via this route is apparent in early tumours. **Dylan Edwards** (UEA) reviewed MMP inhibition as a cancer therapeutic strategy and gave a very good overview of progress (and problems) to date. Whilst being excellent targets, more work is required to identify specific MMP substrates themselves. The complexity of MMP interactions was also illustrated eg: membrane type MMPs are involved in processing other MMPs to active enzymes and there may be situations where MMP inhibition could be deleterious eg: prevention of endostatin production.

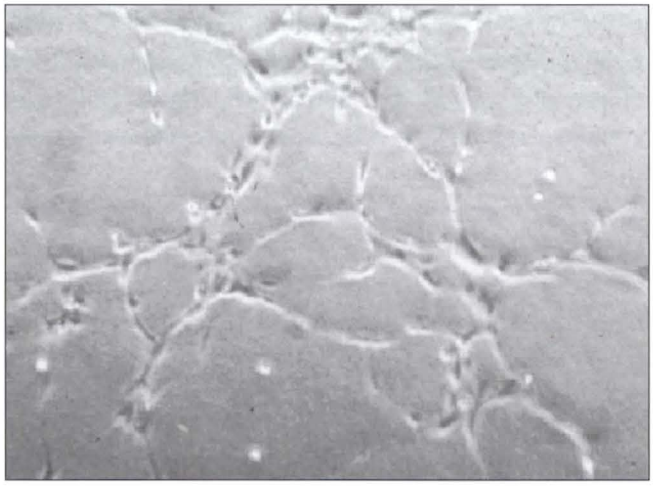

Figure 1: 'Angiogenesis *in vitro*'. Endothelial cells form capillary-like structures when cultured on a suitable extracellular matrix. This can be enhanced by VEGF. Figure courtesy of Ian Bird

Chris Higgins (London) opened the *Inherited Disease* session with a review of ABC-transporters. Approximately 40% of all tumours are resistant to chemotherapy, and this is likely to be due to the presence of multi drug resistance proteins, of which P-glycoprotein is the prototype. Recently multidrug resistance in bacteria has been shown to be due to homologues, and when these bacterial proteins are expressed in mammalian cells they confer drug resistance.

There then came two unexpectedly related talks. **Karl Tryggvason** (Stockholm) described a kidney disease – congenital kidney disease of the Finnish type – which is a genetic mutation resulting the leaking of plasma proteins through the glomerular filtration barrier. The gene responsible was cloned and the product (nephrin) found to be a protein of the Ig-like CAM family exclusively produced in podocytes and located in the glomerular slit region. Cell-surface nephrin appears to undergo homotypic adhesion and may be important for podocyte function. Nephrin$^{-/-}$ mice have congenital kidney disease, so this protein appears to be the major protein responsible for regulation of glomerular filtration.

Andrey Shaw (St. Louis) then talked about a protein originally cloned from T cells which unexpectedly associates with nephrin. Shaw searched for proteins which associate with the cytoplasmic domain of the T cell molecule CD2, and cloned a CD2-associating protein (CD2AP). This has SH3 domains and an actin binding

motif. CD2AP$^{-/-}$ mice develop normally for 3 weeks, then die from acute kidney failure. Histology showed defective glomeruli, and CD2AP was highly expressed in podocytes. CD2AP co-localises with nephrin and was shown to associate with nephrin in co-Ip experiments. It appears that CD2AP associates with the cytoplasmic tail of nephrin and regulates its function in the kidney.

Stephen Tucker (Oxford) wound up the session by describing ATP dependent potassium channels and their role in disease. K_{ATP} channels link metabolism to electrical activity, and importantly link blood sugar levels to insulin secretion. the channels are often occur as two subunits: SUR and KIR. In Finland (again!) there is a congenital disease of hypersecretion of insulin resulting in low plasma glucose levels. This syndrome has been traced to mutations in the *SUR1* gene and the *KIR6.2* gene.

A second *Inherited Disease* session started with a fascinating talk from **Jacques Bonaventure** on syndromic craniosynotoses. These are autosomal dominant conditions in which a major defect is the early fusion of cranial plates and the subsequent deformation of the skull. Mapping of chromosomal mutations have revealed the importance of the FGF receptors in several of these syndromes, particularly FGFR2 and recently the transcription factor Twist has also been implicated. Work is now focussing on whether Twist may regulate FGFR expression during development.

Steve Winder (Glasgow) revealed how his lab is uncovering roles for the utrophin–dystroglycan complex in cell adhesion and signalling and showed how binding of utrophin to this complex can be controlled by specific tyrosine phosphorylation events. Recently he has also shown a role for active MAPK in the formation of new cell adhesions and evidence that MAPK may be targetted to these sites by utrophin.

Two talks by **Christine Kinnon** and **Gareth Jones** in this session, and one from **Laura Machesky** in the *Cytoskeleton in Disease* session, focussed on the functions of the Wiskott–Aldrich Syndrome Protein (WASP). Wiskott–Aldrich Syndrome is a X-linked recessive disorder causing thrombocytopoenia and severe immunodeficiency in sufferers. Chris described how WAS patients displayed defective Fc-mediated phagocytosis and that cells were deficient in their ability to form phagocytic cups. Gareth described his lab's work studying the motility of macrophages from WAS patients. These macrophages don't form filopodia and are defective in their ability to chemotax in a gradient. He also demonstrated that podosome reassembly requires rac. Finally, Laura described the role of WASP in activating actin polymerisation via the Arp2/3 complex. She showed that Fc-mediated phagocytosis requires Rac and Cdc42 function (but not Rho), that Arp2/3 and actin normally co-localise in phagosomes and that delocalisation of Arp2/3 blocks phagocytosis.

Also in the cytoskeleton session, **Birgit Lane** gave an excellent overview on the known roles of intermediate filaments and how mutations in several of these have now been mapped and linked with specific skin disorders such as Epidermolysis Bullosa Simplex. Current work is focussing on possible roles of intermediate filaments in internal GI tract disorders.

In the *Neurodegeneration* session, we heard three talks about different aspects of neural function. **Brian Anderton** (London) reviewed the pathology of Alzheimer's Disease with respect to tangles or paired helical filament formation in the brain. These tangles consist mainly of the microtubule-associated protein tau. Recently a number of inherited senile dementias have been shown to be due to mutations in tau and phophospecific antibody staining has shown that tau in senile plaques is hyperphosphorylated. *In vitro* work has shown that the major tau kinase is GSK3. Many of the tau mutations in dementia map near the splice site of exon 10, preventing splicing out of this exon and leading to changes in phosphorylation.

The Alzheimer's theme was continued by **Willem Annaert** (Leuven) who discussed the generation of amyloid plaques deposited by aberrant amyloid precursor protein production and cleavage. Amyloid β production may be regulated by presenilins and presenilin 1 has been genetically linked to plaque formation. A presenilin1 (PS1)$^{-/-}$ mouse, caused a late embryonic lethal phenotype. However, neurones from these mice had a

decrease in proteolytic processing of amyloid β. **Norman Haughey** (NIH) gave the final talk in this session and summarised his work on calcium homeostasis and the role of presenilins. When PS1 mutants are overexpressed in PC12 cells there is a change in intracellular calcium levels and when the calcium buffering protein calbindin was co-expressed with mutant PS this was protective against amyloid-β-induced apoptosis.

Brian Foxwell (London) opened the *Cytokine Signalling in Disease* session with an overview of anti-TNF therapy for rheumatoid arthritis (RA). A study of 50,000 patients found that over 60% responded to anti-TNF treatment. IL10 is another possible anti-RA treatment; however, IL10 has a short half-life and affects B- and T-cell proliferation, resulting in side effects. The search is now on for IL10 mimetics. He then went onto describe how an adenoviral vector system could infect 100% of cell populations and be used as a vehicle to introduce IκBα into macrophages with much more 'endogenous' results compared with standard transfection techniques. He concluded with mention of IL4 signalling and described how IL4 treatment causes TNF release in humans but not in mice, highlighting the importance of not extrapolating mouse models to humans. This was followed by a heated debate with Luke O'Neill on the use of different cell lines and mouse models.

Melanie Welham (Bath) spoke about the role of PI3K in IL-3 signalling using the pro-B cell line BAF3, which is IL3-dependent. IL3 is an important regulator of mast cells, stimulating cell growth and further IL3 release. A dominant negative from of p85 (DNp85) decreased IL3-driven proliferation of BAF3 cells and increased apoptosis. **Graham Craggs** (Oxford) spoke about the nuclear import of SHP-1, a protein tyrosine phosphatase which plays important roles in cytokine and growth factor signalling. It is commonly believed that SHP-1 is a cytoplasmic protein, however using SHP-1-GFP it was shown that SHP-1 resided in the nucleus of non-haemopoietic cells. and a nuclear localisation signal was localised to the C-terminal domain of SHP-1. His results imply that nuclear import might be a mechanism by which SHP-1 function is regulated, and intimate that SHP-1 could be involved in the dephosphorylation of nuclear proteins.

Dan Lui (London) presented work on the role of FGF2 in breast cancer. FGF2 stimulates the growth of epithelial cells and inhibits cell migration and the induction of angiogenesis. A reduction of FGF2 is a recurrent feature of breast carcinomas, whereas other tumour type often show elevated FGF2 expression. Retroviral-mediated expression of FGF2 in MCF7 cells reduced colony formation in soft agar and induced a branching morphology. FGF2 may therefore represent a potential therapy for breast cancer, although its tumour promoting function in other cell types is a problem.

Luke O'Neill (Dublin) spoke about IL1 signalling in inflammation. IL1 increases the expression of many inflammatory mediators and approximately 90% of IL1 responsive genes are activated by NF-κB. A separate pathway involves the activation of p38. Using highly specific toxins which inactivate small GTPases, it was shown that p38 activation is dependent upon Rac1 and Ras. The session was brought to an end by **Peter Heinrich** (Aachen, Germany) who described the family of IL6 receptors which require the gp130 subunit, which mediates signalling. He challenged dogma by showing that unphosphorylated STAT proteins form heterodimers, indicating that dimerisation alone is not responsible for the nuclear translocation of STAT proteins.

The *Pathology of Cytokines* session kicked off with a talk from **Chris Evans** (Boston) on the use of gene therapy for rheumatoid arthritis (RA), and he summarised the preclinical studies necessary to get approval for clinical trials. The strategy was to express the Interleukin 1 receptor antagonist (IL1RA) in the patients' own cells to counteract the overabundance of IL1 found in RA joints. An adenovirus system allows expression for up to six weeks (in rabbits) and in animal models this blocked an IL1 challenge very well. The current clinical protocol is to take out patients synovial fibroblasts while they are undergoing joint replacement, transform them with IL1RA virus, then put the cells back into the joint days or weeks later. Phase 1 trials started in 1997 and so far nine patients have received treatment. Data is double-blind so the results are not yet known, although IL1RA expression has been confirmed in these patients.

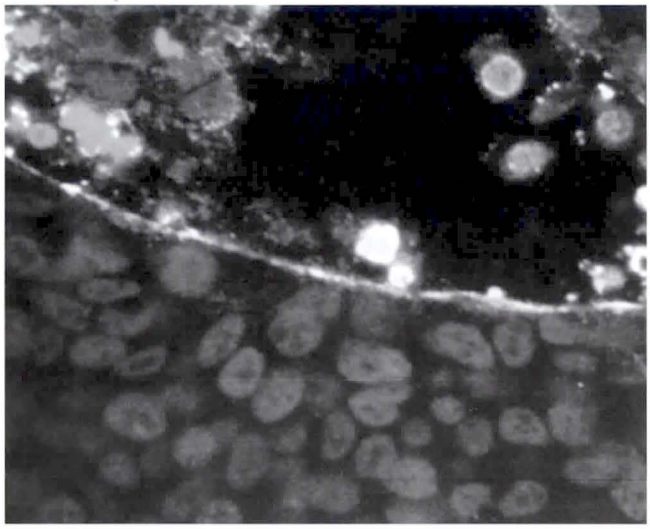

Figure 2. A contractile actinomyosin pursestring as it draws the epithelial margin forward over its mesenchymal wound substratum, as described by Paul Martin. Figure courtesy of Jane Brock and Paul Martin.

Paul Clapham (London) then described the role of chemokine receptors in the aetiology of HIV and AIDS. Along with CD4, the chemokine receptors CCR5 and CXCR4 are both co-receptors for HIV: CCR5 is preferred by macrophage-tropic viruses and CXCR4 preferred by T cell tropic viruses. The importance of CCR5 is underlined by the findings that 18% of Caucasians have mutations in this receptor which slows the disease progress. **Steve Ward** (Bath) summarised what is known about cyclo-oxygenases (COX) in Inflammatory Bowel Disease, and showed that TNF stimulates COX2, and this can be abrogated by IL4 or IL13. Thus enhanced COX2 in IBD leads to enhanced prostaglandins and other proinflammatory mediators. **Fran Balkwill** (London) reminded us that tumour cells account for only 50% of tumour mass, with invading cells accounting for the rest. TNF is found at high levels in ovarian cancers and $TNF^{-/-}$ mice are comparatively resistant to skin carcinogenesis. Thus in spite its name TNF is in fact a tumour promoter. Other results suggested that it may a be a master switch in tumour promotion signalling pathways.

Four talks on *Wound Healing* commenced with **Mark Ferguson** (Manchester) showing that wound healing in embryos is scar-free, and E16 is the last day on which this can occur. TGFβ is the major cytokine controlling scarring in adults, and if anti-TGF is given at the time of wounding, little or no scarring occurs. Anti-TGFβ is now in clinical trials for glaucoma, corneal healing, and are planned for skin and CNS healing. **Fiona Watt** (London) then described a transgenic mouse model where integrins are put under the control of the involucrin gene promoter to overexpress them specifically in the suprabasal layer. β1 transgene gives rise to psoriatic lesions due to hyperproliferation. **Sabine Werner** (Zurich) described KGF, a member of the FGF family strongly upregulated in fibroblasts after wounding. Inhibition of KGF receptor signalling in the epidermis of transgenic mice inhibits wound re-epithelialization. Genes regulated by KGF include all the key regulatory molecules in pyrimidine biosynthesis and she suggested that a new mechanism for growth factors may be the regulation of nucleotide biosynthesis. KGF also affects oxidation metabolism in cells.

Paul Martin (London) closed the meeting by describing the cell biology of embryonic wound healing. The cells make large actin cables with which they draw together like a purse string around the wound edges. This appears to be a Rho-mediated event since C3 transferase inhibited this. He then described other models of wound healing, such as *Drosophila* and zebrafish each with its own advantages for manipulation and examination.

Stuart Kellie, Ian Bird and Graham Craggs



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The Dynamics of the Cytoskeleton

Kathryn Ayscough and Vania Braga

Keystone, Colorado. 3–9 February 2000

This meeting, in the Keystone Symposia series gave a great opportunity to mix business and pleasure in the midst of the awesome scenery of the Rockies. The meeting was organised by Elaine Fuchs and Ron Vale and for added value ran concurrently with another Keystone Symposium 'Intercellular Signalling' with which there was a one day joint session. So, whether you were into skiing, skating, snowshoeing, clean mountain air or just plain science there was a great chance to mix and meet with a diverse group of people linked by a common theme of an interest in cells and the cytoskeleton. The diversity of subjects covered, and the relatively long duration of the meeting means that we cannot hope to cover all the speakers' talks in the detail they deserve. Rather we have chosen themes of particular interest to ourselves and areas in which significant and exciting recent advances were being reported and our apologies to all those we don't mention.

The opening talk of the meeting was given by **Marc Kirschner** with a very broad title of 'The Past , Present and Future'. Needless to say he didn't cover the entire history of the world, nor did he predict the future but gave some interesting insights into studies on cell morphology in which he had been involved, and how, with other research, such studies were contributing – not only to a mass of knowledge but to an increased understanding of what is turning out to be a very complex molecular system. One idea emanating from this talk and which then was reiterated throughout the meeting was the concept of 'self-organising' systems.

Microtubules and Kinesins

The first full day of the meeting was devoted to processes involving microtubules and commenced with an excellent talk from **Yixien Zheng** (Washington) describing the work from her lab on factors regulating microtubule dynamics and organisation In particular she presented some elegant reconstitution studies involving the γ-TUSC complex through which they have been trying to determine the role of γ-tubulin in nucleation of microtubules. Yixien also mentioned work on the nuclear GTPase Ran which has been demonstrated to play a role in microtubule aster and spindle formation. An activity which is required for Ran induced spindle formation is Eg5 which we heard more about from **Rebecca Heald** (Berkeley). The Heald lab has developed a system, using DNA coated magnetic beads. The DNA on the beads assembles into chromatin and induces spindle assembly in mitotic *Xenopus* extracts in the absence of centrosomes and kinetochores. This approach has led to the identification of cytosolic and chromatin associated factors including stathmin and Eg5 which are important in generating the microtubule organisation in spindles.

Paul Nurse (ICRF, London), one of only a few European speakers, discussed work from his lab on the role

Figure 1. *Schizosaccharomyces pombe* cells stained with TAT1 anti-tubulin antibody. Courtesy of Alison Pidoux.

of microtubules in cell morphogenesis using the fission yeast, *Schizosaccharomyces pombe* as a model organism. He described how major cell biological questions – such as determination of cell shape – can be addressed using simple, elegant, genetic approaches and in particular showed some beautiful pictures of microtubules in *S. pombe* and the co-localisation of several proteins which the lab has characterised as being critical for both microtubule organisation and also for normal cell morphology.

Moving onto cytoskeletal motor proteins, **Ron Vale** (San Francisco) compared the structures of kinesin and myosin. These proteins function through interaction with different cytoskeletal components (kinesin with microtubules and myosin with actin) but they have striking structural similarity though kinesin lacks a long lever arm. Using an array of methodologies such as EPR, FRET and cryoelectron microscopy, a model for the mechanism of action has now been elucidated for how a 'head over heels' motion will allow a dimer of kinesin to process along a microtubule.

Finally, **Larry Goldstein** (San Diego) outlined work in his lab on the genetic analysis of kinesins and their regulation in flies and mice. In particular, looking at kinesin dependent movement of cargoes down axons, several *Drosophila* mutants had been identified which caused a clogged axon phenotype and a number of the genes responsible for these identified. Amongst the mutants identified there was *Roadblock* (Dynein 1C), *Gridlock* (Arp1) and *Sunday driver* (a novel membrane protein which interacts with a kinesin light chain).

Actin: dynamics, regulation and roles in movement and development

Tom Pollard (San Diego) opened the session on actin dynamics with an excellent presentation on the recent work from his lab elucidating the role of individual proteins involved in activating and nucleating actin assembly. Pollard's lab have recently demonstrated the importance of WASP in activating assembly of actin filaments by the Arp2/3 complex and also have shown the importance of PAK-activated LIM-kinase in regulation of the actin filament depolymerising/severing protein actophorin (*Acanthamoeba* ADF/cofilin).

Actin dynamics in striated muscle sarcomeres had been thought to be minimal due to the presence of barbed (capZ) and pointed (tropomodulin) end capping proteins. **Velia Fowler** (San Diego) presented her lab's work in which they have observed the rapid incorporation of fluorescent actin monomers at filament ends thereby demonstrating both ends of the filament to be dynamic despite the persistence and uniformity of actin filament lengths. Velia also reported studies on the *SanPodo* mutant in the tropomodulin gene in *Drosophila*. These studies are helping to elucidate the role of tropomodulin during embryogenesis and clearly show the importance of regulating actin filament dynamics at the pointed, as well as the more widely studied barbed end of filaments.

David Drubin (Berkeley) gave the first of three talks in which yeast has been used as a model organism to further our understanding of the role of actin in various cell processes. David described work from his lab in which several proteins which were first identified in yeast genetic screens have now been studied in mammalian cells. One group of proteins including Abp1p, Sla2p and Rvs167p (an amphiphysin homologue) is postulated to link endocytosis with actin dynamics. Abp1p has been shown to be a potent activator of Arp2/3 in yeast and murine Abp1p co-localises with Arp2/3 in lamellipodia. He also described work on a family of kinases called the actin regulating kinases (ARKs). These proteins, also found across the evolutionary spectrum interact with Abp1 and phosphorylate a motif on several proteins thought to be involved in endocytosis.

Matthias Peter (Lausanne, Switzerland) described the role of Cdc42p in polarity establishment during bud emergence and mating in yeast and in particular recent studies in which they have shown how cells can recognise existing bud sites during vegetative growth and then ignore these sites when cells are exposed to mating pheromone. Critical interactions involve the shuttling of Cdc24p, the guanine nucleotide exchange factor for Cdc42p, in and out of the nucleus in a cell cycle dependent manner and the association of Cdc24p with an adaptor molecule Far1p which, during mating is required to relocate Cdc24p from the

nucleus to the Gβγ subunits of the ligand bound pheromone receptor.

Rong Li (Harvard) reported work on the interaction of the two unconventional class I myosins in yeast (Myo3 and Myo5) with Bee1p (the yeast WASP homologue). Using a permeabilised cell assay she has demonstrated the importance of these myosins in actin filament assembly and has shown that the proteins can also interact with the Arp2/3 complex. Other critical factors for actin assembly in this assay are verprolin (which is the homologue of the WASP interacting protein WIP) and Cdc42p.

In a later session at the meeting **Lynn Cooley** (Yale) talked about her work on actin dynamics and cell death during *Drosophila* oogenesis. Using a range of mutants in different actin associating proteins and looking at the localisation of specific proteins within discrete actin structures they have observed that Arp2/3 is required for the formation of ring canal F-actin, though the complex is not enriched in ring canals. Actin bundles which are observed just prior to the final burst of nurse cell cytoplasm transport, requires profilin, fascin and a villin-like protein quail, but not Arp2/3. Also using *Drosophila*, but this time looking at the actin structures in bristles, **Lew Tilney** (Pennsylvania) described his work on the role of actin filaments in bristle elongation. Two proteins appear to be critical for this process, forked initiates the bristle elongation process and facilitates subsequent crosslinking by fascin. He was able to show that inhibitors of actin assembly (eg latrunculin) curtail this elongation while a filament stabiliser (jasplakinolide) accelerates elongation.

Jim Spudich (Stanford) described work from his lab on a number of the myosins. From *in vivo* studies in *Dictyostelium* they have shown that mutants in which myosin II is deleted fail to divide in suspension but the phenotype can be rescued by a GFP-myosin which localises to the cleavage furrow and drives cytokinesis. Rather surprisingly, the myosin II mutant cells could divide when the cells are on a surface and a mutagenic screen was used to identify genes important in this process. This approach revealed cortexillin I as an essential component of the cytokinesis machinery in this situation but mutations in this gene could be suppressed by overexpression of coronin, RacE or dynacortin.

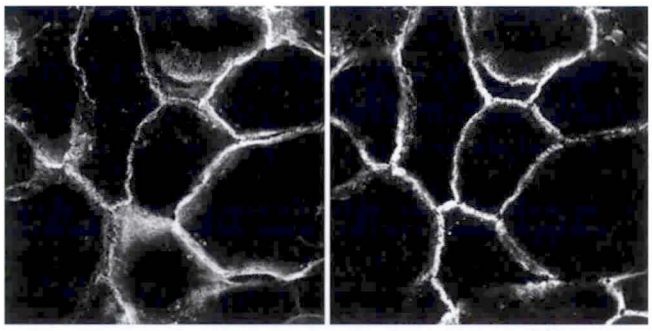

Figure 2. Normal human epidermal keratinocyte cells stained for actin (left) and cadherin (right). Courtesy of Vania Braga.

The Cytoskeleton, Adhesions and Cell Junctions

Elaine Fuchs (Chicago) spoke about an interesting protein, ACF7. This protein is homologous to the *Drosophila kakapo*, which has been shown genetically to play a role on epidermal-muscle attachment. ACF7 belong to the plakin family, but lacks an intermediate filament binding domain. Instead, ACF7 has a microtubule and an actin binding domain. It was proposed that ACF7 may cross linking the two cytoskeletal networks, microtubule and microfilaments. It is a huge protein of 600 kDa that localizes to cell–cell junctions, focal adhesions and as filamentous staining throughout the cytoplasm.

Barry Gumbiner (New York) addressed the question of how homophilic binding of cadherin receptors occurs. The cadherin extracellular domain contains 5 repeats (EC1 to EC5,) which bind to and are stabilized by calcium ions. Crystallography and peptide inhibition evidence in the literature suggest that receptors in opposing cells interact only via the first N-terminal domain (EC1) in an anti-parallel fashion. New evidence from his lab indicated that a construct containing domains EC1–EC2 together is not able to bind very efficiently, as opposed to the full length extracellular domain. Surprisingly, expression of domains EC3–EC4–EC5 mediated calcium-dependent aggregation, which was inhibited by an anti-cadherin antibody. These results suggested that the mechanism of

homophilic interaction should be revisited, as there may be considerable overlap of the cadherin extracellular domains when cell-cell adhesion takes place.

Crosstalk between signalling from adhesion receptors, growth factors and small GTPases was the subject of **Joan Brugge**'s talk (Boston). She spoke about the proto oncogene Vav, that can activate different members of the Rho family of small GTPases. There are 3 distinct members in the Vav family (vav1, vav2 and vav3), but not much is known on how their regulation and signalling differ. Vav activity appears to be regulated by phosphorylation, in a cell type-specific fashion . All 3 members are phosphorylated by treatment with EGF or PDGF and readily co-precipitate with the growth factor receptors. In normal macrophages, vav1 and vav2 are also phosphorylated upon attachment to fibronectin. However, in knockout mice macrophages (vav1$^{-/-}$), there is no defect in adhesion, spreading, migration or phagocytosis, suggesting a functional redundancy among different Vav family members.

Similar results were also presented by **Keith Burridge** (Chapel Hill) showing vav2 association with by EGF receptor and subsequent phosphorylation. Another interesting partner for vav2 was identified as the catenin p120. A possible link between these 2 proteins is revealed by overexpression of p120, which induced alterations in the actin cytoskeleton and activation of the small GTPases Rac and Cdc42. Although the biochemical interaction between vav2 and p120 was demonstrated to be reasonably specific, no functional significance for the association was presented. If there is a biological significance, it might be interesting to find out due to the involvement of vav during tumorigenesis and of p120 in the regulation of cadherin-dependent adhesion.

Overall the meeting brought together researchers working on disparate organisms but linked by a common theme of an interest in the cytoskeleton. The array of techniques described clearly illustrates the strength in diversity and the importance of using complementary methodologies to really begin to penetrate and understand such a complex system.

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The Myology 2000 Conference

Jane Ilsley

I would first like to thank the BSCB for my Honors Fell Travel award, which enabled me to go to the Myology 2000 conference.

Nice is a beautiful city with amazing architecture and it was even sunny most of the time. There were approximately 500 participants despite the clash with the BSCB conference in Warwick! The meeting was held in the impressive Acropolis conference centre in the centre of Nice.

There is a great deal of research in France into neuromuscular disorders, funded predominantly by the AFM (Association Francaise contre les Myopathies), which is the French equivalent of the British Muscular Dystrophy campaign. A large proportion of money for research is raised annually from the French public via a telethon. The scientific council of the AFM has two major objectives, the development of gene-based therapies and the emergence of Myology as a medical discipline in itself. The Myology 2000 conference was sponsored by the AFM. In the programme, there was a good mix of basic research and clinical research with a considerable emphasis on gene therapy and other therapeutics. Although this was an international conference, many of the talks and posters were in French, but at least for the former we were provided with translations.

In his introductory speech, **Michel Fardeau** (President of the Congress) enlightened us about the discipline of myology. The term Myology has recently been coined to encompass the biology of muscle tissue. However, we learnt that it has been studied since the 2nd century B.C! Fardeau named the greek physician, anatomist and physiologist Claudius Galen as the founder of Myology and his influence continued until the Renaissance. Artists during the Renaissance period worked illegally, dissecting cadavers in order to obtain detailed descriptions of muscles. In fact, Donatello was sent to the gallows for dissecting bodies.

It wasn't until the 17th century that the mechanical properties of muscle were thoroughly investigated with detailed studies of muscle contraction and microscopical studies. During the 18th and early 19th centuries, many chemists investigated metabolism during muscle contraction, for example oxygen consumption (Lavoisier), lactic acid production (Berzelius) and the discovery of ATP (Lehman).

In 1849, the physician Duchenne was the first to describe a muscular pathology and during the second half of the 19th century, many more muscular disorders were described. During the first half of the 20th century the progression of basic molecular biology contributed to the understanding of muscle contraction but the major turning point was in 1987 when the Duchenne muscular dystrophy (DMD) gene was cloned. This has accelerated research considerably and allowed mutation analysis of affected families, which has contributed significantly to the understanding of the disease pathogenesis of the muscular dystrophies.

The 4 full days of the conference were divided into four main themes: building up muscle tissue, structure and function of muscle; muscle in extreme situations, and muscle and therapeutic perspectives.

Margaret Buckingham (Institut Pasteur, Paris) presented a plenary talk on the formation of striated muscle. She explained how muscle specific proteins have been cloned thanks to muscle cell lines and the technique of subtractive hybridisation. She also described how looking to other species, for example, *Drosophila* and *C. elegans* and the zebra fish has helped our understanding of myogenesis. Often these species have a single gene homologous to a whole gene family in higher vertebrates. These model organisms are amenable to different experimental approaches, which permit the investigation of the pathways involved, which are often conserved over evolution.

Clinical symposium on cardiomyopathies.

Michel Komajda (Paris) described familial hypertrophic cardiomyo-pathy (FHC) which is characterised by unexplained ventricular hypertrophy which leads heart failure, arrhythmias and a high incidence of sudden death in teenagers with the disease. There are at least nine genes involved, including myosin heavy chain, cardiac Troponin T, and tropomyosin. The heterogeneity of the disease, environmental factors and the limited tools available for identifying carriers and a prevalence of healthy carriers are all factors that complicate diagnosis of the disease. The research also is hampered by stringent ethical regulations in France which make it difficult to obtain muscle biopsies of affected families.

Eloisa Arbustini (IRCC, Italy) described dilated cardiomyopathies (DCM) where the patient's heart cannot contract properly due to dysfunctional ventricles. She stated that it is a genetic disease even more difficult to assess than FHC, due to only 25% of cases being familial, poorly informative pathology and phenotypes looking alike despite the large spectrum of genes involved (e.g. mutations in mitochondrial genes, genes for the dystrophin-associated glycoproteins, intersarcomeric proteins (especially desmin) and nuclear envelope proteins). Overall, just 10% of familial DCM defects can be diagnosed using molecular analysis.

In a later symposium on cardiogenesis and cell therapy approaches for the reconstruction of cardiac muscle, **Philippe Menasche** (Bichat hospital, Paris) stated that heart surgery has its limits and there is room for cell therapy as a viable option in therapeutics of cardiopathies. Initial research focused on repopulating the lethally injured cells with foetal cardiomyocytes, however, problems of availability, immunogenicity and ethics can be overcome by using skeletal myoblasts instead which prove to be just as effective. Culturing the cells after biopsies and before transplantation is necessary, but their use has provided functional improvement of the heart in rats and rabbits. On this basis, primary clinical trials should be implemented to test the safety and feasibility of this method.

Four years ago, **Charles Auffray** (Genexpress, CNRS, France) proposed the term 'transcriptome' to designate the entire complement of gene transcripts which is characteristic of a given cell type, organ, organism or species. Since then, he has been working on the construction of cDNA libraries and expression profiling. He described the GenExpress Integrated Molecular Analysis of Gene Expression (IMAGE) in human skeletal muscle – the knowledge base of which is accessible on the web site: http://idefix.upr420.vjf.cnrs.fr.

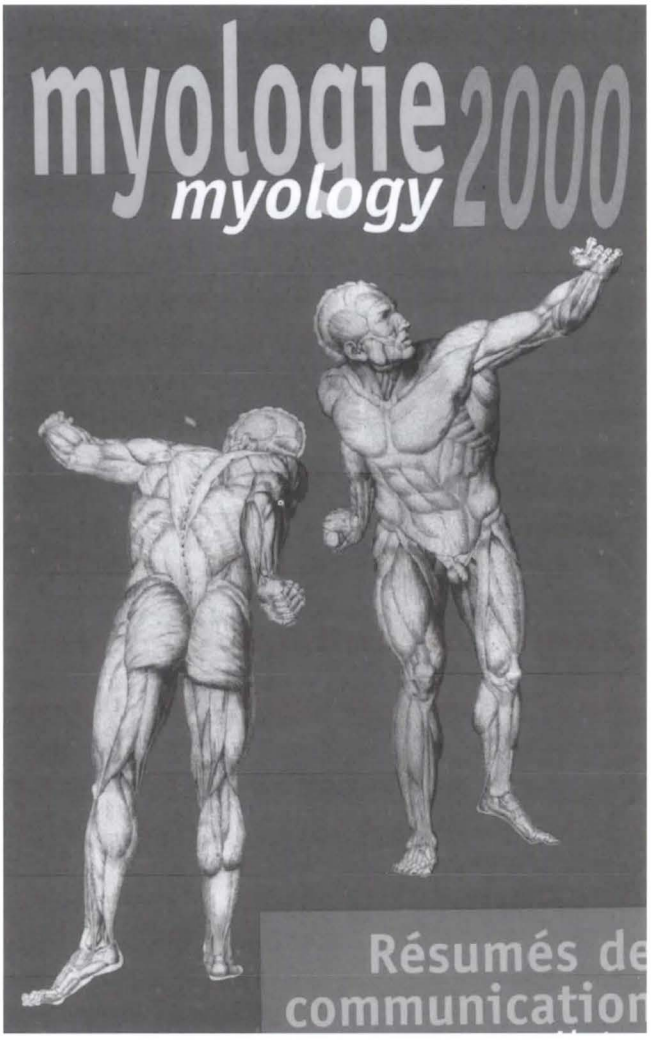
Richard Mulligan (Harvard Medical school) underlined the importance of characterising the phenotypic and functional properties of stem cells before using them in gene therapy studies. He is studying a novel class of stem cells derived from either bone marrow or other adult cells which can be isolated on the basis of a dual-wavelength FACS analysis of Hoechst 33343-stained cells. These were discovered quite by accident when the wavelengths on the machine were set-up incorrectly one day! His aim is to bypass the manipulation of cells *in vitro* and carry out the therapy *in vivo*. If highly purified stem cells can be used for transplantation, and only a small number are required, this would greatly facilitate any strategies requiring gene transfer.

To start the second day off, **Roger Craig** gave an excellent lecture describing the structure of sarcomeric

proteins and how this relates to their crucial function. **John Sparrow** from the York molecular motors group described the acto-myosin *in vitro* motility assays and optical trap assay, which he, J. Molloy and C. Veigek use to investigate actin-myosin interactions. They used the motility assay to look at actin mutants generated in *Drosophila* – used because they enable the extraction of sufficient quantities of actin for biochemical assays. *Drosophila* has six actin genes but ACT88F is an actin gene which is only expressed in flight muscle, thus allowing *Drosophila* mutants to be selected because they can't fly. Using this assay, they confirmed the presence of a secondary myosin-binding site on actin and showed that actin mutants outside the proposed myosin-binding site can affect *in vitro* motility, suggesting that dynamic changes in actin structure may be important for acto-myosin force development.

Kevin Campbell (University of Iowa) gave an all-encompassing lecture on the molecular organisation and function of the dystrophin-glycoprotein complex (DGC) in skeletal, cardiac and smooth muscle. He described how his group discovered sarcospan and confirmed that it is a member of the DGC because it co-purifies with the DGC and is absent in DMD patients and the *mdx* (dystrophin-deficient) mouse. He went on to describe the mouse models available for studying the DGC and their work on restoring α-sarcoglycan to the α-sarcoglycan knockout mouse. They use magnetic resonance imaging (MRI), which is a non-invasive tool which can assess muscle damage and progression of the disease. A gadolinium containing contrast reagent is injected into the muscle, and only damaged muscles take up the dye.

As part of the *muscle-ageing symposium*, **Eric Shoubridge** (Montreal Neurological Institute) discussed the segregation of mitochondrial DNA sequence variants in skeletal muscle in disease and aging. He showed that somatic mutations in mtDNA accumulate with age in skeletal muscle but fortunately seem to show no phenotype and are clonal expansions of deleted mtDNAs. So normal ageing and mtDNA disease (neuromuscular diseases) can be viewed as opposite ends of a spectrum of muscle respiratory chain dysfunction.



The lectures on the final day concentrated on therapeutic perspectives. Over the course of the conference, an ever re-occurring theme was how the use of transgenic mice has been crucial in research both for assessing the function of genes and the outcome of gene therapy studies. At the beginning of his talk, **Eric Hoffman** described the phenomenon of familial skewed X-inactivation, which was discovered in girls with Duchenne muscular dystrophy. Their X-activation is skewed so that the maternal X chromosome is selected against because it carries a lethal mutation and the paternal X chromosome with the DMD mutation is selected for. This event is characterised by recurrent spontaneous abortions in the affected families and a high female to male ratio due to the exclusive use of the paternal X-chromosome.

He continued on to explain that one of the long-term goals in DMD research is to identify the genes that dictate the progression of the disease by using expression profiling in patient muscles. He described the advantages of using 'Genechips' (25-mer oligos synthesised directly onto chips) over cDNA arrays: their high specificity (due to high redundancy (approximately 40 copies of each gene on a chip), more features per slide (300,000) compared to cDNA arrays, ease of inter-experiment comparisons, and all the results can be processed by informatics on computers. The main disadvantage is that cDNA arrays cost about $100 per slide and each Genechip set is $10,000! He said that many genes had increased or decreased expression in DMD muscle compared to healthy and they are in the process of characterising these and fitting them into pathological cascades.

Lee Sweeney (University of Pennsylvania) also described the successful use of AAV in γ-sarcoglycan deficient mice. The expression of γ-sarcoglycan is restricted to muscle due to a creatine kinase promoter in the AAV. Two months after injection of the virus, 20–40% of cells were expressing γ-sarcoglycan and this rose to 40–70% after four months. Therefore, AAV must be infecting satellite cells of the muscle. Consequently, no re-treatment was necessary and no immune response was detected. Sweeney also described studies his group has been carrying out using amino glycosides such as gentamycin, which can suppress premature stop codons in culture. They have shown for the first time that gentamycin works *in vivo* in *mdx* mice in which it allows expression of the DMD gene which has premature stop codons. One disadvantage is that there is a very narrow dosage range because too little doesn't work and too much is toxic. In *mdx* mice, 10% of normal dystrophin is enough to give protection against myopathy and allows relocalisation of the dystrophin-associated glycoproteins. Clinical trials are currently in progress to assess the use of gentamycin in humans.

This novel treatment could prove effective in 3–10% of DMD patients which have premature stop codons and also other diseases caused by this phenomenon. The amount of drug used will differ from patient to patient depending on the type of stop codon they have but the stop codon in the *mdx* mouse is one of the hardest ones for amino glycosides to suppress, so if gentimycin worked in the *mdx* mouse, there are high hopes for its success in humans. The drug will have to be administered daily but could be a good therapy to use while waiting for better therapies to be developed.

Kay Davies (University of Oxford) showed dramatic results of upregulating utrophin (the autosomal homologue of dystrophin) to rescue the DMD phenotype. Utrophin shares functional domains and protein-binding partners with dystrophin so it is hypothesised that it can replace dystrophin in DMD. Also, it is less likely to cause an immune response compared to replacing dystrophin because patients already have utrophin in their muscles. Delivery of utrophin to muscle using a recombinant adenovirus corrected the muscle pathology in a six week old dystrophin/utrophin null mouse. This therapy would require repeated injections of tetracycline to induce the utrophin gene and it is not clear whether the therapy would work in older patients (5–10 year olds).

Another method is to upregulate transcription of the endogeneous utrophin gene. Utrophin has two promoters which express the full length utrophin protein: A and B. Upregulating promoter B is a safer target because upregulating promoter A may disrupt neuromuscular junction localisation of utrophin and cause another disease. Currently, screens are underway to identify molecules that increase activity of promoter B in muscle and so far the results look encouraging with dramatic effects in *mdx* mice.

I would like to thank the organising committee of Myology 2000 and apologise to all the other excellent speakers that I couldn't mention in this brief summary of the conference.

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BSCB Autumn Meeting 2000 – Cell and Molecular Biology of Apoptosis

Heriot Watt University, Edinburgh. 10 - 13 September 2000

General Information

Dates

Arrive Sunday 10 September in time for dinner
Depart Wednesday 13 September after lunch.

Travel

The conference will be held entirely within the Edinburgh Conference Centre at Heriot Watt University. Further information about the site is available on http://www.ecc-hw.com. Full details about travel to Heriot Watt University and further instructions about the conference and site will be sent to registrants approximately 4 weeks before the conference.

Registration

The number of registrants is limited. In the event that the meeting is over-subscribed, priority will be given to those who present posters. The deadline for registration forms and abstracts is 28 July 2000; those registering after this date are subject to a strictly enforced late registration penalty of £35.

The accompanying registration form is also available at: http://www.bscb.org

Meeting Charges

The all-inclusive fees are as follows
Resident BSCB member, standard accommodation £320
Resident BSCB member, ensuite accommodation £350
Non-resident BSCB member £260
Non-BSCB member ADD £35

The fee for residents covers accommodation and all meals for the duration of the conference, The conference dinner will be limited to the first 120 registrants with the remainder dining at the conference venue. The fee for non-residents includes all meals for the duration of the conference including the conference dinner, but accommodation must be arranged independently.
Registrants who are not members of the BSCB can apply to join well in advance of the 28 July deadline to take advantage of the member price. Application forms to join BSCB are available on page 36 or can be found on the website http://www.bscb.org

Honor Fell Travel Awards for BSCB Members

PhD students and postdocs should remember that Honor Fell awards are available to cover conference costs in part. An Honor Fell application form should be submitted directly to the BSCB independently of registration (see page 38). Alternatively you can find an application form on the website: http://www.bscb.org

Abstracts and instructions for abstract submission:

Abstracts must be submitted electronically by email as text-only messages (no attachments) to: catherine.moorhouse@man.ac.uk

There will be an evening poster session but abstracts may be selected for oral presentation. Please indicate in the subject field of the email if you wish to be considered for a talk as outlined below.

The format should be as follows:
Subject field: surname-firstname / POSTERONLY or POSTER/TALK
Text field:
Top line – abstract title in capital letters
Next line – author names
Next line – affiliation
Leave one line blank

Type abstract in one paragraph; maximum 300 words.
Abstract deadline is **28 July 2000**

All conference information can also be found on: http://www.bscb.org

BRITISH SOCIETY FOR CELL BIOLOGY AUTUMN MEETING

EDINBURGH CONFERENCE CENTRE
HERIOT WATT UNIVERSITY
10TH-13TH SEPTEMBER 2000

Cell & molecular biology of apoptosis

Plenary speaker
Stanley Korsmeyer (USA)

The control centre for apoptotic regulation

Bcl-2 integrators
Richard Youle (USA)
Jean-Claude Martinou (CH)
Barbara Conradt (D)

Events at the mitochondria
Marcel Leist (D)
additional speaker

Mechanisms for feeding extracellular survival signals into the apoptotic control centre

Soluble factors
Alun Davies (UK)
Renato Baserga (USA)
additional speaker

Signal transduction
Caroline Dive(UK)
John Blenis (USA)
Christine Watson (UK)

Adhesion signals
Julian Downward(UK)
Caroline Damsky (USA)
Charles Streuli (UK)

Executing apoptosis

Upstream & downstream of caspases
Yuri Lazebnik (USA)
Gerry Cohen (UK)
Bill Earnshaw (UK)

Regulation of the apoptotic cascade
Jurg Tschopp (CH)
Marcus Peter (D)
Chris Gregory (UK)

Linking caspases to phagocytic clearing
David Ucker (USA)
John Savill (UK)

Further talks will be selected from Abstracts
See http://www.bscb.org for Meeting updates and further information

Cell and Molecular Biology of Apoptosis

Registration form

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

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

Registration, is being handled by The Biochemical Society, will not be processed without receipt of a cheque or money order in pounds sterling made payable to 'The Biochemical Society', or appropriate credit/debit card details.

Registration checklist:

- registration must be made in writing; fax copies will not be accepted.
- enclose registration form, indicating your name, phone number, fax number, e-mail address and dietary and parking requirements.
- either enclose a Sterling cheque for the relevant amount, payable to 'The Biochemical Society', or enclose credit card details.

Name .....

Title Prof / Dr / Mr / Ms

Address .....

.....

.....

.....

Telephone .....

Fax .....

E-mail .....

Please indicate details:

Resident, standard accommodation £320 ❑

Resident, ensuite accommodation £350 ❑

Non-resident £260 ❑

Non-member ADD £35 ❑

Late registration ADD £35 ❑

Vegetarian ❑

Other dietary needs ❑

please state...

Any other requirements ❑

please state...

Car park space required ❑

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Signature: Date:

This form should be sent to: The Meetings Office, The Biochemical Society, 59 Portland Place, London W1N 3AJ. (Tel: 02071580 3481)

Other forthcoming meetings

BSCB Spring Meeting
University of Sussex
3–6 April 2001

Polarity and Adhesion in Cell Function and Development

Organisers: Charles ffrench-Constant, Andy Furley, David Wilkinson, David Garrod, Alan Hall

Sessions will focus on: Junctions, Migration, Signalling, Morphogenesis and the Cytoskeleton

Invited speakers include: E. Fuchs. G. Borisy, M. Krasnow, F.Giancotti, and R. Hynes.

BSCB Autumn Meeting 2001
University College London
19–21 September 2001.
'Cell Biology and Neurobiology- a Meeting for Martin Raff'

BSDB / Biochemical Society Joint Symposium
Glycoconjugates in Development
University of Sussex,
18–21 December 2000.

Further information will appear in the next newsletter:
questions to Jamie.Davies@ed.ac.uk

Hannah Symposium 2000
Kevoca Conference Centre, Hannah Research Park , Ayr, Scotland.
6–8 September 2000

The Natural History of Breast Cancer

The Symposium will focus on critical non-genetic factors that influence susceptibility to breast cancer, together with their underpinning biological mechanisms. The programme will address, in terms of mammary development and function, the influence of critical stages in development, and the effects of diet and reproductive history on susceptibility to mammary neoplasia in later life.

Confirmed speakers include Adlercreutz (Helsinki), Andres (Berne), Clarke (Manchester), Cunha (San Francisco), Dickson (London), Ekbom (Uppsala), Hankinson (Boston), Haslam (Michigan), Hilakivi-Clarke (Georgetown), Holly (Bristol), Newcomb (Seattle), Peaker (Ayr), Svanborg (Lund), Trayhurn (Aberdeen), Wang (London).

Programme, abstract form and registration information available from: Symposium Office, Hannah Research Institute, Ayr, KA6 5HL, UK; tel: 01292-674000; fax: 01292-674003; e-mail: symp2000@hri.sari.ac.uk; website: www.hannah2000.abelgratis.co.uk/

Biochemical Society Meetings

Beyond the Genome
16–20 July 2000
18th International Congress of Biochemistry and Molecular Biology, Understanding and exploiting molecules and cells in the third millennium.

International Convention Centre, Birmingham, UK. Full details on http://www.iubmb2000.org
email: info@iubmb2000.org

Gene Action and Cellular Function in Parasitic Protozoa
13–15 July 2000
Chancellor's Conference Centre, Manchester University
Registration deadline: 2 June 2000
Poster abstracts deadline: 14 April 2000

Innate Immunity
21 July 2000
GlaxoWellcome, Stevenage, Hertfordshire, UK.
Registration deadline: 9 June 2000
Poster abstracts deadline: 14 April 2000

Fatty Acid Desaturases: Form Function and Future
30 Jul – 2 Aug 2000
51st Harden Conference, Wye College, Kent, UK,
Registration deadline: 2 June 2000
Poster abstracts deadline: 7 April 2000

Signalling in Plants
18–22 Sept 2000
52nd Harden Conference, Wye College, Kent, UK
Registration deadline: 29 May 2000
Poster abstracts deadline: 21 July 2000

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

MICROBIOLOGY TECHNIQUES
A two-day laboratory course
4–5 Sept 2000
Hatfield, Herts UK
Details and application forms from Dr Virginia Bugeja, Department of Biosciences, University of Hertfordshire, College Lane, Hatfield, Herts AL10 9AB UK.

tel: (01707)284590 fax: 286137 e-mail:V.Bugeja@herts.ac.uk
Website:
www.herts.ac.uk/natsci/STC

PROTEIN TECHNIQUES
A two-day laboratory course
4–5 Sept or 11–12 Sept 2000
Hatfield, Herts UK
Details and application forms from
Prof. John Walker,
Department of Biosciences,
University of Hertfordshire
College Lane, Hatfield, Herts AL10 9AB UK.
tel: (01707) 284546 fax: 284510 e-mail: J.M.Walker@herts.ac.uk
Website:
www.herts.ac.uk/natsci/STC

NUCLEIC ACID TECHNIQUES
A three-day laboratory course
6–8 or 13–15 September 2000
Hatfield, Herts UK
Details and application forms from
Dr Virginia Bugeja,
Department of Biosciences,
University of Hertfordshire
College Lane, Hatfield, Herts AL10 9AB UK.
tel: (01707)284590 fax: 286137 e-mail:V.Bugeja@herts.ac.uk
Website:
www.herts.ac.uk/natsci/STC

UNDERSTANDING THE USE OF MASS SPECTROMETRY IN PROTEOMICS
A one-day lecture workshop course.
In conjunction with Protein Works
20 September 2000
Hatfield, Herts UK
Details and application forms from
Mrs Vera Jones,
Science Training Centre, University of Hertfordshire
College Lane, Hatfield, Herts AL10 9AB UK.
tel:(01707)284590 fax: 286137 e-mail:v.g.jones@herts.ac.uk
Website:
www.herts.ac.uk/natsci/STC

BSDB Autumn Meeting 2000

Branching morphogenesis

University of York, 13th-15th September 2000

Branching morphogenesis occurs in prokaryotes, fungi, plants and animals, at scales varying from single cells through complex tissues and sometimes to the gross architecture of organisms themselves. Branching is now being studied intensively in a variety of important experimental systems, and for this reason the British Society for Developmental Biology is devoting a timely meeting to the subject.

Speakers Include:

Clare Greirson (Trichoblasts), Sarah Guthrie (Neurons), Tony Trinci (Filamentous fungi), Savério Bellusci (Lungs), Jocelyn Malmy (Roots), Christos Samokovlis (Insect Trachea), Ottoline Leyser (Shoots), Axel Thomson (Prostate glands), Thomas Berleth (Leaf venation), Jamie Davies (Kidneys), Jose De Celis (Insect wing venation), Jeffrey Rosen (Mammary glands) Volker Nehls (Blood vessels).

Please contact the meeting organisers for details and information on how to register:
Dr Jamie Davies, Edinburgh (Jamie.Davies@ed.ac.uk)
Dr Ottoline Leyser, University of York (hmol1@york.ac.uk)

Minutes of the BSCB Annual General Meeting

6 pm, 31 March 2000 University of Warwick

1. Minutes

The minutes of the 1999 AGM had been circulated to all members in the Society's Newsletter and were approved.

2. President's report

The incoming President, Fiona Watt, first thanked Ron Laskey, the retiring President on behalf of the Society for his work on the Society's behalf. She also thanked Birgit Lane for having acted for six years as Secretary to the Society. She congratulated Anne Ridley as the first BSCB Hooke Medal winner and reminded the membership that nominations for next year's award should be sent to the Secretary, Michael Whitaker.

Fiona was pleased to announce that the amount of money available for the Honor Fell Travel awards had been doubled, thanks to the generosity of the Company of Biologists. The aim was to double the maximum available for individual awards to encourage attendance at meetings outside the UK. She announced that Viki Allan was retiring from the BSCB Committee and thanked Viki for her contribution. Finally, she thanked the organisers of the Warwick meeting (Claire Isacke and Stuart Kellie) for having put together such an outstanding meeting.

3. Secretary's report

The incoming Secretary, Michael Whitaker, asked the meeting to confirm the membership of those listed who had joined the Society in the previous year. He announced the Executive Committee's nomination of Jonathan Pines, Jo Adams, Angus Lamond and Roy Quinlan as new members of the Committee. No nominations were received from the membership. The four nominees were elected to the Committee by the meeting. The Secretary also asked the meeting's permission to make a minor alteration to the Constitution, which had been made inconsistent by an amendment to the list of Officers of the Society at a previous AGM. Paragraph 7 of the Constitution was amended so that all the Officers mentioned in Paragraph 1 were also mentioned in Paragraph 7, which sets out the conditions of the Officers' tenure and re-election.

4. Treasurer's report

The Treasurer presented the Society's balance sheet, noting that last year's Spring meeting had made a significant loss. He added that this year's meeting was in positive financial balance. He announced an increase in the annual subscription to the Society to £25 (£10 for students), remarking that the subscription had last been increased in 1994. The meeting noted that the current Treasurer's term of office had formally ended, but agreed that the outgoing Treasurer would continue for a further year to provide overlap with the new Treasurer, yet to be appointed.

5. Meetings Secretary's report

In the Meetings Secretary's absence, The Secretary gave notice of the Societies future meetings. He reported the view of the Executive Committee that the proportion of international speakers at the Society's meetings should be limited to around one third of the programme, to allow UK speakers more prominence. The meeting agreed, emphasising the importance of choosing some speakers from among those submitting posters, as had happened at Warwick this year.

6. UKLSC/IoB consolidation

The Secretary reported that Government was increasingly insisting on the formation of a single organization with whom it could consult about Biosciences. The Institute of Biology had begun to take the role of representing UK bioscience on behalf of academic societies. However, it acknowledged that its own membership was not representative of the bioscience community. The UK Life Sciences Committee had been formed to represent academic life sciences. The two are now proposing to join with other large bioscience societies to develop a single voice for UK Bioscience. The Secretary would be representing the Society at a meeting to discuss details on April 7th and asked the meeting for its views. It was agreed, given that the University bioscience curriculum and teaching accreditation in bioscience were being discussed by Government, that some overarching body with whom consultation could be made was essential. However, it was also essential that efficient mechanisms for communicating and verifying policy were developed.

The British Society for Cell Biology

Trustees Report for the Year Ended 31 December 1999

The trustees have pleasure in presenting their report for the year ended 31 December 1999.

Trustees

Prof. R. Laskey (President)
Prof. E. B. Lane (Secretary)
Dr. S. Kellie (Treasurer)
Dr. C. Streuli (Meetings Secretary)
Dr. S. Winder (Membership Secretary)
Dr. K. Ayscough (Newsletter Editor)
Dr. V. Allan
Dr. L. Cramer
Prof. A. Hall
Dr. C. Hawes
Dr. S. Hughes
Dr. R. Insall
Dr. P. Shaw
Dr. M. Stewart
Prof. M. Whitaker
Dr. W. Earnshaw (appointed 15/4/99)
Dr. P. Luzio (appointed 15/4/99)
Dr. Theo Bloom (resigned 15/4/99)
Dr. C. Isacke (resigned 15/4/99)
Prof. N. La Thangue (resigned 14/4/99)

Contact Address

The contact address of the Society is c/o Margaret Clements, Dept. of Zoology, Downing St., Cambridge, CB2 3DY.

Status

The Society is a registered charity, number 265816.

Objects

The object of the Society is to promote the knowledge of cell biology.

Review of Activities

The financial results of the Society are set out on page 34. Reports on the Society's meetings and other activities are to be found in the six-monthly Newsletter.

S. Kellie
Trustee

Independent Examiners Report to the Trustees of the British Society for Cell Biology on the Financial Statements for the Year Ended 31 December 1999

I report on the accounts of the Society for the year ended 31 December 1999, which are set out page 34.

Respective responsibilities of trustees and examiner

As the charity's trustees you are responsible for the preparation of the accounts; you consider that the audit requirement of section 43(2) of the Charities Act 1993 does not apply. It is my responsibility to state, on the basis of procedures specifies in the General Directions given by the Charity Commissioners under section 43(7)(b) of the Act, whether particular matters have come to my attention.

Basis of the independent examiner's report

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

Independent examiner's statement

In connection with my examination, no matter has come to my attention:

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

   have not been met; or
2. to which, in my opinion, attention should be drawn in order to enable a proper understanding of the accounts to be reached

David Cooke MA (Oxon) FCA, David Cooke and Co. Chartered Accountants, 6 Seacourt Road, Botley, Oxford OX2 9LD. 28 February 2000

The British Society for Cell Biology

Statement of Financial Activities for the Year Ended 31 December 1999

	£	1999 £	£	1998 £
Income				
Subscriptions		20666		19267
Mailing list		1444		2026
Interest		2658		3528
Advertisements and fliers		1725		1590
Sponsored lectures		2000		2000
Capitation grant (Company of Biologists)		16245		15541
Meetings grant (Company of Biologists)		14000		14000
Meetings returns		8009		500
Other		111		273
		66948		58725
Less: expenses				
Direct Charitable:				
Meetings	42855		16713	
Newsletter & leaflet costs	4866		7927	
Sponsorship	250		250	
Honor Fell Travel Awards	12694		17608	
	60665		42498	
Administration and other expenses				
Secretarial	–		1036	
Committee travel and expenses	1363		1435	
Subscriptions	1250		1901	
Replacement computer	965		–	
Post, stationary, computer consumables	2850		2649	
Fax and phone	70		71	
Bank charges	234		336	
Accountancy and audit	317		294	
Miscellaneous	697		181	
	7746		7903	
Total expenses		68411		50401
Surplus/(Deficit) for year	**(1463)**		**8324**	

Balance sheet as at 31 December 1998

Current assets	***1999***	***1998***
National Savings Bank Investment Account	*49194*	*47082*
Abbey National Five Star Account	*16998*	*16451*
Midland Bank current Account	*8661*	*12760*
	74853	*76293*
Less: Current Liabilities		
Creditors and accruals	*317*	*294*
Net Assets	*74536*	*75999*
Financed by:		
Accumulated Fund brought forward	*75999*	*67675*
Surplus/(deficit)	*(1463)*	*8324*
	74536	*75999*

Approved: Dr. Stuart Kellie Trustee
Dr. Fiona Watt Trustee
28 February 2000

Treasurer's Report for 1999: Main Points

• We had an operating deficit of about £1400 this year, due mainly to reduced revenues and increased costs of the Spring Annual Meeting. Steps have been taken to help prevent this from happening again.

• Subscription revenues were slightly increased. A number of untraceable/old members have resigned, but these have been replaced with new members, the majority of which are students. Our membership now stands at 2006, of which 991 are paying by Direct Debit.

• Due to the loss made by the spring meeting, the amount spent on Honor Fell Travel Awards had to be reduced to £12694 (down from £17,600 in 1998), however in 2000 we hope to increase our HFTA budget to £30000 due to a generous donation from the Company of Biologists. This is detailed elsewhere in the Newsletter. I would like to thank Alan Hall for his work in administering the Travel Awards.

• Our major sources of income, other than subscriptions, was our Capitation Grant and Meeting Grant from the Company of Biologists, £162451 and £14000 respectively. As always, the Society is grateful to the COB for their continuing support for cell biology in the UK. We are also grateful to Yamanouchi Research Institute and Garland Publishing for their continued sponsorship of the Yamanouchi Lecture and the Borden Lecture at our Annual Meeting.

• Other major expenditures, were the six-monthly Newsletter at £4866, a replacement computer for Margaret Clements (£965) and subscriptions to ECBO, the Institute of Biologists, and the UK Life Sciences Committee, totalling about £1250.

New members from April 1999

Total 105

Abbott, Johanna K.R.
Anilkumar, Dr. N.
Appleton, K.M.A.
Atkinson, Susan J.
Baird, Fiona E.
Barrett, Dr. Kathy
Bayat, Ardeshir
Beare, Alice
Berika, Mohmed
Bulmer, Julie
Burns, Dr. S.
Cafferty, William B.J.
Callister, Deborah
Carrington, Louise
Collie, Marcus
Correa, Maria Laura
d'Adda di Fagagna, Fabrizio
Dafou, Dimitra
Dalvi, Nafisa
Dawe, Helen R.
Dodson, Helen
Draviam, Viji M.
East, Lucy
Edwards, Jill C.
El-Aleem, Seham A. Abd
Ellis, Sara
English, Jane L.
Evans, Richard
Fry, Dr. Andrew M.
Gedge, Lucinda
Gomes, Anita R.
Gourlay, Louise
Green, Kirsty A.
Grossman, Emily
Hansen, Soren Prag
Harbott, Lene K.
Hill, Dr. Alison J.M.
Hobbs, Robin M.
Ivanova, Dr. Anna
Jamieson, Annie K.
Jess, Thomas
Jones, Dr. Keith T.
Jordan, Nicola
Kaur, Jasber
Krasnapolski, Martin Alejandro
Kureishy, Nina
Lamond, Dr. A.I.
Laude, Alex
Lingiah, Gavin
Llewellyn, Dr. David
Long, Heather
Lopez, Carballido
Luckman, Dr. S.P.
Lucy, Professor Jack A.
Marron, Dr. Marie B.
Marston, Daniel J.
Martin, Anthony
Martin, Ina V.
McKeague, Anne L.
McLaughlin, Dr. Paul
McMullan, Rachel
Mellor, Dr. H.
Metcalfe, Dr. A.D.
Milton, Alasdair
Moore, Dr. Jonathan D.
Moore, William
Nikolic, Dr. Margareta
Owens, Dr. Dewi W.
Penter, Rebecca
Pidoux, Dr. Alison
Pigeon, Helena K.
Pirinen, Niina
Pople, Jennifer
Price, Jillian
Quintana, Juan H. de
Raftopoulou, Myrto
Renshaw, Derek
Reynolds, Jon P.
Robb, Allison
Roberts, Dr. G.T.
Rogers, Maria Florencia
Rowntree, Rebecca K.
Schell, Dr. Michael J.
Schmidt, Dr. Anja
Seabra, Professor Miguel
Sever, Dr. Richard
Stephens, Dr. David
Stoeber, Kai
Stone, Miranda
Taylor, Ruth R.
Tippins, Dr. Richard
Turner, Laura
Volioti, Georgia
Wafaei, Ahmad Najem
Wall, Steven J.
Warn, Alba
Watkin, Harriet
Watson, Richard
Watt, Stephen A.
West, Heloise
Whalley, Dr. Tim D.
Wroblewski, Lydia E.
Zago, Manola
Zhao, Dr. Min
Zimmermann, Dr. F.

Application to join the BSCB

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

Please note that student membership is valid for a maximum of 4 years, and will then automatically become a regular membership.

Please complete and return this form and the direct debit form (opposite) to: Margaret Clements, Department of Zoology, Downing Street, Cambridge, CB2 3EJ.

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

DIRECT Debit

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 Margaret Clements, Department of Zoology, Downing Street, Cambridge, CB2 3EJ.**

To The Manager, Bank/Building Society

.....

Address

.....

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..... Postcode .....

Originator's identification number 9 4 1 4 5 1

FOR BSCB USE ONLY
This is not part of the instruction to your bank/building society

5. Originator's reference number (for office use only) BRITSO ☐☐☐☐

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

2. Name of account holder

.....

3. Account number ☐☐☐☐☐☐☐☐

4. Sort code ☐☐–☐☐–☐☐

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

Signature .....

Date .....

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Please cancel any standing order payable to the British Society for Cell Biology WITH IMMEDIATE EFFECT.

Name of Bank/Building Society

Account Number ☐☐☐☐☐☐☐☐

Customer's Account Name

Branch Sort Code ☐☐–☐☐–☐☐

Signature .....

Date .....

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- You can cancel a Direct Debit at any time, by writing to your Bank or Building Society. Please also send a copy of the letter to the BSCB.

Honor Fell Travel Awards

Jointly funded by the BSCB and the Company of Biologists

Honor Fell Travel awards are made to provide financial support for Young BSCB members to attend meetings. Applications are considered for any meeting relevant to cell biology. The amount of the award depends on the location of the meeting. Awards will be up to £250 for UK meetings, up to £350 for European meetings and up to £450 for meetings in the rest of the world.

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.

Awards will be given 4 times a year in January, April, July and October. 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:

.....

.....

.....

.....

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: .....

British Society for Cell Biology

Committee Members 2000

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E-mail: ms@mrc-lmb.cam.ac.uk

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BSCB assistant

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Fax: 01223 353980.
E-mail: zoo-jeb01@lists.cam.ac.uk

Schools Liaison Officer

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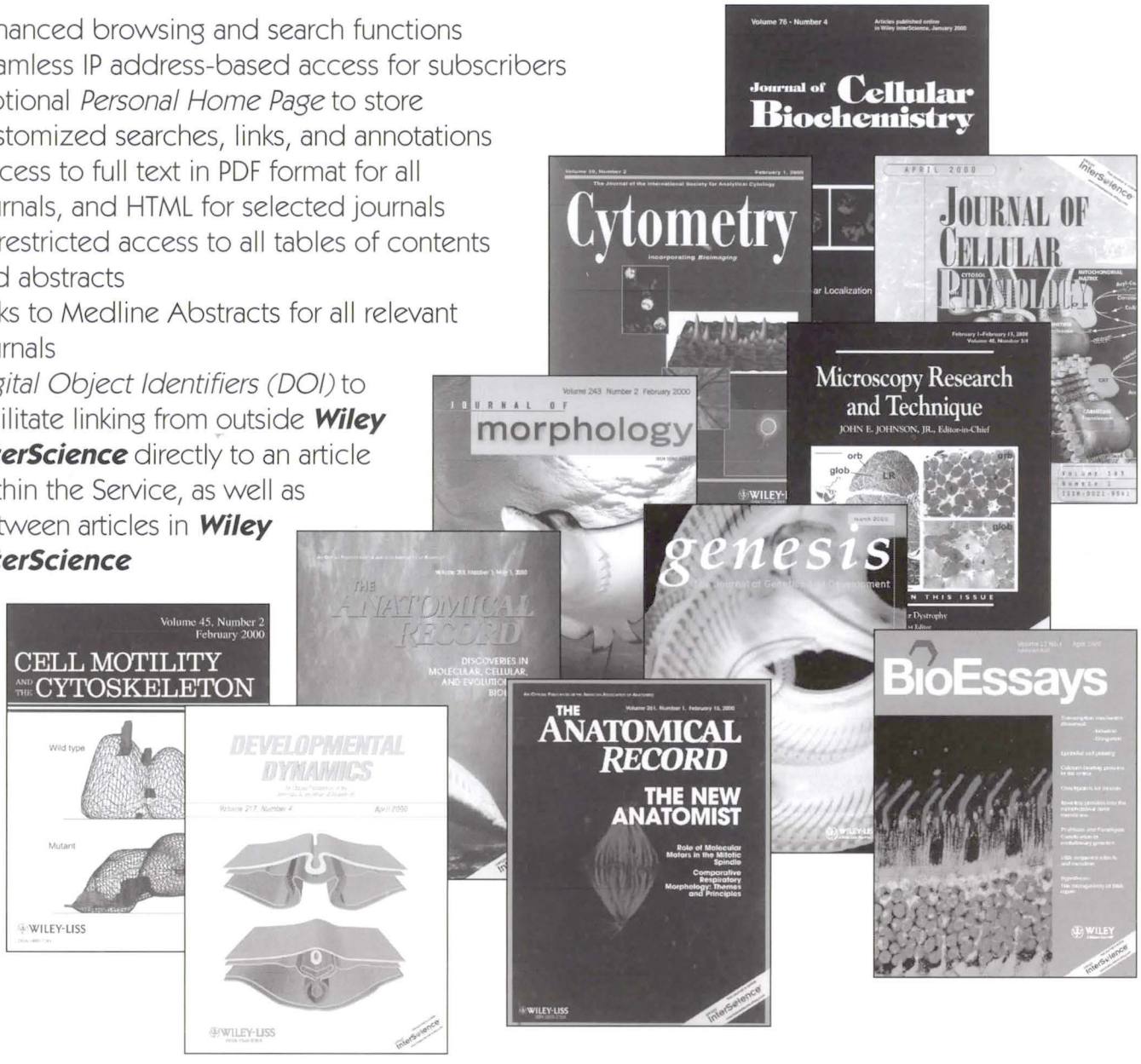

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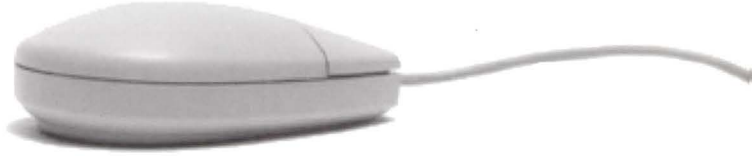

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