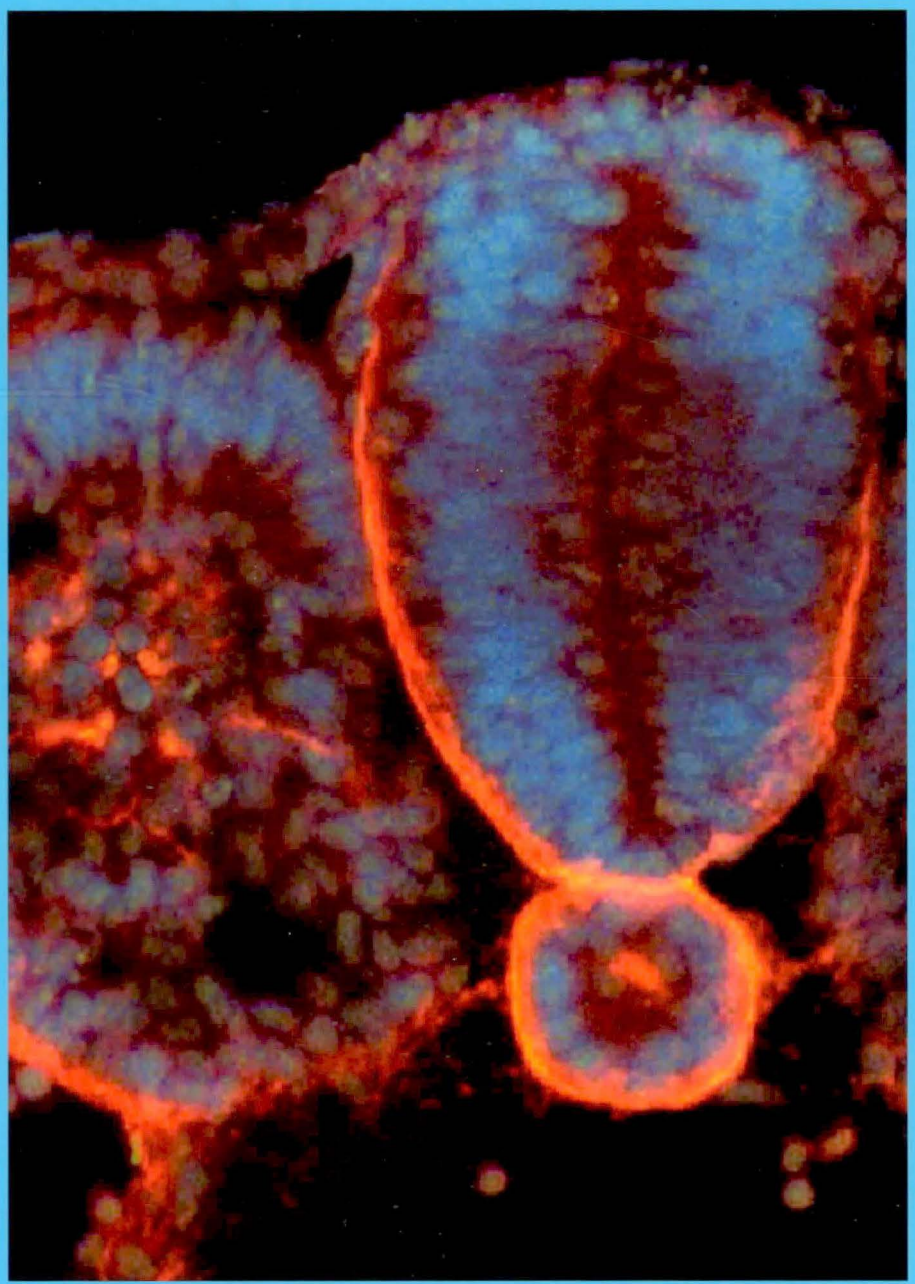
BSCB NEWSLETTER



BRITISH SOCIETY FOR CELL BIOLOGY

Winter 2000

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 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:
For the final version of articles and other materials and adverts, the deadline is 1 April for publication in June and 1 October for publication in December. Please note that the first version of any material must be received by the editor at least 2 weeks prior to this deadline so that any changes can be made.

Advertising Information
Single advertisement:
Back cover £600
Inside front cover Black and White: £275
Full inside page, black and white only £220
$^1/_2$ Inside page, black and white only £110
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Four advertisements, to cover two years. The costs are reduced by 30%.
Mailing List (Peel-off Labels) – £225.00 + p&p

Supply either on a floppy or zip disk for Macintosh (Quark version 4 or 3.32; EPS; or JPEG, TIFF or Photoshop at 300 dpi min) with margins: top 26mm, left/right/bottom 20mm. Page size 218x280mm. Alternatively, supply film: single/four colour positive, right reading, emulsion down, screen 133x150.

Please note, there is only one colour advert slot per newsletter.

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

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

New members should also complete an application form to join the BSCB (form 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.

Development	£256/171	(paper **only**);
	£74/49	(online **only**)
	£277/185	(paper **and** online)
Journal of Cell Science	£193/129	(paper **only**);
	£51/34	(online **only**)
	£213/142	(paper **and** online)
Journal of Experimental Biology	£192/128	(paper **only**);
	£51/34	(online **only**)
	£213/142	(paper **and** online)
Trends in Plant Science	£89/71	
Current Opinion in Plant Biology	£131/105	
Trends in Cell Biology	£89/71	
Current Opinion in Cell Biology	£131/105	
Current Biology	£105/84	

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

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

BSCB Newsletter
Winter 2000

Editorial

Welcome to all new BSCB members and I hope that you will enjoy this newsletter as well as being part of this growing society. Of particular importance at this time of year is when the programme for the next Spring meeting is announced. It looks to be a really excellent meeting down in Sussex with a central theme of Tissue Morphogenesis. The meeting will be held on the University of Sussex campus from 3–6th April 2001 and, in addition to the main programme which is running in conjunction with the BSDB, the BSCB is running two further sessions on '*Regulation of Cell Motility*' and '*Biogenesis of post-Golgi organelles*'. Information about the venue as well as the scientific programme is later in the Newsletter and, for the first time, registration and abstract submission can be made online.

We also continue our series covering Cell Biology in Europe – this time focussing on Portugal. I am expecting a brain drain from the UK to Portugal after reading Alvaro's description of life over there. If anyone from other European countries would like to contribute to this section please feel free to contact me at the email address given below.

This past year has also seen some excellent meetings and we have reports here from the BSCB Autumn meeting on Apoptosis and also from the Thrombospondin meeting held in Wisconsin.

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

Newsletter editor: Kathryn Ayscough
Design/layout: Giles Newton
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http://www.bscb.org

Front cover
Cross-section through the thorax of an embryonic chicken, stained for thrombospondin (in red) with DAPI counterstain for nuclei (blue). Thrombospondin is highest in matrix surrounding the neural tube and notochord (centre of image) and between cells within the forming somite (lefthand side). Courtesy of Richard Tucker, UC Davis.

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BSCB Hooke Medal

We are extremely pleased to announce that the BSCB has awarded its second Hooke Medal to Iain Hagan working in the University of Manchester. Iain will hopefully be able receive the medal at the Spring 2001 BSCB/BSDB joint meeting in Sussex.

Nominations for the next BSCB Hooke medal can be sent to Michael Whitaker, Secretary of the BSCB committee at any time. Nominations should include a brief resume of the nominee and a list of their recent relevant publications.

Iain Hagan: BSCB Hooke Medal Winner

The second Hooke Medal of the British Society of Cell Biology has been awarded to Iain Hagan for his work on mitosis in the fission yeast *Schizosaccharomyces pombe*. Iain has long been faithful to fission yeast, beginning with his PhD studies in 1984 under the guidance of Jerry Hyams and Paul Nurse. While a PhD student he cloned and characterised the fission yeast B-type cyclin, the product of the *cdc13* gene, and also provided further insights into the connection between cell size and cell division.

With this flying start in the cell cycle field, Iain could have chosen the safe option and gone to do a post-doc in the UK or the USA, but instead made the courageous decision to go to Japan to work with Professor Mitsuhiro Yanagida at Kyoto University in 1989. Although Professor Yanagida is one of the foremost researchers in the fission yeast field, it was still very unusual for a European to go to Japan for a post-doc, but this sense of adventure and doing the unexpected is a characteristic of Iain in person and in his research.

The four years Iain spent in Japan allowed him to experience a different culture, to learn Japanese, and to make very significant discoveries in how the mitotic spindle is formed and regulated by kinesin motor proteins. The two *Nature* papers that Iain published on the cut7 kinesin-related protein were two of the first to identify this important class of motor proteins, and helped to open up this exciting area of research on how the mitotic spindle assembles through the action of motor proteins.

This period in Iain's life cemented his interest in the mitosis, and, after returning as a Cancer Research Campaign Return Fellow to set up his lab at the University of Manchester in 1993, he has continued to work on how the spindle is assembled and chromosomes are segregated. In doing so, Iain has developed the techniques for imaging mitosis in fission yeast, helping to make it one of the organisms of choice for cell biologists. His work on the manner in which the spindle pole body signals to the cell cycle machinery through the plo1 kinase, has been particularly influential, but it is typical of Iain's breadth of interest that he has also made important contributions to our knowledge of the role of the actin cytoskeleton in fission yeast conjugation.

Lastly, I should personally add that Iain is a great colleague to discuss ideas with – often as not in the bar – and his long standing and fruitful collaborations with a number of other colleagues testifies to his interactive nature. This was also recognised by the Human Frontier Science Program who awarded him a 10th anniversary medal, along with nine other former Human Frontier Science Program Fellows, to mark his scientific achievements after finishing their fellowship, and his promotion of international collaboration.

In awarding Iain the Hooke Medal the BSCB has recognised a rising star in British and international cell biology.

Jon Pines
Cambridge

BSCB Committee News

Firstly a welcome onto the executive committee for Jo Adams (UCL) who, having being elected onto the committee after the AGM, has now agreed to take over the position of treasurer from Stuart Kellie (Yamanouchi, Oxford) who has served the committee as treasurer for over 6 years. Jo will officially take over in January 2001 and many, many thanks to Stuart for all the work that he has put in on behalf of the society.

If anyone out there feels that they would like to serve the community of cell biologists in the UK and would like to be considered for election onto the BSCB committee please feel free to indicate your interest to any of the Committee members before next year's AGM which will take place at the spring meeting in Sussex.

Web news

For those of you on the look out for useful websites for cell biology information, pictures and movies, there has been an amalgamation of several sites to generate a site with many useful resources for cell biologists both working in the field and those involved in teaching:

World wide web virtual library of cell biology: *http://vl.bwh.harvard.edu*

Also, to let everyone know that registration and abstract submission for this year's BSCB/BSDB Joint Spring meeting at the University of Sussex will be made online.

Details of the meeting are later in the newsletter – for those of you who want to go straight to the website to register, the address is:
www.meetings-secretariat.com

The site is also accessible through the BSCB's own webpage (*www.bscb.org*).

IMPORTANT! UKLSC Database

The BSCB is an active member of the UK Life Sciences Committee (UKLSC), the aim of which is to act as a single representative voice for the life science community as a whole, in matters of policy, education and public awareness of science.

The UKLSC is about to produce both text and web based directories of the members of its constituent societies.

These databases will be compiled from address information held by the BSCB on its membership database.

If your address details are wrong, have changed recently or you do not wish your details to be included in the UKLSC database, either text and/or web, please contact Margaret Clements at the address on page 32 of this newsletter.

Cool pics wanted

The Wellcome Trust Photo Library, as part of its continuing drive to acquire new and exiting images to add to its collection, is offering a prize of £100 for the best image at the Sussex meeting and £50 for the runner up. Any post-doc or PhD student as well as lab heads who have an image (could be a shot of a whole embryo or a stunning confocal or EM shot, or whatever) which is either aesthetically gorgeous or tells a cool scientific tale (or both) should bring it to Sussex for entry into the 'Cool Pics' competition.

You should provide both a print for display and the original (either the slide or digital file). It would be really good if you could also bring a brief description of the image content. Jenny Whiting from the Wellcome Trust Photo Library will be at the meeting and will judge the images. She will also be selecting images for the library from all entries submitted so bring as much as you can!

The Photo Library provides images to both the academic community and the media and any pictures used commercially will earn royalties. You do need to be the copyright holder of the images to contribute them to the library which unfortunately means no published images (similar ones are OK).

For more info, contact Jenny Whiting on j.whiting@wellcome.ac.uk or on 020 7611 8347.

Awards

TCS Biologicals Cell Biology Prize

TCS Biologicals is delighted to announce that, through the collaboration of the British Society for Cell Biology, we will be offering a prize at the next BSCB conference. The aim of this prize is to reward outstanding research presented at the Spring Meeting and is in addition to the long running BSCB Young Cell Biologist of the Year.

The winner of the TCS Biologicals Cell Biology Prize will be given the opportunity to present their poster at a key European meeting with all expenses covered by TCS Biologicals. It is hoped that this prize will not only assist the individual to promote their own work, but will also help to maintain a high profile for the quality British research currently being performed across the UK by presenting it to the wider scientific community.

Details of the prize and how to enter your work for consideration by the judging panel will be distributed prior to the Spring 2001 Meeting to be held at the University of Sussex. So good luck with your research and we look forward to reading your posters.

Bursaries for young scientists from central and eastern Europe

Six bursaries are available for young scientists from, and currently working in, Bulgaria, Commonwealth of Independent States, Czech Republic, Slovakia, Estonia, Hungary, Latvia, Lithuania, Poland, Romania, and the former states of Yugoslavia to attend the BSCB/BSDB Joint Spring Meeting at University of Sussex, 3–6 April, 2001.

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

Applications, in duplicate, including a brief CV and concise reasons for wishing to attend the meeting should be sent to the BSCB secretary:

Professor Michael Whitaker
Dept. Physiological Sciences
The Medical School
Framlington Place
Newcastle NE2 4HH

Applications should be received by 26 February, 2001.

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

Young Cell Biologist of the Year Poster Prize 2001

All research students are invited to enter the next poster competition at our Spring 2001 meeting to be held at the University of Sussex, 3–6 April, 2001.

The first prize is a trip to the USA to attend the ASCB 2001 meeting to be held in Washington DC on the 8–12 December, 2001 with an opportunity to present their winning poster. The poster will be judged by a panel of internationally renowned scientists based on scientific merit and presentation.

Honor Fell Travel Awards

Honor Fell Travel Awards are available to provide financial support for young BSCB members to attend scientific meetings and conferences. Applications are considered for any meetings relevant to cell biology, although the applicant must be presenting a poster or talk. For more details and an application form, see page 30.

School News

David Archer. BSCB Schools Liaison Officer

ASE Meeting 2001

The BSCB will be supporting a lecture at the Centenary Annual Meeting of The Association for Science Education to be held at the University of Surrey from 4–6 January 2001. The BSCB lecture will be presented at 2.00 pm on Friday 5 January in the Griffiths Lecture Theatre.

Paul Martin has kindly agreed to give a lecture entitled '*Wound Healing: Lessons from Embryos*'. Synopsis: Embryos heal wounds very rapidly and efficiently and without leaving a scar. Studying how they do this can tell us as much about the natural tissue movements of embryogenesis as well as suggesting ways in which we might make adult tissues repair more efficiently. We are using various animal models (chick, mouse, fish and fly) in order to dissect out the signals that activate wound repair mechanisms and to analyse the cell behaviours that underlie closure of a wound.

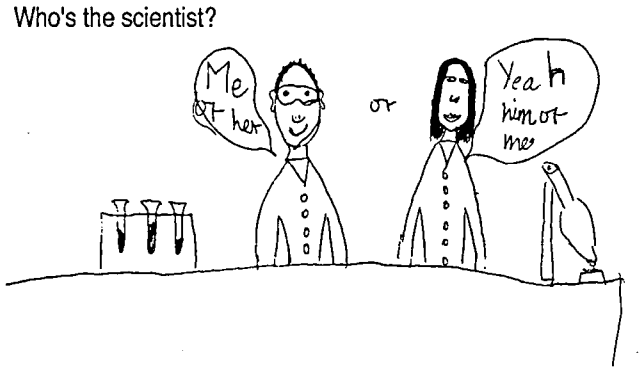
Notes from the school bag

Teachers attending the Association for Science Education Annual Meeting in January were told that studying biology at A-level had a dramatic input with regard to their attitude to genetic engineering with the students being much more supportive of genetic engineering than students taking other subjects. The study by John Raffen of the School of Education, University of Cambridge also found that more than 66% of male British students but only 30% of British female students supported transferring cancer genes to mice in order to study disease.

A Department for Education and Employment analysis of GCSE and A level results for 1997/98 indicated that high scoring GCSE students who chose to take science subjects were likely to obtain better grades than high scoring students who studied arts subjects. Interestingly the high-flyers who studied a combination of science and arts subjects at 'A' level obtained half a grade less in each A level than those high-flyers who studied either all sciences or all arts subjects.

Drawn to a male concept

Despite the age of 'girl power' – and I apologise if you dislike this term – it seems that most girls over the age of six associate the concept of 'scientist' with a male figure. This is one of several findings from research carried out by L and D Newton of the Department of Education, University of



In a quick and completely uncontrolled test one of the membership secretary's daughters was asked to draw a scientist. The result was two scientists, though interestingly the one with a microscope was female. Maybe a step in the right direction? Thanks to Tania for the picture.

Newcastle. In a 'Draw-a-Scientist' test they gave to 1143 pupils between ages of 4+ to 11+ from 80 schools in England, they found that up to the age of six girls drew scientists as predominately as female figures. After about the age of six, girls switched their concept to male figures.

Boys of all ages drew predominately male figures. By 11 years of age, over 80% of scientists drawn by both girls and boys were shown as male figures. As children became older, they increasingly gave their scientists beards and, by eleven, about one-third of boys and two-thirds of girls depicted their scientists as bald or balding. Over half the 11 year olds of both sexes drew their scientists wearing white coats and many wearing spectacles (hopefully safety specs!). Results from a 'Draw-a-Person' test with a sample size of 200 showed that boys draw predominately male figures and girls female figures.

Many pupils put backgrounds in their drawings. Seventy-five percent of these were 'chemical' in style, with more girls depicting chemical scenes than boys – who depicted ideas from space, force, energy and the Earth.

Various attempts have been made to change the image of science and scientists in older children. These have included visits to laboratories and scientists visiting schools. In general such activities are thought to have met with little success from the point of view of altering the concept of scientist.

In a study of teenagers and role models researchers Bromwick and Swallow at the University of Lincolnshire and Humberside found that 75% of boys in their sample and 52% of the girls admired famous males. Ten percent of the sample of boys held females in high standing. Interestingly the researchers noted that a significant number of both boys and girls in the sample refused to be associated with hero worship.

Where next, GMOs? Plant science research and public hostility

David E. Evans and Chris R. Hawes

There can be no doubt that plant science research has been revolutionised by the development of techniques for genetic manipulation. Hardly a single area of research remains in which previous knowledge has not been added to by the production and analysis of mutants, by over- or under expressing genes or simply by cloning, sequencing and comparing. Even taxonomy is receiving new direction as genome comparisons reveal surprising relationships between species. In the face of increasing pressure on Government funding, support from industry for both pure and applied research has been gratefully received by many laboratories. Through all this, plant science research in the UK has achieved a status and a volume that would only have been dreamed of even in the 1970s. Indeed, this has laid the foundations of a new industrial revolution, built not on factories contributing to rising global CO_2, the real pollution problem of the present day, but on GMOs – solar powered and non-polluting. Just as in the first industrial revolution, the UK science base has been at the forefront of this new revolution.

Into this scene have come the protests against GMOs, the scares, the scandals, the interpretation and misinterpretation, the premature press releases and the media hype. So far, protest has concentrated largely on commercial crops and on field trials rather than basic research. The key question for the plant science research community is: what impact will this have on our work? To answer such a question accurately requires foresight not available to these authors; however, it is possible to identify some key areas of concern.

The first concerns legislation. No gene cloning experiment can be conducted without stringent risk assessment and compliance according to EU and UK law. Various groups have been influential in establishing the regulations under which work is carried out – HSE, ACGM, ACRE – and in all this, the reasoned views of scientists have been considered. Risk assessments, based on the nature of the gene introduced and the nature of organism put the onus on the experimenter and the organisation within which they work to establish safety. Should this change and safety be assessed by factors other than soundly based risk-assessment procedures, the implications for plant science research will be very great. At worst, the costs of unneeded levels of containment and delays resulting from external bureaucracy would seriously hamper work. There is no good scientific reason at present to tighten legislation- but we should be pro-active in ensuring that reason in this prevails. There is one way in which researchers can force the rapid imposition of unneeded control; by failing to adhere to the ones we now have. We live in a time where one scare or scandal would be enough to bring about a costly change in the whole area.

The second concerns the risk of direct action. One aspect of the GM debate has been the destruction of field trials – trials designed to provide the hard evidence on which future legislation can be based. It has been rumoured that isolated incidents of damage to labs and offices have also occurred. It goes without saying that such direct action does a great deal of harm. If it persists it is likely to result in the redirection of research and development funding to countries where the 'climate' is less hostile. UK institutions are also less likely to be happy to support work in their own facilities if vandalism, violence and associated adverse publicity are likely to occur. Plant science laboratories have not traditionally considered security a priority. It is evident now that they should, perhaps especially in universities where open access is available. While colleagues would doubtless be sympathetic at the time, the real implications of the trashing of valuable experimental material or data from a research project for an individual worker are long term and serious.

The third concerns funding. Will the EU, UK research councils and industry continue to fund plant science research in the current climate? The political dimension of EU funding has always been evident. It is interesting to speculate the extent to which a shift in political will driven by adverse publicity has resulted in the reduction

in emphasis on plant science research between Framework IV and Framework V. Research councils are also not separate from political control, nor industrial sponsors immune from public opinion. While it is hard to see whether a shift in funding patterns has already taken place, it is almost inevitable that funding will be affected over the coming years – just at a time when massive opportunities are emerging. Here, it is very important that the UK plant science community fights to keep its funding for sound, innovative projects. It will be all to easy for others to build on the foundations already laid in part by UK scientists and to profit directly from it, while ensuring through patenting that we are excluded from its benefits.

Will controls and restrictions on our work increase, perhaps proving prohibitively costly to implement? Will direct action result in the destruction of experiments and data? Will there be a decline or redirection of funding away from plant science? The answers lie to a great extent in the tenacity with which the research community continues to defend and publicise the benefits of its work and respond to the new climate. It also requires courage on the part of those who control budgets in continuing to be willing to support the best applications and act with foresight rather than bend to short term changes in public perceptions. Burying our heads in the sand will achieve nothing; the world in which we work has changed in the last five years and it will be many years before it changes back. Meantime, we must be prepared to defend our work and to be pro-active, especially in reminding others of past achievements and the opportunities that lie ahead. Failure to do so will not prevent developments from taking place; rather it will mean that they will occur elsewhere – more slowly, in a less safe and appropriate environment – and many good opportunities will be lost. It will also hinder the opportunity to tackle real problems- industrial pollution, climate change, a secure and nutritious food supply and loss of biodiversity, to name just a few.

David E. Evans *Chris R. Hawes*

Research School of Biological and Molecular Sciences, Oxford Brookes University.

Cell biology in Europe – Portugal

Alvaro Tavares

Although Portugal is not traditionally known for its scientific tradition, Cell Biology has been, none the less, one of the more active areas of research. Entry into the European Community in 1986 was welcomed at all levels of society, and led to an explosion of new construction (roads, public buildings, institutes, etc), and the standards of living have risen enormously. Entry into the EU was especially important for scientific research, as it brought significant improvements in funding. With a higher budget, research centres could enhance the numbers of graduate students, the quality of supervision and offer first class formal education in the form of practical and lecture courses. There was a huge effort in crafting opportunities and a great number of the PhD fellowships attributed are for work in a foreign country (42%).

Figure 1.
In Portugal the largest research centres are located in Lisbon, Porto and Coimbra.

The amount of time required to finish graduate studies has also changed. New Portuguese PhDs were not competitive with their European peers because of traditionally long graduate studies. Things have changed drastically, and graduate studies are now being completed in fewer years (approaching an average of four to five years). The number of PhD dissertations in Portuguese universities has increased by 10% a year for the last ten years (depending on the area; biology, for example, had an increase of 16%). As a consequence, Portugal has one of the highest increases in the number of researchers in Europe: between 1992 and 1995 the increase was around 7% while the average of the EU was 1.9%. But it should be said that the number of researchers in the country is still relatively low compared to the other European countries (2.4% of the active population compared to 4.6% across the EU) so there is still some road to travel and plenty of opportunities for newcomers.

The money attributed to science has also been steadily increasing – now 0.6% of the PIB (although the EU average is 1.9%) – and there has been an increase in scientific collaboration with other countries over the last 20 years. In 1980, 28% of the papers were the result of collaborations; by 1996, this number had increased to 49%, the UK being the country with most scientific co-operation. The higher budget attributed to science attracted many of those who did their graduate or post-graduate studies abroad, and as a result many dynamic groups have emerged over the last decade. Furthermore, the Ministry of Science recently decided to strength molecular and cell biology research in Portugal by creating positions for investigators and to open new research institutes (most of which have not yet finished their recruiting). There is also now, for the first time, a situation where it is relatively easy to get a post-doc fellowship (and the candidate need not to be of Portuguese nationality).

Research and student life at Portuguese universities is very different from that at UK universities. In Portugal most research takes place at universities and in basic research institutions, with the drawback that most department and group heads have heavy teaching commitments.

Research sites in Lisbon

The Instituto Gulbenkian de Ciencia (IGC) (www.igc.gulbenkian.pt) was founded and is supported by the Fundação Calouste Gulbenkian (FCG) (www.gulbenkian.pt) to carry out biomedical research and education. The Institute's scientific interests are focused on the genetic basis of development and evolution of complex systems, prioritising organism-centred approaches in experimental models. A strong theoretical sector (Estudos Avançados de Oeiras, EAO) is also one of the Institute's specialities, the others being an investment on international exchange in the form of graduate courses, workshops and symposia.

In 1997, the FCG decided to restructure the Institute. One of the first measures was the creation of an international Scientific Advisory Board that includes Sydney Brenner, Nicole Le Douarin, Martin Raff, Kai Simons, Susumu Tonegawa, Hans Wigzell and Lewis Wolpert. The IGC uses the flexibility of its private statute to operate as a 'host institution', offering excellent facilities and services to foreign and Portuguese research groups or individual scientists, in particular to post-doctoral fellows who are expected to develop their projects and form their groups in complete autonomy. Among the recently formed groups at the IGC are those of Jose Antonio Belo (anterior neural induction of the vertebrate embryo), Isabel Palmeirim (somite formation and vertebrate segmentation), Jose Feijo (mechanisms of plant cell growth and morphogenesis), Alvaro Tavares (cell cycle regulation), Pedro Simas and Michael Parkhouse (virus modulation of the immune response), Helena Soares and Lisete Fernandes (stress and dynamics of microtubules), and Jocelyne Demengeot (mechanisms that rule the immune system ability to exert self / non-self discrimination).

There is an effort to make the IGC an international research Center, and foreigners at the Institute (students, post-docs and group leaders) account for about one-third of the total working people. The Institute was almost completely re-built from the inside in the last 4 years and is now fully equipped for modern cell biology research (including an up to date multi-photon confocal microscope and a cell sorter), and since 1993 it has offered an attractive Ph.D. programme which is open to students from all over the world. The IGC is still in its recruiting phase for group leaders.

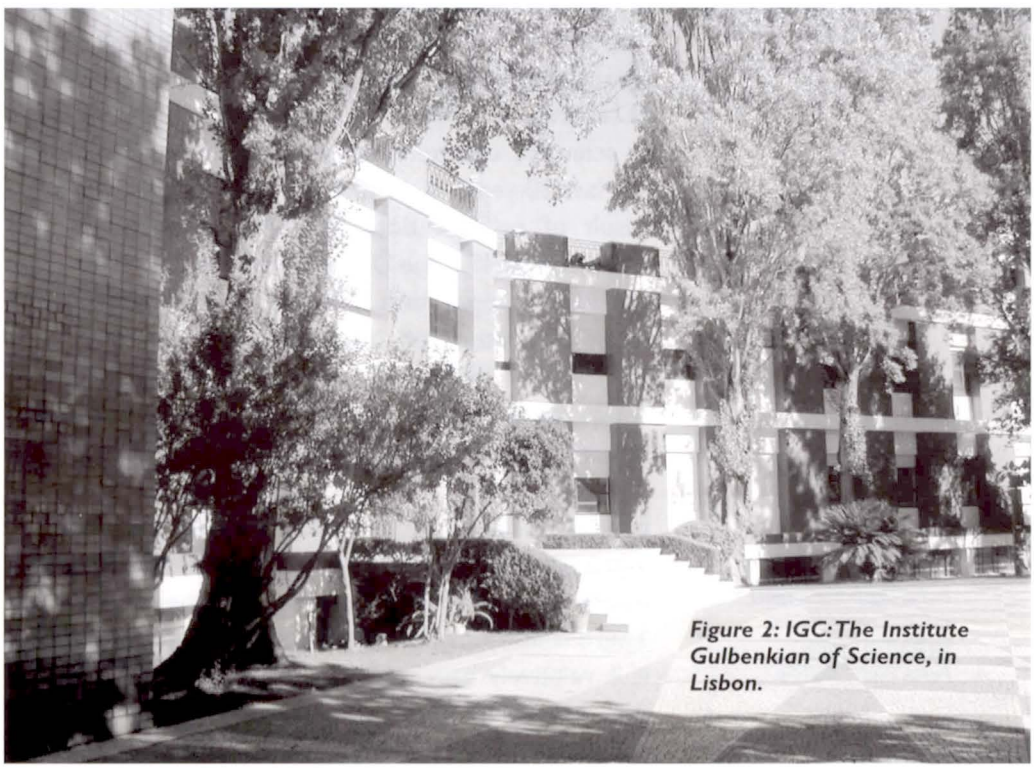


Figure 2: IGC: The Institute Gulbenkian of Science, in Lisbon.

The IGC is located in the campus of Oeiras, a campus that includes

other basic and applied research institutions in biology, biotechnology and chemistry (as the Institute for Chemical and Biological Technology, the Institute for Experimental Biology and Technology, and the National Agronomical Station) with complementary interests.

The Institute of Technology for Chemistry and Biology (ITQB; www.itqb.unl.pt) was established recently to facilitate scientific interactions between groups working on biotechnology and basic research. Although still growing, in these few years of existence it has succeeded in becoming a highly dynamic and high-profile Institute, with research activities covering a wide range of topics. The major groups in the Biology Division focus on: molecular epidemiology and the study of molecular mechanisms of antibiotic resistance (Herminia Lencastre), molecular mechanisms of morphogenesis (Paulo Tavares), regulation of transcription (Claudina Rodrigues-Pousada), mechanism and control of mRNA degradation (Cecilia Arraiano), plant biochemistry and morphogenesis (Jorge Almeida/Lizete Galego), role of oligosaccharides in the intracellular targeting of glycoproteins (Julia Costa), and the genetics and molecular biology of sporulation (Adriano Henriques). There is also a very strong group using non-invasive NMR techniques to study whole cell physiology (Antonio Xavier, Helena Santos). The ITQB has a true international atmosphere with 42% of the post-graduates coming from foreign countries.

Another Institute located at the Oeiras campus is the Institute for Experimental Biology and Technology (IBET; www.ibet.pt), the largest private-non-profit biotechnology research organisation in Portugal. It brings together public institutions and private companies as partners and collaborators. IBET's targeted economic areas are pharmaceuticals and health care, agroforestry, agroindustry and the environment. Areas of research at IBET are cell biology and biochemistry, microbiology, and molecular biology.

Also in Lisbon is the Centre for Molecular Biology and Pathology (CEBIP; www.fm.ul.pt), created in 1991 and located at the Lisbon Faculty of Medicine. CEBIP aims to foster research in the field of molecular and cell biology and its application to the study of human disease. At present, the research groups at CEBIP have scientific interests ranging from basic research on regulation of gene expression to clinical aspects of cellular immunology and cancer biology. As well as using common laboratory infrastructures, the researchers working at CEBIP benefit from sharing expertise in a variety of experimental methodologies specific to each group. The CEBIP harbours groups working on fields as diverse as molecular and cell biology, cellular immunology, skin biology and pathology, developmental biology, blood-cell differentiation and cancer biology.

The major scientific interest of the Molecular and Cell Biology group at the CEBIP (M. Carmo-Fonseca) is to understand how gene expression is regulated within the spatial and temporal environment provided by the living cell nucleus. The Developmental Biology group (Domingos Henrique) investigates the molecular and cellular mechanisms that originate the vertebrate nervous system. The Cellular Immunology group develops clinical immunology research projects in the areas of HIV immunopathogenesis and T cells in drug allergy. The Hematopoietic Biology group is interested on the mechanisms controlling the differentiation of blood cells (Leonor Parreira). The Cancer Biology and Experimental Therapy Group aims to study the cellular and molecular mechanisms involved in the acquisition of a selective advantage by human cancer.

Some groups working on Cell Biology can be found at the Faculty of Science, University of Lisbon (www.fc.ul.pt), for example in the Center for Genetics and Molecular Biology (Genetic Analysis of Cell Division in *Drosophila*; Rui Gomes) and in the Center for Cell Biology and Plant Biotechnology (Plant Cell Biology; Salomé Pais, José Barroso, and Rui Malho).

Research sites outside Lisbon

Created in 1290, the University of Coimbra (www.uc.pt) is one of the oldest in Europe. With a population of around 21 000 students and strong medical and biochemical tradition and facilities, it is an excellent place to study. The University is divided into

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several faculties, and research usually involves collaborations between different faculties. The Centre for Neuroscience and Cell Biology (CNC) was founded in 1990. Members of the staff of three faculties – Faculty of Science and Technology, Faculty of Medicine and Faculty of Pharmacy – form the scientific core of the CNC, which made it possible to integrate from the beginning some groups with a long-standing tradition in research.

The CNC is a research unit that covers various interdisciplinary areas, with cell biology as a dominant area of research. The Department of Cell Biology focuses on neurosecretion in isolated nerve terminals, cell death (apoptosis) and excitotoxicology (Arsélio Carvalho, Carlos Duarte, Celeste Lopes). The Department of Neurochemistry's main objectives include studies of neurodegeneration and molecular mechanisms of membrane fusion (Catarina Oliveira, Maria Pedroso Lima), while the Department of Molecular Biology (Euclides Pires) is concerned with the structure–function relationship of proteins.

In Porto, the Molecular and Cellular Biology Institute (IBMC; www.ibmc.up.pt) was created recently, and is one of the largest institutes dedicated to basic science in Portugal. Many groups were already working at other departments of the University of Porto and were transferred to the IBMC, bringing with them a lot of expertise to the benefit of the many young groups that were also created. The more international groups are interested in the molecular genetics and biogenesis of mitochondria (Arnaldo Videira), the molecular genetics of cell division (Claudio Sunkel), molecular structure (Ana Damas), molecular immunology (Maria de Sousa), microbiology and immunology of infection (Rui Appelberg), morphophysiology (D. Lima), and stress in micro-organisms (Pedro M. Ferreira).

There are still some small dispersed groups working in other institutes. For example, at the Institute of Molecular Pathology and Immunology (www.ipatimup.pt), Manuel Sobrinho-Simoes' group is looking at health sciences and health related issues with an emphasis on oncobiology. The Cellular Biology Center, University of Aveiro (www.bio.ua.pt), created in 1997 under the direction of Edgar Cruz e Silva, has interests in plant physiology, neurosciences, biochemistry, toxicology and microbiology, .

Living in Portugal

At the extreme southwest corner of Europe, Portugal has a population of just under 10 million and a pleasant climate all year round. Portugal is relatively free of crime and the people are generally very open and amicable, which makes it easy for foreigners to adapt to live in the country. Knowledge of the English language is common in the Portuguese population, especially among the younger generations, so most shopping can be done with no knowledge of the Portuguese language. Unlike most European countries, TV programs and films at the cinemas are not dubbed (meaning that the language is English most of the time). Living is relatively cheap in Portugal (food, clothing and transportation are cheaper than in Britain, for example).

Lisbon, the capital, has a rich cultural life and many national museums and art galleries. Although small compared to other European capitals, Lisbon boasts an outstanding cultural calendar. The city hosts both modern and traditional events from classical music, ballet and opera to street festivals, fairs and pop and rock concerts that are held all year round. The city also offers excellent late-night entertainment. Walking, cycling, riding and watersports (such as surfing, windsurfing, and sailing) are widely enjoyed and easily arranged. Winter sports are a little harder to come by.

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BSCB Autumn Meeting 2000 – The Cell and Molecular Biology of Apoptosis

Andrew Gilmore and Rosemary Gibson

This year's BSCB Autumn meeting was held in Heriot Watt University, just outside Edinburgh. This proved to be quite an appropriate location, being the city in which Andrew Wyllie first thought of falling leaves and coined the term apoptosis. It was a wonderful meeting, despite grey weather, an escalating petrol crisis and street protests by truck drivers, making delegates nervous about how they would get home! The organisers went to great lengths to entertain the delegates, including a ceilidh, dinner in Edinburgh, a midnight demonstration by the Edinburgh fire department (when the alarms went off in the halls of residence), and a by now world famous impersonation of the structure of laminin.

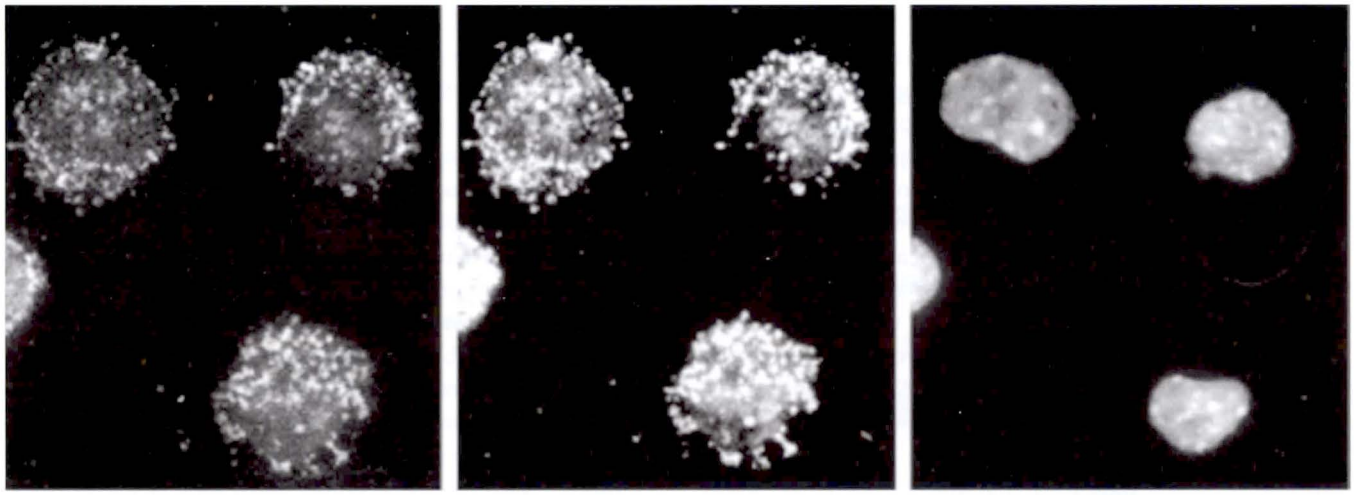
The science covered all aspects of apoptosis, from the layers of regulation in different types of cells through to phagocytosis of the apoptotic debris. Necessarily, we have not been able to report on every talk; apologies to those not included but great thanks to the scientific organisers, Tony Metcalfe, Charles Streuli and Bill Earnshaw for putting together such a great meeting.

Stanley Korsmeyer (Harvard) set the scene for the forthcoming exciting days with his keynote presentation. His talk on integrating the cell death pathway concentrated on how diverse survival signalling pathways converge to regulate Bcl-2 family proteins, the 'gate-keepers' of apoptosis. This family of related molecules ultimately control mitochondrial dysfunction, a key theme for much that followed, and subsequent activation of caspase proteases. One family member, Bad, is phosphorylated on at least three different serine residues. Phosphorylation of Bad promotes cell survival, and different signalling pathways may target each site. That all roads lead to the mitochondria was emphasised by work he presented on Bid. Bid is cleaved by caspase 8 during FAS/TNFR mediated apoptosis, and subsequently translocates from the cytosol to mitochondria. FAS induces caspase 8 activation directly, but in Bid knockout mice it does not effectively induce apoptosis, indicating a mitochondrial requirement. Bid appears to work at the mitochondrial surface by inducing oligomerization of other Bcl-2 family proteins, which then form pores to release cytochrome c, a process independent of the mitochondrial permeability transition pore. Bax, another member of the Bcl-2 family, progressively forms dimers and then tetramers which are capable of forming pores large enough for the passage of cytochrome c.

The following morning, the theme of Bcl-2 family genes continued with **Barbara Conradt** (Munich) presenting data on Egl-1, a Bad homologue in *C. elegans* which is expressed only in those cell lineages destined to undergo apoptosis. Mutations in the *egl-1* promoter that lead to its expression in other lineages results in ectopic cell death. The somatic sex determining gene Tra-1 regulates *egl-1*, resulting in expression that controls sex specific patterns of apoptosis.

Richard Youle (Bethesda) pursued the role of Bax and its subcellular localization. He described the translocation of Bax from a cytosolic, soluble distribution in healthy cells, to an insoluble mitochondrial one following an apoptotic insult. His use of monoclonal antibodies to specific epitopes within the N-terminus of Bax has demonstrated that this translocation is accompanied by a conformational change. He presented the 3D solution structure of Bax and showed it has a very similar structure to the two other members of the family which have been determined, Bcl-X and Bik. The Bax structure however, has been solved for the full-length molecule, including the hydrophobic C-terminal tail and shows that the tail fits into a groove on the surface, therefore masking hydrophobic residues and allowing Bax to be soluble. This groove is homologous to the groove on Bcl-X previously shown to bind the Bak BH3 domain.

Jean-Claude Martinou (Geneva) presented data showing that Bax induces cytochrome c release from isolated mitochondria without mitochondrial swelling, laying down the argument again that the permeability transition pore (PTP) is not involved. Moreover, he has not observed an interaction between Bax and any of the PTP components. The mechanism by which Bax is translocated to the mitochondria appears to require Bid,



Mitotracker **Bax** **Nuclei**

Figure 1: Bax localizes to mitochondria following an apoptotic insult. Mammary epithelial cell detached from ECM show strong co-localization of Bax with mitotracker prior to nuclear condensation. Figure courtesy of Andrew Gilmore and Charles Streuli.

a close relative of Bad. The caspase 8 cleavage of Bid discussed by Korsmeyer appears to allow Bid to transiently interact with Bax, an interaction which induced Bax to associate with mitochondria. Martinou reported that Bax oligomerizes on the mitochondria and that this appeared to require a factor present on the mitochondrial surface, and was therefore an event independent of the initial translocation of Bax. Thus activation and pore formation occur in a complex and multi-step process.

Conformational changes are a common feature of the activation of Bcl-2 family proteins, and **Caroline Dive** (Manchester) presented data that the Bax homologue, Bak, undergoes similar modifications following a number of diverse apoptotic insults. Use of a panel of monoclonal antibodies indicated that Bak undergoes a two step activation. Initially the N-terminus becomes exposed, and this precedes morphological changes associated with apoptosis. However, a second site within Bak, the BH1 domain becomes exposed later, concomitant with morphological changes within the cell. This echoed the multistage activation of Bax discussed by Martinou.

A number of speakers dealt with the plethora of survival signals and the mechanism by which these regulate apoptosis. Many diverse survival signals appear to feed into the very same parts of the apoptosis machinery, the Bcl-2 proteins. **Renato Baserga** (Philadelphia) talked about the role of IGF-1 signalling in haematopoietic cells. As well as its role as a mitogenic signal, IGF-1 also acts as a potent survival signal in many cells. IGF-1 induced cell survival through regulating the phosphorylation of Bad. This occurred through a mechanism independent of IRS-1 and the mitogenic role of IGF, and did not occur following insulin receptor activation. It appears that the survival signal initiated by IGF-1 activates MAP kinase, and requires the translocation of Raf to mitochondria, an event without which the survival effect of IGF-1 was not seen. **John Blenis** (Harvard) showed that the signals from IL-3 in haematopoietic cells also regulate survival through Bad. He has been looking at the kinase Rsk-1, a downstream target of the MEK-MAP kinase pathway activated by IL-3. Dominant negative MEK blocked IL-3 survival signals, and constitutively activated Rsk-1 restored survival. This appeared to be through the phosphorylation of Bad on serine 112 by Rsk-1. Bad can also be phosphorylated on serine 136 by the kinase Akt. Phosphorylation on both sites was found to provide more potent protection from apoptosis than phosphorylation on either alone. Furthermore, phosphorylation on these sites appeared to promote Bad phosphorylation on serine 155, providing even stronger inhibition of its pro-apoptotic function.

Charles Streuli (Manchester) had also examined Bad phosphorylation, this time in mammary epithelia where both growth factors and the extracellular matrix (ECM) provide independent survival signals. He presented data demonstrating that Bad became dephosphorylated in the mammary gland following weaning, just prior to the massive increase in apoptosis associated with the involution of the gland. A number of growth factors regulated Bad phosphorylation in mammary epithelial cells,

including EGF and IGF. IGF has a potential role in regulating involution, and **Christine Watson** (Cambridge) showed up-regulation of IGF-BP5, a negative regulator of IGF signalling, at the start of involution, and a role for STAT transcription factors. ECM appears to control epithelial cell survival through a different mechanism, and adhesion did not regulate Bad phosphorylation. Charles Streuli showed that this occurs via focal adhesion kinase (FAK) and PI3-kinase. In the absence of ECM and FAK activation, Bax rapidly redistributes to the mitochondria where it induces apoptosis, and cells from Bax knockout mice were found to survive for longer in suspension.

The ECM connection was pursued in two further presentations, which indicated that the signalling pathways used are very different depending upon cell type. **Caroline Damsky** (San Francisco) has looked at synovial fibroblasts. Like epithelial cells, FAK was the primary signalling molecule, but unlike them it appeared to work through JNK and not PI3-kinase. She presented data showing that phosphorylated JNK was found in the focal adhesions of cells, but not if FAK signalling was blocked with a dominant negative construct. In contrast, growth factor induced survival did work through PI3-kinase. This indicated that not only did different pathways work in one cell in response to different signals, but that the same signals could work through different pathways in different cell types.

Julian Downward (ICRF) was interested in how oncogenes regulate a cell's susceptibility to apoptosis. Ras can have a role in both protecting cells from apoptosis, and also sensitising them, activating multiple downstream pathways. Ras transformation of MDCK cells made them insensitive to detachment induced apoptosis. Detachment of cells from ECM appeared to activate the death receptor pathway, resulting in caspase 8 activation and Bax translocation to mitochondria. The kinase Akt appeared to be in the pathway, and activated constructs protected from death. Ras activation of Akt may protect cells from apoptosis through transcriptional regulation, via the forkhead transcription factor. He is currently using gene chips to investigate which genes may be regulated following Akt activation.

Analysis of the influence of extracellular signals on cellular survival was continued by **Alun Davies** (St Andrews), who has examined the role of NFkB in growth factor-mediated survival of primary trigeminal and nodose neurones. His group has used two approaches: over-expression of p50 or p65 NFκB subunits and inhibition using a 'super IκB'. Microinjection of p50/p65 expression plasmids enhanced survival of trigeminal neurones in the absence of NGF and IκB negated this effect.

Aviva Tolkovsky (Cambridge), **Marcel Leist** (Konstanz) and **Jane Roskams** (Vancouver) addressed diverse issues relating to the mechanisms of neuronal cell death. Tolkovsky works with NGF-dependent sympathetic neurones, which die by apoptosis when NGF is withdrawn. Commitment to death can be delayed by caspase inhibitors, although the cells still release cytochrome c from their mitochondria; when NGF is restored, the neurones recover and re-fill their mitochondria with cytochrome c. However axotomy induces caspase-independent death and caspase inhibition does not therefore facilitate neuronal recovery when the cells are re-fed with NGF. Tolkovsky reported that the caspase inhibition does however result in the selective elimination of mitochondria from the cells. Other organelles remain intact, but the cells die subsequently by a non-apoptotic death she termed 'limoktonia'. A similar phenomenon was observed in HeLa cells treated with staurosporine, hinting at its generality in eukaryotic cells.

Leist has examined the characteristics of neuronal cell death induced by a range of different agents and suggests that there are many potential pathways leading to the demise of a neurone. With colchicine, for example, classical caspase-dependent apoptosis occurs and caspase inhibition or ATP depletion with ketohexoses can prevent the cell death. With other agents (including the ganglioside GD_3) cytochrome c is released, but caspase inhibition has no effect on the progression of neuronal death. Glutamate induces a rise in intracellular calcium and cell death without activation of caspase-3; interestingly, phosphatidylserine is still flipped to the external leaflet of the cell membrane, suggesting that although these cells do not exhibit the classical sequence of apoptotic changes, they can still be phagocytosed. Should we therefore define this as apoptosis or necrosis? The distinctions may be blurred in neuronal cell types.

Olfactory receptor neurones (ORN) die by apoptosis upon removal of their target, the olfactory bulb. Roskams showed that the neurones up-regulate and then sequentially activate caspases-9 and -3 following target deletion. Activation begins in the synaptic region, that is, at the site of the lesion and subsequently, active enzymes are detected in the cell bodies, suggesting a retrograde spread of caspase activity.

Gerry Cohen (Leicester) continued the themes of how proteins involved in apoptosis associate and are activated, issues which are shared between researchers interested in the Bcl-2 family of proteins and the caspases. Cohen's group has isolated the apoptosome, a high molecular weight complex which is key for co-localising the cellular components required for caspase activation. In the presence of ATP, the 700 kDa complex contains Apaf-1 and the active forms of caspases-3, -7 and -9. The active enzymes are also present in a free form, not associated with the apoptosome. Cohen has also observed an even larger complex of 1.4MDa. However this contains only caspase-9 and Apaf-1 without the effector caspases-3 or -7 and this complex is catalytically inactive. Why? Apaf-1 has a cleavage product present only in the 1.4MDa complex, and Cohen proposed that the larger complex may contain inappropriately oligomerised or folded Apaf-1 molecules which are more susceptible to proteolysis and render the complex catalytically inactive.

The caspase dependence of nuclear apoptosis was described by **Bill Earnshaw** (Edinburgh). He summarised work from his laboratory that chicken DU249 hepatoma cells have three discernible stages of apoptosis: a condemned phase, in which cells can be rescued by survival factors, a committed phase during which caspases are activated and rescue is no longer possible and an execution phase when the morphological features of apoptosis are induced. Execution phase cell extracts contain caspase-activated factors which can induce DNA fragmentation and apoptotic nuclear morphology even in the presence of caspase inhibitors. One such factor is ICAD (inhibitor of CAD, caspase-activated DNAase). Although neither ICAD or CAD possess classical nuclear localisation sequences, the ICAD-CAD complex can enter the nucleus, ICAD acting as a chaperone as well as inhibitor of CAD. The presence of ICAD blocks DNA fragmentation by CAD but not chromatin condensation; Earnshaw suggests that this requires topoisomerase II. CAD can bind topo II and indeed topo II may target CAD to specific sites on the DNA. Therefore, the classic features of apoptosis seen in the nucleus, namely chromatin condensation and DNA fragmentation depend on three enzyme activities: caspases, CAD and topo II.

In the final sessions discussing cellular execution, **Jurg Tschopp** (Lausanne) and **Marcus Peter** (Chicago) talked about different aspects of the Fas-induced death cascade and its control. Beginning with the death receptors themselves, Tschopp described how the receptors

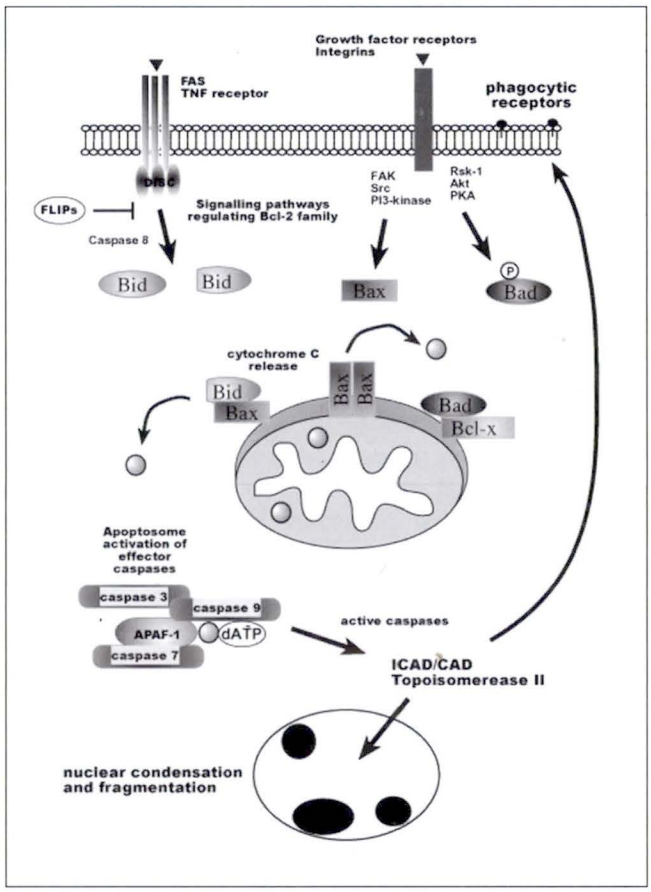


Figure 2: Multiple steps in apoptosis regulation. The mitochondria has a central role in the regulation of apoptosis in response to multiple pathways. Growth factors, adhesion to ECM and death receptors can all regulate the function of Bcl-2 family proteins such as Bid, Bad and Bax. Following an apoptotic signal, these translocate to mitochondria, where they promote the release of cytochrome *c*. Cytochrome *c* in the cytoplasm is involved in the activation of caspases, in the apoptosome, leading to the morphological signs of apoptosis and phagocytosis.

may not exist as monomers which trimerise upon ligand binding as previously thought, but may associate in inactive trimers at the cell surface; ligand binding then leads to a conformational change exposing the intracellular death domains that recruit adaptor proteins and activate caspase-8, leading to the rapid death of the cell. However, death is not inevitable upon ligand binding by death receptors. Receptor mediated death can be regulated by inhibitors known as FLIPs, which possess death effector domains and caspase-like regions but lack enzyme activity. The levels of these proteins can determine whether a cell lives or dies; for example, tumours may overexpress FLIP, thereby enhancing cell survival. Likewise, the timing of deletion of reactive T cells in an immune response is regulated by the timing of FLIP expression. Mice

knocked-out for many of the proteins in the Fas death receptor pathway present similar phenotypes; in addition to the anticipated defects in receptor-induced apoptosis, there is for example, no T-cell proliferation in the FADD knockout mice.

So do these proteins therefore play a role in cell proliferation? Perhaps, since proliferating cells assemble a death inducing signalling complex (DISC), with FADD, caspase-8, and a long isoform of FLIP, which in turn recruits Traf-1 and 2, and these act as adaptors in the pathway leading to NFkB activation. Multiple pathways therefore can be activated by death receptors and their interplay probably depends on expression levels and on post-translational regulation; for example, Tschopp closed by describing the protein FIST, a serine/threonine kinase, localised mainly in the nucleus, which can phosphorylate FADD and inhibit activation of JNK in response to Fas ligand.

Peter has shown that inactive caspase-8 exists predominantly at the mitochondria. Caspase-8 is however activated in the DISC – at the plasma membrane – and then translocates to the mitochondria to activate further caspase-8, whereupon the pro-domain remains at the mitochondrion and the active enzyme is released. Interestingly, Peter described a novel role for Bcl-X, namely that it can regulate caspase-8 function through interfering with the release of active enzyme from the mitochondria, an effect unique to caspase-8 and not shared by other caspases.

The meeting closed – as many apoptosis conferences do – with a discussion of how the cellular remains of apoptotic cells are cleared by phagocytosis. Many macromolecules are implicated in phagocytic recognition of dying cells and **Chris Gregory** (Nottingham) discussed CD14, a pattern recognition receptor previously identified as a receptor for lipopolysaccharide (LPS). His laboratory has shown that the ligand for CD14 on dying cells is not phosphatidylserine (PS), a lipid already implicated in phagocytosis, but ICAM-3 and overexpression of ICAM-3 can increase phagocytosis. In contrast to PS-stimulated phagocytosis, CD14-mediated clearance is caspase-3 independent. The CD14 knockout mice, which are resistant to septic shock induced by LPS, display increased levels of apoptosis, and Gregory suggested that this may result from reduced clearance of apoptotic cells rather than increased cell death *per se*.

Both Gregory and **John Savill** (Edinburgh) pointed out the critical anti-inflammatory significance of phagocytosis in cell death, particularly for cells such as neutrophils, which are packed with degradative enzymes. Moreover, Savill described how phagocytosis of apoptotic cells can elicit anti-inflammatory responses such as decreased production of agents such as TNFα and increased secretion of IL-10 from macrophages. Future therapies might therefore employ phagocytosis as a novel strategy (for example, in the fight against inflammatory and autoimmune diseases), and possibly even by inducing cells such as tumour cells to display 'eat-me' signals and so target themselves for phagocytosis.

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The biology of thrombospondins and other modulatory extracellular matrix proteins

N. Anilkumar and Soren Prag

The second international meeting on the Biology of Thrombospondins and other Modulatory Extracellular Matrix Proteins was held at the University of Wisconsin in Madison, between June 4–8 this year. Madison is located at an isthmus between the two lakes, Monona and Mendota, and the conference was held in the old buildings of the University with a beautiful overview of the lake. The meeting was perfectly organised by Deane Mosher with help from Josephine Adams. We apologise that for reasons of space we cannot mention all the presentations made at the meeting and have therefore chosen to highlight the main areas of current activity and the new themes which emerged at the meeting.

Modulatory extracellular matrix proteins are defined as non-constitutive matrix components which act to regulate cell shape and behaviour. The major matrix protein families in this grouping are the thrombospondins, the tenascins and SPARC (secreted protein, acidic and rich in cysteine) family proteins. The meeting brought together an inclusive international program of the major active labs to cover current topics relating to the structures and functions of modulatory matrix proteins.

Regulation of Angiogenesis

The regulation of angiogenesis is now a major focus for the development of therapeutics to inhibit tumour growth. Thrombospondins-1 and -2 act to inhibit angiogenesis in culture and in mice through interactions of the three thrombospondin type 1 repeats (TSRs). The effects of thrombospondins-1 and -2 in inhibiting angiogenesis, and their cellular mechanisms of action, was one of the major important themes of this meeting. A pioneer in the field of thrombospondin is **Jack Lawler** (Harvard, USA). At the meeting he described experiments to characterise which portions of the TSRs are responsible for the inhibition of tumour growth. Injection of recombinant protein, containing different combinations of the repeats, into mice inhibited tumour growth in a dose-dependent manner. The inhibition of tumour cell growth and induction of apoptosis by the second TSR was dependent on inclusion of the transforming growth factor-β (TGF-β) activating sequence. **Olga Volpert** (Northwestern University, USA) described the signalling pathway responsible for these properties of thrombospondin-1. She presented how thrombospondin-1 activates the CD36 receptor at the surface of endothelial cells and initiates a signalling cascade involving p53fyn, caspase-3 and the stress-activated kinase p38 MAPK which leads to activation of a second caspase cascade. This was impressively and convincingly shown using endothelial cells from the respective null mice. The induction of endothelial cell apoptosis by TSP-1 was inhibited upon addition of caspase inhibitors. Thus, the fate of endothelial cells is likely determined by the equilibrium of survival and apoptotic signalling cascades.

Donald Senger (Harvard, USA) talked about the role of collagen receptors α1β1 and α2β1 in vascular endothelial growth factor (VEGF) driven angiogenesis. VEGF induces a 5–7-fold increase in protein expression of these collagen receptors on dermal microvascular endothelial cells, through induction of the transcription of their mRNAs. VEGF did not induce increased expression of other collagen-binding integrins. Induction of α1β1 and α2β1 expression by VEGF promoted cell spreading on collagen I gels which was abolished by a combination of α1-blocking and α2-blocking Abs. Strikingly, *in vivo*, a combination of α1-blocking and α2-blocking Abs markedly inhibited VEGF-driven angiogenesis.

Roy Silverstein (Cornell, USA) presented a detailed description of CD36 as the anti-angiogenic receptor for thrombospondin-1. CD36-null mice show abnormal plasma lipid and lipoprotein profiles and have marked impairment of fatty acid utilization in several tissues. This is in agreement with the reported fatty acid transport function of CD36. Although superficially normal, the CD36-null mice have higher brain capillary densities that wild type controls and fail to show any anti-angiogenic responses to TSP-1.

Joanne E. Murphy-Ullrich (University of Alabama, USA) discovered some years ago that thrombospondin-1 activates latent TGF-β by an inter-molecular interaction involving the peptide motif RFK between the first

and second TSR. To determine the biological significance of this effect, she showed that the phenotype of thrombospondin-1 null mice reverted towards the wild type when the null mice were treated with peptides derived from the TSR. Moreover, treating wild-type animals with blocking peptides derived from thrombospondin-1 could induce a phenotype similar to that of TGF-β null animals.

The thrombospondin type 1 repeat (TSR) superfamily

Over the last couple of years, many new additional proteins that contain TSR have been identified. The question of whether they have properties in common with thrombospondin-1 and thrombospondin-2 is of more than academic interest because of the anti-angiogenic activities of the TSR of thrombospondins. A second theme at the meeting thus related to the biological roles of these new relatives. In her Plenary Talk, **Judith Kimble** (University of Wisconsin, USA) gave an overview of her lab's work on GON-1 metalloprotease in *C. elegans*. The *gon-1* gene encodes a secreted metalloprotease with a metalloprotease domain and multiple TSRs. The TSRs are postulated to anchor GON-1 to the extracellular matrix and thereby localise its metalloprotease activity. In *gon-1* mutants, gonadogenesis is impaired such that no arm elongation occurs and somatic gonadal structures are severely malformed. Kimble described how Gon-1 is expressed in two sites, leader cells and muscle, and expression in each site has a unique role in forming the gonad. The *gon-1* gene is crucial for migration of the gonadal leader cells. In wild-type animals, the leader cells migrate hundreds of microns, whereas in gon-1 null mutants, the leader cells do not move at all. The *gon-1* mutants also have defects in gonadogenesis that are unrelated to leader cell migration, suggesting that gon-1 might have a second role in gonadogenesis.

Following from this talk, it was interesting to hear the effects of a TSR relative being knocked-out in mice. **Kauji Kuno** (Kanazawa, Japan) described the work in his lab on a matrix-anchored metalloprotease, ADAMTS-1. He reported that the precursor and processed forms of ADAMTS-1 are secreted from cells and bind to the extracellular matrix (ECM) in a heparin-sensitive manner. Analyses of deletion mutants of the ADAMTS-1 protein revealed that a spacer region and the three TSR were important for a tight interaction with the ECM. Targeted disruption of the mouse ADAMTS-1 gene resulted in growth retardation with malformation of adipose tissue, kidneys and the adrenal medullary gland. Interestingly, in relation to the *gon-1* mutants, female mice had malformed ovaries and low fertility. ADAMTS-1 thus appears necessary for normal growth, female gonadogenesis and the morphology and function of certain organs.

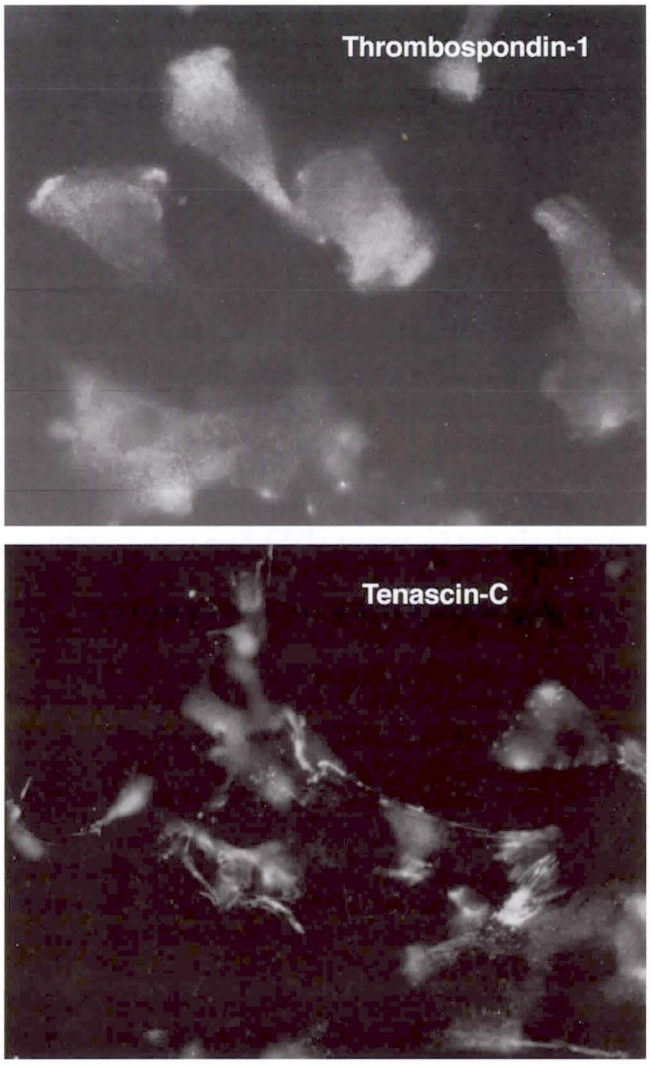

Figure 1. Thrombospondin-1 and tenascin-C in the matrices of locomotory myoblasts. Images courtesy of the Adams lab.

Luisa Iruela-Arispe (UCLA, USA) described studies on the secretion and processing of ADAMTS1. She showed that human ADAMTS-1 is secreted, then processed in two consecutive steps to release two active forms. Matrix metalloproteinases 2, 8 and 15 were able to release the smaller form from the larger one. This second processing step removes two TSR from the carboxyl terminal end of ADAMTS-1 and alters the affinity of the protein for heparin. ADAMTS-1 has anti-angiogenic activity in culture assays, and she hypothesises that processing could be relevant for modulating the anti-angiogenic properties.

Sunil Apte (Cleveland Clinic, USA) gave an overview of the whole ADAMTS family, of which there are now around 25 members, and introduced a new subfamily of TSR proteins, the ADAMTS-like (ADAM-TSL) proteins. This family includes Punctin, a novel extracellular matrix component of muscle tissues.

There were two interesting talks that related to the structure of TSR. The first NMR-structure of a TSR-containing protein – heparin-binding growth-associated molecule or HB-GAM – was presented by **Ilkka Kilpeläinen (**Helsinki, Finland). HB-GAM is an extracellular matrix-associated protein implicated in the development and plasticity of neuronal connections in the brain which contains two repeats distantly related to TSR at primary sequence level. The NMR structure shows two domains of antiparallel β-sheets separated by a long flexible hinge. This structure opens the way to new studies of TSR and their functions.

There has been some debate about whether TSR bind heparin directly. An interesting talk from **Jan Hofsteenge** (FMI, Switzerland) cleared up some of the contradictions. He described a new protein modification, C-mannosylation of tryptophan residues, in which carbohydrate is attached to the indole moiety of tryptophan within a specific recognition sequence. This modification is not made in plants, yeast or eubacteria. Analysis of several TSR revealed multiple C-mannosylation sites within the TSR. The functional significance of this modification remains mysterious, yet when using synthetic peptides in comparison to native protein, one should be aware of the possibilities for different properties due to the absence of these post-translational modifications.

Cellular actions of modulatory matrix proteins

The cell biology and signalling mechanisms initiated by thrombospondins emerged as another hot topic of discussion in the meeting. The diversity in cellular responses to thrombospondin-1 is explained by differential expression by different cell types of multiple thrombospondin-1 receptors that induce distinct signals. **David Roberts** (NIH, USA) reported that integrin α3β1 is an activation-dependent adhesion receptor for thrombospondin-1 in breast carcinoma cells and small cell lung carcinoma cells. This integrin is also expressed in endothelial cells but its ability to recognize thrombospondin-1 is suppressed in confluent cells. When activated in subconfluent cells, it contributes to cell spreading on TSP-1. **Bill Frazier** (Washington University, USA) discussed the role of CD47 in cross-talk to integrin α2β1. He described how activation of α2β1 by thrombospondin-1 or a CD47-binding peptide stimulate smooth muscle migration by activation of a heterotrimeric G-protein.

The co-organiser, **Jo Adams** (UCL, UK), described cellular responses to thrombospondin-1. In contrast to fibronectin, collagen and various other extracellular matrix proteins, cell adherence to thrombospondin-1 promotes the formation of actin-containing microspikes. These microspikes contains the actin-bundling protein, fascin and are negatively regulated by protein kinase C-dependent phosphorylation of fascin. The formation of these spikes depends on activities of Rac and Cdc42, but not of Rho GTPase. **Mark Ginsberg** (TSRI, USA) presented a poster of recent data describing how the α4 integrin cytoplasmic domain binds to paxillin. Fusion of the α4-tail to the αIIb-integrin resulted in a reduction in the formation of focal adhesion and stress fiber formation, indicating that the association of paxillin and α4 promotes integrin signalling that leads to focal adhesion disassembly.

Paul Bornstein (University of Washington, USA) presented interesting studies of thrombospondin-2 null fibroblasts. These skin fibroblasts show altered adhesion properties in culture. Compared to wild-type cells, the KO cells show increased activity of metalloproteinase-2 (gelatinase-A). When KO cells are treated with the specific gelatinase-A inhibitor, TIMP2, or with neutralising antibodies against metalloproteinase 2, they regain full spreading on several matrix proteins.

Alan Rapraeger (Madison, USA) discussed the mechanisms by which heparan sulphate chains on proteoglycans mediate stable high affinity binding of fibroblast growth factors (FGFs) to their receptors and regulate FGF activity. Basement membrane and cell surface heparan sulphate proteoglycans differ in their ability to bind individual FGF family members, presumably due to domain structure differences in the heparan sulfate chains.

Tenascins, SPARC and New Modulatory Proteins

The other major theme of the meeting was advances in understanding the biology and mechanisms of other modulatory extracellular matrix proteins. **Ruth Chiquet-Ehrismann** (FMI, Switzerland) reported a novel family of proteins, teneurins, which are predominantly expressed in the nervous system. Chicken teneurin-1 and teneurin-2 were found as two homologues of the *Drosophila* pair-rule gene product Ten-m and *Drosophila* Ten-a. By *in situ* hybridisation, they showed that teneurin-1 and -2 are expressed by distinct populations of neurons during the time of axonal growth, most prominently in the developing visual

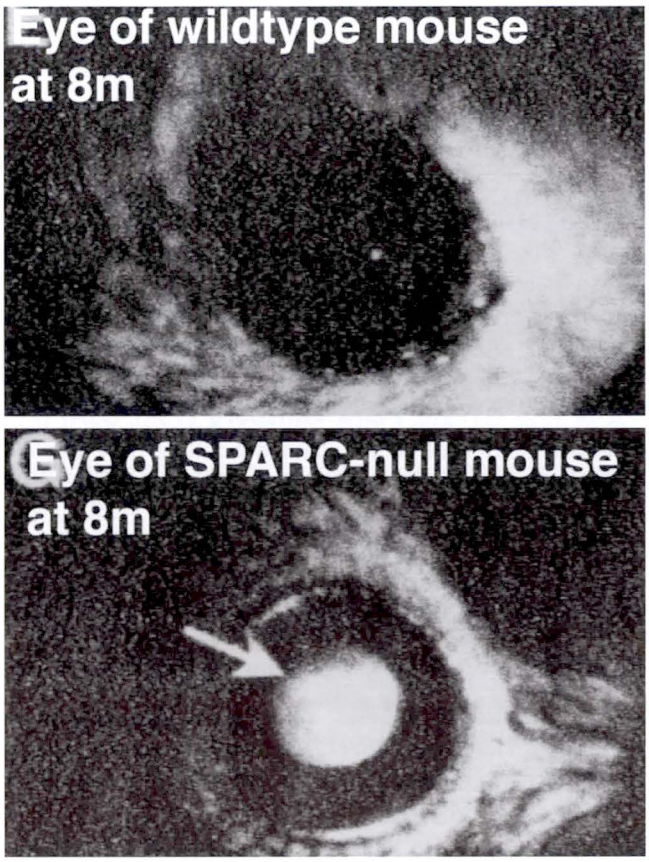

Figure 2. SPARC-null mice develop cataracts by 8 months (from Bassuk *et al.*, 1999, *Expl Eye Res* 88: 321–331).

system. The expression of teneurin-2 in neuronal cells leads to a significant increase in the number of filopodia and to the formation of enlarged growth cones. These proteins might have a function in the formation of neuronal connections.

Jean Schwarzbauer (Princeton, USA) discussed cellular effects of tenascin-C in a matrix gel designed to provide a matrix similar to that of healing wounds. Inclusion of tenascin-C in the gel alters actin cytoskeletal organisation in cells by modulating Rho GTPase activity.

Helen Sage (Washington, USA) described the phenotype of SPARC-null mice. A prominent phenotype is aberrant connective tissue responses to injury. In culture, cells from these mice exhibit significantly enhanced rates of proliferation. Most strikingly, all strains of mice lacking the *sparc* gene develop early-onset of cataracts associated with aberrant proliferation and differentiation of lens epithelial cells. Additional phenotypic alterations associated with aging, connective tissue, wound healing, and/or angiogenesis suggest unanticipated roles for SPARC in the maintenance of certain organs and tissues through life.

In his stimulating talk, **Matthias Chiquet** (Bern, Switzerland) presented data about the regulation of tenascin-C and collagen XII production by mechanical stress. It is well known that connective tissue cells adapt their ECM to changes in mechanical load. A feedback mechanism must exist, by which cells that sense mechanical stress from their matrix respond by an altered pattern of protein expression, and thus remodel the ECM to adapt to changing mechanical requirements. The production of tenascin-C and collagen XII, ECM proteins typical of tendons and ligaments, was found to be high in fibroblasts attached to a stretched collagen matrix, but suppressed in cells on a relaxed matrix. The response to a change in stress was rapid and reversible, and was reflected at the mRNA level. Both the tenascin-C and the collagen XII gene promoters contain 'stress-responsive' enhancer regions with similarity to 'shear stress response elements' found in other genes.

Richard Tucker (Davis, USA) gave us an entertaining and educational talk about his work on tenascin-Y, the avian homologue to the mammalian tenascin-X. Tenascin-Y is localised with the ependymal cells in the spinal cord and in the brain. In the adult, the protein levels are high at boundaries between the Schwann cells in the peripheral nervous system and the central nervous system. Tenascin-Y inhibits Schwann cell motility *in vitro* and thus might define a boundary *in vivo*.

The meeting ended with dinner and dancing to a local jazz band. This second ad-hoc meeting on modulatory matrix proteins generated a lot of enthusiastic discussion amongst the participants and we anticipate will be continued in a series of meetings.

N. Anilkumar and Soren Prag
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Gower Street, London WC1E 6BT, UK.

BSCB/BSDB Joint Spring Meeting 2001: Cell and Tissue Morphogenesis

University of Sussex, Brighton, UK. 3–6 April 2001

General Information

Arrive Tuesday 3 April in time for reception and dinner (1830); depart Friday 6 April afternoon.

Conference site:
The conference will be held on the University of Sussex campus. Further information about the site is available on: http://www.susx.ac.uk/menus/for_visitors.html

Full details of travel to the University of Sussex and further instructions about the conference and site will be sent to registrants approximately four weeks before the conference.

Registration
You can either register online (see Abstract Submission) or by downloading a PDF registration form on the web, downloading it and and sending it to the Meeting Secretariat.

The number of registrants is limited. In the event that the meeting is oversubscribed, priority will be given to those who present posters. The deadline for registration forms is 16 February 2001; those registering after this date are subject to a strictly enforced late registration penalty of £30.

Posters and Abstracts
There will be a joint poster session between the BSDB and BSCB.

Please note that, when submitting their poster, poster authors should state clearly their status (e.g. PhD student, post-doc, lab head, etc.), and whether the poster is to be entered into the poster competition (not lab heads), or to be considered for a short oral presentation (open to all).

Instructions for Abstract Submission
Abstracts must be submitted online to:
http://www.meetings-secretariat.com/
Abstract deadline: **16 February, 2001.**

Meeting Charges
The fee for residents covers registration, accommodation and all meals for the duration of the conference, excluding the Conference Dinner. The fee for non-residents covers registration teas, coffees and lunches each day.

Charges (further details on website)

2 nights full member	£140
2 nights student member	£115
3 nights full member	£175
3 nights student member	£150
non-residents (per day)	£50
Late booking	add £30
Non-members	add £35

Travel Grants
Remember that Honor Fell travel awards are available to cover basic travel and conference expenses. See Page 30.

Further details about the meeting can be found on the meeting website:
http://www.meetings-secretariat.com/

For queries, please contact:
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The Biochemical Society
59 Portland Place
London W1B 1QW, UK

Tel: +44 (0) 20 7580 3481
Fax: +44 (0) 20 7637 7626
E-mail: meetings@biochemistry.org

BSCB/BSDB Joint Spring Meeting 2001: Cell and Tissue Morphogenesis

University of Sussex, Brighton, UK. 3–6 April 2001
Organisers: Charles ffrench-Constant, Andy Furley, David Garrod, Alan Hall and David Wilkinson

Wednesday, 4rd April

Session 1	***Junctions*** ***Chair: David Garrod***
E Dejana (Milan)	Intercellular junctions in endothelial cells and their role in modulating cell growth and apoptosis
T Uemura (Kyoto)	Control of single-cell patterning by the seven-pass transmembrane cadherin
P Bryant (Irvine)	Organizers of signaling pathways at cell junctions
Selected Abstract	To be announced
Coffee	
E Peles (Rehovot)	Cellular junctions of myelinated nerves
Selected Abstract	To be announced
E. Knust (Dusseldorf)	Protein scaffolds and cell polarity in *Drosophila*
B Gumbiner (New York)	Cadherin Regulation in Tissue Morphogenesis
Lunch/posters	
Session 2	***Morphogenesis*** ***Chair: David Wilkinson***
D Wilkinson (London)	Control of cell movement during hindbrain patterning
K Basler (Zurich)	Genetic control of cell segregation at compartment boundaries
M Krasnow (Stanford)	Oxygen response pathways and airway branching in *Drosophila*
Coffee	
A Chisholm (Santa Cruz)	Cell signalling in *C. elegans* neural and epidermal morphogenesis
J Hardin (Madison)	Regulation of epithelial cell migration and adhesion in *C. elegans*
BSCB Hooke Medal Lecture	

Thursday, 5 April

Session 3	***Migration and Connection*** ***Chair: Andrew Furley***
N Brown (Cambridge)	Genetic dissection of integrin functions in morphogenesis
D Van Vactor (Harvard)	Mechanisms that control axon guidance in the *Drosophila* embryo
G Rougon (Marseille)	Cross-talk between IgCAM and semaphorin signalling in the formation and plasticity of neuronal networks
Selected Abstract	To be announced
Coffee	
L Zipursky (LA)	Molecular genetic dissection of axon guidance and targeting in *Drosophila*
Selected Abstract	To be announced
Y Jin (Santa Cruz)	A genetic study of synaptogenesis in *C.elegans* neuromuscular junctions
D Colman (New York)	The synaptic junction in the CNS: evolution, architecture and plasticity of an adhesive device
Lunch/posters	
Session 4	***Cytoskeleton*** ***Chair: Charles ffrench-Constant***
P Martin (London)	Epithelial sealing in morphogenesis and repair
Selected Abstract	To be announced

G Borisy (Madison)	Actin machinery for cell protrusion
Selected Abstract	To be announced
Coffee	
D Kiehart (Duke)	To be announced
A Hall (London)	The control of cell migration by Rho GTPases

BSDB Waddington Medal Lecture

BSCB and BSDB Annual General Meetings
Conference Dinner

Friday, 6 April

Session 5	***Signalling***	***Chair: Alan Hall***
M Peifer (Chapel Hill)	Cell adhesion, signal transduction and cancer: the Armadillo connection	
A Harwood (London)	Intercellular junctions and cell signalling in *Dictyostelium*	
M Ginsberg (La Jolla)	The inside story on integrins	
F Giancotti (New York)	To be announced	
Coffee		
F Watt (London)	Role of cell adhesion in regulating epidermal stem cell fate	
S Dedhar (Vancouver)	Integrin linked kinase (ILK) mediated regulation of epithelial–mesenchymal transformation	
R Hynes (MIT)	Cell adhesion in cancer	

Workshops

Wednesday, 3 April	***I: Regulation of cell motility***	***Organisers: Laura Machesky and Rob Insall***
Dave Knecht (Storrs)	From actin filaments to a cytoskeleton: insights from live cell imaging	
Frank Gertler (MIT)	Regulation of Cell Motility by Ena/VASP proteins	
Sutherland Maciver (Edinburgh)	Regulation of actin dynamics by ADF/cofilin	
Cornelis Weijer (Dundee)	The control of chemotactic cell movement during *Dictyostelium* development	
Harry Mellor (Bristol)	Co-ordinated regulation of the actin cytoskeleton by novel Rho GTPase Rif	
Britta Qualmann (Magdeburg)	The functional interface of endocytosis and the actin cytoskeleton	

Thursday, 4 April	***II: Biogenesis of post-Golgi organelles in health and disease***
	Organiser: Dan Cutler
Juan Bonifacino (Bethesda)	Role of mammalian Stoned proteins in endocytic trafficking
Gillian Griffiths (Oxford)	Rab 27 is critical for exocytosis of secretory lysosomes
Reg Kelly (UCSF)	Endocytic processes in the nerve terminal
Michael Marks (Philadelphia)	The role of Pmel17 and multivesicular bodies in the biogenesis of melanosomes: implications for the etiology of heritable pigmentation disorders
Scottie Robinson (Cambridge)	Adaptor complexes: Friends and family
Miguel Seabra (London)	Rabs and the formation of haemopoietic organelles
Richard Swank (Buffalo)	Altered Vesicle Trafficking in Mouse Pigment Mutants: an Overview
Sharon Tooze (London)	Biogenesis of neuroendocrine secretory granules requires both membrane fusion and budding

Meeting details, including Registration Forms and Abstract Submission, are available at:
http://www.meetings-secretariat.com/ Abstract and Registration deadline: **16 February 2001**

Other forthcoming meetings

BSCB Autumn 2001 Meeting
Cell Biology and Neurobiology – a meeting for Martin Raff

19–21 September 2001
University College London
Organisers: Anne Mudge (a.mudge@ucl.ac.uk), Bill Richardson (UCL), Ben Barres (Stanford)

A meeting to acknowledge the great impact that Martin has made and continues to have on British Cell Biology. The meeting aims to bring together speakers representing many of the fields in which Martin has himself worked.

Sessions will include: development, immunology, cell biology, application of basic science to medicine and science policy.

Speakers include: David Anderson, Marc Tessier Lavigne, Tom Jessell, Keith Roberts, Alan Hall, Tim Mitchison, Jordan Raff, Ron Laskey, David Lane, Charles Weissman and Bruce Alberts.

BSCB/BSDB Joint Spring Meeting 2002
Cell Regulation through Molecular Machines

20–23 March 2002
University of York

BSCB Autumn Meeting 2002
5th Abercrombie Meeting

September 2002
St Catherines College, Oxford.

Canadian Society of Biochemistry, Molecular & Cellular Biology Meeting 2001
Dynamics of Intracellular Organelles and Molecular Machines

8–11 February 2001
Mont Sainte-Anne in Beaupre, Quebec

Speakers include:
Peter Walter, Tom Rapoport, David Andrews, Hidde Ploegh, Walid Houry, John Glover. Jeff Kelly, Nancy Bonini, David Westaway, Danny Schnell, Felix Kessler, Gordon Shore, Ed Hurt, Brian Burke, Richard Wozniak, John Aitchison, David Thomas, John Bergeron, David MacLennan, William Wickner.

Organisers: David Williams, John Aitchison and Kay Craig

There will be travel awards for postdocs and grad students posters, fabulous skiing and a trip to Quebec City during the Winter Carnival. Contact Kay Craig at kcraig@med.mcgill.ca for further information

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European Tissue Culture Society (ETCS) International Meeting

Granada, Spain
30 September–3 October 2001

Invited speakers include: M. Bretscher, W. Franke, I. Freshney, P. Fromherz, N. Fusenig, J.D. Gearhardt, A. Pfeifer, A. Vescovi, A. Wobus

Topics include: Embryonic and somatic stem cells, Cell migration and extracellular matrix, Functional competence of immortalised cell lines, Cellular interactions, and Cell line validation. There will also be technology-based tutorials.

Workshops on these and additional topics will include oral presentations selected from submitted abstracts. For further details and to pre-register: email etcs_esp@gva.es , FAX +34 96 3868718. Or visit the website: *www.san.gva.es/centros/lafe/ETCS/ETCS_ESP-granada-2001-00.htm*

Techniques in Molecular Biology

University of Hertfordshire Courses for 2001

Website: www.herts.ac.uk/natsci/STC

Molecular biology update
A four-day laboratory course
9–12 April 2001, 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

RNA extraction and analysis
A one-day laboratory/lecture course
5 July 2001, Hatfield, Herts UK
Details and application forms from Dr Ralph Rapley,
Department of Biosciences, University of Hertfordshire
College Lane, Hatfield, Herts AL10 9AB UK.
tel:(01707) 285097 fax: 286137
e-mail: R.Rapley@herts.ac.uk

PCR methods and applications
A one-day laboratory/lecture course
6 July or 13 July 2001, Hatfield, Herts UK
Details and application forms from Dr Ralph Rapley,
Department of Biosciences, University of Hertfordshire
College Lane, Hatfield, Herts AL10 9AB UK.
tel: (01707) 285097 fax: 286137
e-mail: R.Rapley@herts.ac.uk

An introduction to bioinformatics
A two-day computer/lecture course
3–4 July or 10–11 July 2001, Hatfield, Herts UK
Details and application forms from Dr Henry Brzeski,
Department of Biosciences, University of Hertfordshire
College Lane, Hatfield, Herts AL10 9AB UK.
tel: (01707) 284554 fax: 286137
e-mail: H.Brzeski@herts.ac.uk

Microbiology techniques
A two-day laboratory course
3–4 September 2001, 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)285948 fax: 286137
e-mail: V.Bugeja@herts.ac.uk

Protein techniques
A two-day laboratory course
3-4 Sept or 10-11 Sept 2001, 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

Nucleic acid techniques
A three-day laboratory course
5–7 Sept or 12–14 Sept 2001, 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)285948 fax: 286137
e-mail:V.Bugeja@herts.ac.uk

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: .....

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

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1) .....

2) .....

Applicants without proposers should enclose a brief curriculum vitae.

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

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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):

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

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Date of joining BSCB: ....

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

Key publications (2) or research interests:

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

.....

.....

Estimated expenses: Travel: .....

Subsistence: .....

Registration: .....

Other: .....

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

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

.....

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

Supporting statement by Head of Department:

The applicant requires these funds and is worthy of support

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British Society for Cell Biology

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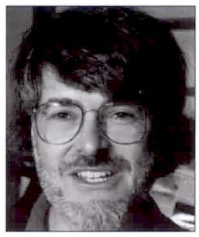

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