

Connective Issues

BSMB Newsletter

British Society for Matrix Biology

Committee: Prof. Tim Hardingham (Chairman), Dr Rose Maciewicz (Secretary), Prof. John Gallagher (Treasurer),
Prof Malcolm Davies, Dr Jay Dudhia, Prof Jo Edwards, Dr Alvin Kwan, Dr Jo Lewthwaite, Dr Louise McKenna, Dr Garry Rucklidge

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No. 53 January 1999

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Editorial

by Rose Maciewicz

Happy New Year and welcome to the 53rd edition of Connective Issues our first as the British Society for Matrix Biology. Yes, it is now official the Charity Commission has approved the name change of our Society. As part of this rejuvenation the Committee has worked diligently to improve the Society. Please note that thanks to Jay Dudhia we now have a live website which can be accessed via <http://www.bsmb.ac.uk>. If you visit the site, and we do hope you will, please note as they say in the trade, parts of the site are 'under construction'. So bear with us as it develops! Also you will note in 1999 the Society created a new award '**the Young Investigator Award**' to recognise the work of a young investigator in the field of matrix biology. Details of the competition were published in Connective Issues 52 and can also be found on our website. The Committee is pleased to announce that this award will be presented at the Spring BSMB meeting in Oxford on Wednesday 31st March 1999. Finally a copy of the modified Constitution is included in the Newsletter.

Many thanks to Professor Tim Hardingham and Dr. John Sheehan from The Wellcome Trust Centre for Cell Matrix Biology for organising the Autumn 1998 BCTS meeting in Leicester. It was an excellent meeting. For those of you who could not attend a report of this meeting is enclosed.

You should note that this is the last newsletter before the Spring BSMB meeting in Oxford, so please take note of the information and deadlines contained in this Newsletter for Programme, Registration forms, Bursary application form and travel directions. The Annual General meeting will be held at this meeting on Tuesday 30th March at 17:00 in the Academic Centre, John Radcliffe Hospital, Headington, Oxford. The preliminary agenda is included in the Newsletter and additional agenda items are welcomed.

Please make note in your diaries of our Autumn 1999 and Spring 2000 meetings. The Autumn 1999 meeting will be held in Aberdeen on Monday and Tuesday, 6th and 7th September 1999. The Spring 2000 BSMB meeting will take place in London from Monday 3rd April to Tuesday 4th, April 2000. More information about these meetings, including the preliminary programmes, can be found within the Newsletter. Please also note that we are co-organising a one day meeting with the

British Inflammation Research Association (BIRAs) on Thursday 25th February 1999. This meeting will be in London and a programme is included in this Newsletter.

The BSMB Committee is pleased to announce that we awarded eight Bursaries to attend the FECTS meeting in Uppsala. The recipients were: Miss Claire Allen (CTBL, Cardiff); Dr. Joe Bird (The Royal Veterinary College, London); Miss Kirsten Ferguson (CTBL, Cardiff); Dr. Janet Anderson-MacKenzie (Collagen Research Group, Bristol); Miss Louise McKenna (Royal Veterinary College, London); Mr. Anthony Reardon (Wellcome Trust Centre for Cell-Matrix Biology, Manchester); Dr. Allison Reith (Wellcome Trust Centre for Cell-Matrix Biology, Manchester); Miss Sian Thomas (CTBL, Cardiff).

If anyone has items for inclusion in the next newsletter or website, these should be e-mailed or posted to Dr. Rose Maciewicz at the address on the cover page. Please contribute if you can!

Current BSMB Committee

Officers:

Chairman, Prof. Tim Hardingham (University of Manchester; tharding@fs1.scg.man.ac.uk)
Honorary Treasurer, Prof. John Gallagher (Patterson Institute, Manchester; JGallagher@picr.man.ac.uk)
Honorary Secretary Dr. Rose Maciewicz (Zeneca Pharmaceuticals; rose.maciewicz@alderley.zeneca.com)

Elected Members:

Dr. Alvin Kwan (University of Wales, Cardiff; kwanap@cardiff.ac.uk)
Prof. Malcolm Davies (University of Wales, Cardiff; daviesm6@cf.ac.uk)
Dr. Garry Rucklidge (Rowett Research Institute, Aberdeen; gjr@rri.sari.ac.uk)
Dr. Jo Lewthwaite (Eastman Dental Institute, UCL, J.Lewthwaite@eastman.ucl.ac.uk)
Dr. Jay Dudhia (The Royal Veterinary College, London; jdudhia@rvc.ac.uk)
Prof. Jo Edwards (UCH Medical School, London; jo.edwards@ucl.ac.uk)

ex officio Member:

Dr. Louise McKenna (University Erlangen, Germany; 'Louise.McKenna@patho.med.uni-erlangen.de')

To aid in your choice here follows thumbnail sketches of the nominees.

Charity Commission Approval of Name Change

As you are aware in 1998 the Committee of the British Connective Tissue Society proposed that the Society should change its name. A ballot was held and this was ratified early in 1998. In the summer of 1998 the Secretary undertook to get this name sanctioned by the UK Charity Commission. The committee is now pleased to announce that the Charity Commission has formally agreed to the name change. We are now known as the **British Society for Matrix Biology (BSMB)**.

Nominations for BSMB chairman and committee members

The Chairman (TH) and three committee members (AK, MD, JE) are due to retire in **Spring 1999**. We have had no nominations for the position of Chairman, therefore the Committee has nominated Professor Tim Hardingham to stand another 3 year term of office. Professor Hardingham has agreed to this nomination. We have received the following nominations (in alphabetical order) for the position of Committee member:

Dr. IAN CLARK - University of East Anglia; nominated by Gillian Murphy (UEA) & David Young (Manchester)

Dr. ANTHONY DAY- University of Oxford; nominated by Alvin Kwan (Cardiff) & Tim Hardingham (Manchester)

Dr. ANTHONY HOLLANDER- University of Sheffield; nominated by David Buttle (Sheffield) & John Tarlton (Bristol)

Dr. NORMAN McKIE - University of Newcastle; nominated by Tim Cawston (Newcastle) & Bruce Caterson (Cardiff)

Dr. ALISON REITH - University of Manchester; nominated by Jill Moss (Imperial College London) & Janet Anderson-MacKenzie (Bristol)

Because we have more nominations than Committee places a Ballot is required. You will find this ballot attached to the end of this Newsletter. Please indicate your choices by placing a cross (☒) in the boxes by no more than 3 names (ballot papers voting for more than 3 names will be deemed null and void). Please return your ballot paper by **February 15, 1999** to the BSMB Secretary.

Name: Ian Clark
Position: Research Fellow, funded on an ARC Postdoctoral Fellowship
 School of Biological Sciences at the University of East Anglia

Research Interests: My current research interests centre around control of MMP and TIMP gene expression with projects in the lab. on: mapping human TIMP-1 promoter (my own favourite); induction of TIMP-1 by retinoic acid in connective tissue fibroblasts; cell-specific effects of retinoic acid, comparing osteoblasts to fibroblasts; alterations in TGF-beta signalling to MMP and TIMP gene expression during ageing; growth factor effects of TIMPs; and MMPs in plants (it's good to have a hobby!).

Why I Want To Be On The Committee:

I've been a member of the BCTS/BSMB for 12 years, and presented work at a number of their UK meetings and also the FECTS. Now, (despite the old adage that the ideal number of people needed to get something done by committee is three, two of whom are absent!), I feel that it is my turn to join the committee and contribute to the organisation and administration of the Society. UEA has recently expanded its research interests to become a centre for matrix biology, and it is important that we are represented; hopefully Norwich will be the location for a BSMB meeting in the not-too-distant future. I would bring to the committee my experience of research in both a clinical-scientific and a University environment, in the UK and US; new ideas such as establishing one day workshops for graduate students to present work and get together; my enthusiasm to take on an active role in the Society.

Name: Anthony Day
Position: MRC Senior Scientist
 MRC Immunochemistry Unit,

Department of Biochemistry, University of Oxford
Research Interests: I took up my present post of MRC Senior Scientist (MRC Immunochemistry Unit, Oxford) in June of this year after having been an Arthritis and Rheumatism Campaign Research Fellow for nearly 7 years. My main research interests are the investigation of the structural basis of hyaluronan-protein interactions and the functional roles of hyaluronan-binding proteins in ECM (particularly cartilage) and cell migration. For more detailed information see <http://www.bioch.ox.ac.uk/info/brochure/day.html>.

Why I Want To Be On The Committee: I am keen to promote the field of matrix biology through serving on the BSMB Committee. I have been a member of the BSMB since 1994.

Name: **Anthony Hollander**
Position: Lecturer (permanent, Higher Education Funding Council funded)
University of Sheffield Medical School

Research Interests: Cartilage Biology; Osteoarthritis; Collagen Degradation Mechanisms; Tissue Engineering; Gene Therapy (Gene Transfer To Chondrocytes)

Why I Want To Be On The Committee:

I want to get more involved with the society so that I can have some influence over its future direction, particularly with regard to the format of meetings and the participation of younger scientists. Sheffield is an active centre for various aspects of connective tissue research. The city should be represented on the BSMB committee and at present it is not. I have experience of active committee work at the University of Sheffield. I understand the need for all members of a committee to be active participants.

Name: **Norman McKie**
Position: University Lecturer (permanent funded by the Higher Education Funding Council Department of Medicine (Rheumatology), Newcastle-Upon Tyne

Research Interests: Structure and function of metalloproteinase-disintegrins in cells of connective tissue origin. The role of the functional domains of Tissue Inhibitor of Metalloproteinases-3 (TIMP-3) and an analysis of its matrix binding properties.

Why I Want To Be On The Committee:

I have worked in labs with a large component of connective tissue research in Cambridge, Sheffield and Newcastle and have a pretty broad knowledge of the field with technical experience gained in all three centres. I am especially interested in Bioinformatics and molecular aspects of Biochemistry with regard to matrix molecules and have formed a group dedicated to the structural/functional analysis of enzymes and inhibitors relevant to connective tissue turnover. I feel that my broad background in the field may be of use on a committee. I have also arranged a couple of one day meetings on my own subject area and feel that this type of experience would also be useful from an organisation point of view. In addition I actually want to get involved as I would

like to really get into promoting matrix research as there is not half enough people doing this type of work.

Name: **Alison Reith**
Position: For the past 18 months I have been working as a BBSRC funded postdoctoral Research Associate at the School of Biological Sciences at the University of Manchester.

Research Interests: I am currently investigating the role fibrillin microfibrils at the dermal-epidermal junction in skin. This work has involved establishing three-dimensional in vitro models for studying basement membrane formation in the skin.

Why I Want To Be On The Committee:

During my research career I have studied proteases and metalloproteinases, integrin expression, collagen synthesis and the role of ECM proteins in tumour invasion, renal basement membrane composition in normal and polycystic disease as well as developing *in vitro* models for studying basement membrane formation in kidney and skin. I have actively attended BCTS meetings since joining the Society in 1990. As a young scientist, the BCTS has provided me with an invaluable network of friends and collaborators and has supported me financially via travel bursaries to attend and present my work at the FECS meetings. The BCTS has also provided me with a platform to present my research to an informed audience which has been both objective and supportive. I should like to be a member of the committee as I feel that it is now time that I gave something back to a society which has given me so much.

Membership Renewal Information

The BSMB Committee has decided to update its membership files for the 1999 year. We are requesting that all members complete the attached form 'AUDIT OF MEMBERSHIP DETAILS', which can be found on page x of the appended section of the newsletter, and forward it to the BSMB Secretary. The form should be self-explanatory but if you have any queries please contact the Secretary by e-mail.

Note that the 1999 membership fee is £10.00 for full members and £2.00 for student members. As in the past no subscription is payable by Honorary Members. If you pay by Direct Debit can you please give us your Bank details so that we can confirm this arrangement and also can you please

inform you Bank that our name has changed. To facilitate this for you we have drafted a form for you to notify your bank of the change of the name of the Society. Please complete and send directly to your bank as soon as possible (do not send to the Society). This form can be found on page xi of the appended section of the newsletter. If you have any queries or problems with this form please contact BSMB assistant secretary, Dr. Jo Lewthwaite, Cellular Microbiology Research Group, Eastman Dental Institute, 256 Gray's Inn Road, London, WC1X 8LD, UK. Email: J.Lewthwaite@eastman.ucl.ac.uk, Tel: 0171 915 1247, Fax 0171 915 1259.

If you do not pay by Direct Debit please send us a cheque immediately. Please note membership fee is collected on January 1st of the year. If you fail to send us your 1999 subscription fee we will have to cancel your membership. Please help the Society to stay solvent by paying your membership fees.

Under the provision of the Data Protection Act 1994 s.33(3) we are required to inform our members that we are holding a mailing list on computer disc. The information is only for the purpose of distributing or recording the distribution of articles of information to members and consists only of their names; addresses and other particular necessary for such distribution. A member objecting to the information being held as mentioned should notify the current BCTS Secretary.

Encourage your colleagues to join the BSMB!

The BSMB can only exist with the support of its members. Please encourage your students, and colleagues, both clinical and non-clinical, to join us. Membership offers reduced registration to all BSMB Meetings, the opportunity to apply for student bursaries and free subscription to our newsletter Connective Issues. A copy of the application form is included on page ix of the appended section of the newsletter.

Website information

The BSMB now has its very own Website. The Society previously had Web pages hosted by Manchester University, however, this has now lapsed. Following a decision made by the Committee that the Society should have its own Website, we now have registered with UKERNA the domain name bsmb.ac.uk. The Website for this domain name has very kindly been hosted by the

Computing Services at the Royal Veterinary College, University of London. Access to the site can be gained by using this name (just type <http://www.bsmb.ac.uk>).

There has been some delay in getting the site on the internet partly because we had to wait for the official name change of the Society, and also because of major changes to the Computing Servers at the RVC during the summer. The site includes information on the Society, forthcoming meetings, the Newsletters, links to related Societies, job opportunities and hopefully, at some stage a members area to express views. We also hope to form links with or perhaps host pages for our European Continental counterparts.

Annual General Meeting

The Annual General meeting will be held at this meeting on March 30th at 17:00, The Academic Centre, John Radcliffe Hospital, Headington, Oxford. The preliminary agenda follows. Items for inclusion should be sent to the BSMB Chairman, Professor Tim Hardingham.

BSMB ANNUAL GENERAL MEETING - preliminary agenda

1. Approval of the minutes of the last AGM held on 30th March, 1998, University Hall, University of Wales Cardiff
2. Matters Arising
3. Secretary's report
4. Treasurer's report
5. Election of Chairman and New Committee Members
6. Any other business

IJEP poster competition

Please note that for Spring BSMB meeting at Oxford there will be a poster competition for young BSMB members. The poster competition, which is sponsored by the International Journal of Experimental Pathology, is designed to reward PhD students and post-docs, who put a lot of time and effort into making an excellent collection of posters at the BSMB meetings each year. If you are a PhD student or post-doc please enter the competition. You have nothing to lose and you may just win £100. If you are a supervisor, encourage your PhD students and post-docs to enter the competition. Not only will they receive some money, but more

importantly it will draw attention to their work and receipt of the reward might be a useful addition to their CV! **Please note that this is an open competition and is not be restricted to posters dealing with the theme of the Spring 1999 meeting, i.e. wound healing.** Closing date for the is Monday 15th February 1999.

Competition rules:

1. It will be held once per year and at a BSMB meeting selected by the Committee.
2. The competition will be publicised well in advance.
3. The competition is open to PhD students and recently qualified post-docs (up to 2 years), who must indicate prior to the meeting their intention to enter the competition.
4. Up to 3 scholarships of £100 each will be awarded, dependent on the quality of the presentations. If the quality is low, no awards will be made.
5. The posters will be judged by at least 3 Committee members who will view the posters as well as discuss the work with the poster presenter. Criteria for judging the posters will be: clarity of the presented poster; scientific content; and scientific understanding of the work.
6. The award to be used by the recipient as they choose.
7. The recipients of the award to be notified at the meeting and also to be listed in the next newsletter issue.

BSMB Bursaries for Oxford meeting

We are offering BSMB bursaries to attend the Spring 1999 BSMB meeting in Oxford. Young members of the Society are encouraged to apply for bursaries (up to a maximum of £75) to assist with attending these meetings. An application form is included with this newsletter. Bursaries will only be considered if they are submitted on a current BSMB Bursary application form. Applications should be sent to the Secretary and not to the meeting organiser. **The application should be accompanied by a copy of the abstract to be presented at the meeting and a one page curriculum vitae.**

The deadline for receipt of bursaries to attend the Oxford meeting is Monday 15th February 1999. The applications will be reviewed rapidly by the Committee and applicants will be informed of the outcome on or around 28th February 1999.

Criteria for Bursaries

1. Applicants should have been members of the Society for at least 1 full calendar year.

2. Applicants should be submitting an abstract and presenting a poster for the meeting to be attended.
3. Applicants should be at an early stage of their career and unlikely to have access to travel funds. Most often where support for an overseas meeting is given this is the first such meeting they attend. For this reason emphasis is always given to young researchers who are generally in short term contract positions, i.e. mainly graduate students and occasionally early post-docs.
4. The work described in the abstract must be novel and likely to be of a quality that would reflect well as a BSMB supported contribution.

One Day Meeting between BSMB and BIRAs

The BSMB Committee are pleased to announce a one day meeting between the BSMB and the British Inflammation Research Association (BIRAs). The title of this meeting is **'In vivo models of Matrix Remodelling - Classical to Modern Approaches'**. The meeting will be held on Thursday 25th February 1999 at the Paul Garrod Lecture Theatre, Robin Brook Centre, St Bartholomew's Hospital, West Smithfield, London EC1A 7BE. The co-organisers and chairs of this meeting are Dr. Adrian Moore (Department of Experimental Pathology, St Bartholomew's Hospital, West Smithfield, London EC1A 7BE, tel: 0171 982 6125, fax ; 0171 982 6095, email: A.R.Moore@mds.qmw.ac.uk) and Dr. Rose Maciewicz (BSMB Secretary). Further information about the programme, registration forms and directions will be sent out in the New Year.

Preliminary Programme for BSMB/BIRAs

| | |
|-------------|--|
| 9.30-10.00 | Registration & coffee |
| 10.00-10.45 | Professor Willoughby Forty years of inflammation research. |
| 10.45-11.30 | Dr. Dean Willis Modelling the adaptive response to inflammation |
| 11.30-12.15 | Dr. Mike Seed Inflammatory angiogenesis |
| 12.15-1.45 | Lunch Wykham Balme Rooms |
| 1.45-2.15 | Dr. Patricia Sime TNF gene transfer in the lung |
| 2.15-3.00 | (Speaker to be confirmed) Role of transgenics in the development of new animal models of disease |
| 3.00-3.30 | Tea |
| 3.30-4.00 | (Speaker to be confirmed) |

| | |
|-----------|---|
| 4.00-4.45 | Transfer of Human RA synovium to SCID mice as to model lymphocyte trafficking Dr. John Waterton |
| 4.45-5:15 | Advanced imaging techniques in assessing <i>in vivo</i> matrix remodelling |
| 5:15 | General discussion Close of meeting |

Spring 1999 BSMB/ETRS meeting in Oxford Programme

The Spring 1999 meeting of the Society on 'The Molecular and Cell Biology of Wound Healing' will take place in Oxford from Tuesday 30th March to Thursday 1st April and is being jointly organised with the European Tissue Repair Society (ETRS). Final details of the meeting, registration forms, travel information are all included in this Newsletter (page iv of appended section). If further information is required the contact persons for the meeting are Prof. Malcolm Davies (01222 492233 Ex 5292; Fax 01222 453643; daviesm6@cf.ac.uk) and Dr. Robert Steadman (01222 492233 Ex 5315; rsteadman@cf.ac.uk). Please note that there will be a IJEP poster competition at the meeting (see below) and that the winner of the BSMB Young Investigator Award will give a seminar and be presented with an award.

Autumn 1999 BSMB meeting in Aberdeen

A conjoint meeting of the Bone and Tooth, and the BSMB will be held at The University of Aberdeen, King's College Conference Centre, Aberdeen, Scotland on Monday and Tuesday, September 6th and 7th 1999. The theme of the meeting will be "Biology and Therapeutic Strategies in Skeletal Disease". The complete programme is shown below. There will be a Dinner and Ceilidh on the evening of 6th September. As this will be a full two-day meeting, travel the day before the meeting is advised. The local organisers are Prof. Stuart Ralston (BATS), Department of Medicine and Therapeutics, Polwarth Building, Medical School, Foresterhill, Aberdeen (Tel 01224 681818 ext 53015, email s.ralston@abdn.ac.uk) and Dr Garry Rucklidge, (BSMB) Skeletal Research Unit, The Rowett Research Institute, Bucksburn, Aberdeen AB21 9SB (Tel 01224 716640, email gjr@rri.sari.ac.uk).

Monday 6th September

Session 1 - Collagen and Bone

| | |
|-------------|--|
| 9.30-10.10 | Connective Issues No. 53 January 1999 Prof. J. Risteli (University of Oulu, Finland) Markers of bone turnover |
| 10.10-10.50 | Prof Paul Bornstein (University of Washington, Seattle, USA) Collagen (regulation, knockouts also thrombospondin) |
| 10.50-11.10 | Coffee |
| 11.10-11.50 | Dr Nick Bishop (University of Sheffield) Osteogenesis imperfecta |
| 11.50-12.40 | Short presentations selected from submitted abstracts |
| 12.40-14.00 | Lunch and posters |

Session 2 - Cartilage matrix

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|-------------|--|
| 14.00-14.40 | Prof. Robin Poole (Montreal) Collagen Turnover in Cartilage Matrix |
| 14.40-15.20 | Prof. Bruce Caterson (Cardiff) Mechanisms of cartilage degradation in arthritis |
| 15.20-16.00 | Coffee and posters |
| 16.00-17.00 | Poster discussion (6 x10min) |

Dinner and Ceilidh - buses depart 19.00 prompt.

Tuesday 7th September

Session 1 Osteoclast-Matrix interactions

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|-------------|---|
| 09.00-09.40 | Prof. T Chambers (St George's, London) Recent advances in osteoclast biology |
| 09.40-10.20 | Dr. MH Helfrich (University of Aberdeen) Adhesion mechanisms in bone. |
| 10.20-11.00 | Coffee and posters |
| 11.00-12.30 | Short presentations selected from submitted abstracts |
| 12.30-13.30 | Lunch and posters |

Session 2 - Cell therapy and cartilage

| | |
|-------------|--|
| 13.30-14.10 | Dr Anders Lindahl (Gothenberg) Treatment of cartilage injuries with chondrocyte transplantation -Cellular and molecular aspects |
| 14.10-14.50 | Dr. Frank Barry (Osiris) Stem cell strategies for skeletal repair |
| 14.50-15.30 | Prof. James Richardson (Oswestry) |
| 15.30 | Close of meeting and coffee |

Spring 2000 BSMB meeting in London - The Millennium Meeting

The Spring 2000 BSMB meeting 'Molecular Cell Biology of the Synovial Joint' will take place in London from Monday April 3rd to Tuesday 4th, 2000.

It will be held at the Royal Veterinary College, University of London, at the Camden Campus of the College. The organisers of the meeting are Professor Michael Bayliss (0171-468 5268, Fax: 0171-388 1027, mbayliss@rvc.ac.uk) and Dr. Jay Dudhia (0171-468 5269, Fax: 0171-388 1027, jdudhia@rvc.ac.uk). The programme will include sessions on Bone and Cartilage, Synovium and Vasculature, and Tendon. Further details and a provisional programme for the Meeting will appear in the next Connective Issues. In addition to plenary speakers, a number of authors submitting abstracts will be selected by the organising committee to give short presentations of their work. At this meeting there will be 2 further special talks – the first for the **BSMB Young Investigator Award**, and the second for a **Veterinary Investigation Award**. Some of the sponsorship for this meeting will be from Veterinary research funding bodies and from pharmaceuticals with a major interest in Veterinary medicine, and therefore the second award will be supported by these sources.

BSMB Young Investigator Award. This award aims to recognise the contribution made by younger researchers to the field of matrix biology. The recipient will be invited to give a special seminar at the spring meeting of the Society and will be awarded a one hundred pound honorarium and presented with a certificate. The cost of attending the meeting will be met by the Society including registration, reasonable travel costs, accommodation and meals. The closing date for receipt of applications is 1st October 1999 and should be sent to BSMB secretary.

Guidelines for applicants for BSMB Young Investigator Award

1. The applicant should be 35 years or under on the date of the presentation of the award.
2. The applicant should have been a member of the Society for at least 12 calendar months.
3. The applicant should apply in writing to the secretary of the Society providing a letter (1 side of A4) stating why they should be considered for the award; a 1 page CV; and a supporting letter from their head of department. The applicant should send one copy of the publication that they think definitively describes the research they have carried out in the field of matrix biology.
4. A committee of four (BSMB Chairman, BSMB Secretary and 2 co-opted BSMB members) will review the application and decide on a winner.

Veterinary Investigation Award. Veterinarians who are active in laboratory-based research in matrix biology are invited to submit applications for this award. Submit your application to the Meeting organisers (Professor M. Bayliss, Royal Veterinary College, Department of Veterinary Basic Sciences, Royal College Street, London, NW1 0TU). The deadline for application is 31st October 1999. Applications will be sent for independent review by the meeting organisers, and the recipient will be invited to give a special seminar at the meeting and will be awarded a two hundred pound honorarium and presented with a certificate. The cost of attending the meeting will be met by the Society including registration, reasonable travel costs, accommodation and meals.

Guidelines for applicants for Veterinary Investigator Award

1. The applicant should be 35 years or under on the date of the presentation of the award.
2. The applicant should apply in writing providing four copies of the following: a letter (1 side of A4) stating why they should be considered for the award; a 1 page CV; and a supporting letter from their head of department. The applicant should send four copies of the one publication that they think definitively describes the research they have carried out in the field of matrix biology.

Other meeting announcements

Further details of the Bone and Tooth Society Annual Meeting on June 22nd-24th 1999 to be held at the University of Bristol is appended. This meeting has been designed to appeal to all scientists and clinicians with an interest in the skeleton and its disease. Please have a look to see if it interests you.

Report on the British Connective Tissue Society Meeting, Leicester University, 21st-22nd September, 1998

Leicester University hosted the Biochemical Society Glycobiology Group/BCTS joint colloquium entitled, 'The Biology of Hyaluronan' on the main campus. Up to 100 delegates from all corners of the world registered for the meeting. Speakers were invited from laboratories in the UK, USA, Sweden, Finland, Canada and Germany. The meeting was organised by Professor Tim Hardingham and Dr. John Sheehan from The

Wellcome Trust Centre for Cell Matrix Research, Manchester. Financial support for the Symposium was provided by Arthritis Research Campaign, Seikagaku Corporation, Biomatrix Inc., Pfizer and Zeneca.

The scientific programme was divided into sessions describing (1) the bacterial and mammalian hyaluronan synthase (HAS) family (2) the biosynthesis of HA (3) the structure and biophysical behaviour of HA (4) biomedical applications of HA (5) interactions of HA with the HA binding protein, TSG-6, and the HA receptors, CD44 and RHAMM, in the role of HA in the regulation of cell differentiation during embryogenesis and in extracellular matrix organisation.

Paul Weigel (Oklahoma, USA) presented data on hyaluronan synthases from *Streptococcus pyogenes*. Radiation induced inactivation of the bacterial HAS predicted a higher molecular weight for the active species than would be expected from the amino acid sequence. Bovine cardiolipin could stimulate bacterial HAS activity and was shown to be important for HAS function. A pore consisting of HAS protein and 16 cardiolipin molecules (responsible for the extra mass in the inactivation experiments) was put forward as a possible model for the membrane bound complex.

Andrew Spicer (Davis, USA) continued the discussion of the HAS family focussing on mammalian HAS1, 2 and 3. HAS cDNA expression in transfected mammalian cells indicated different catalytic activities for the 3 enzymes and a much shorter HA chain length synthesised by HAS 3, which was independent of substrate concentration. All three HAS genes had been inactivated by gene targeting to produce HAS1, 2 or 3 deficient mice. HAS 1 and 3 null mutants appeared normal, whereas the HAS 2 knockout was lethal at around embryonic day 10 of development. Embryos had a very poor blood supply, due to inadequate yolk sac blood vessel development, and massively enlarged hearts and pericardial sacs.

In the following session **Evi Heldin** (Uppsala, Sweden) discussed how TGF- β , PDGF and TPA affected the regulation of HA-synthases. All three agents stimulated HA synthesis in mesenchymal cells. In mesothelial cells and human lung fibroblasts, PDGF BB upregulated the steady state mRNA level for HAS 2. HAS 1 expression monitored by in situ hybridisation was strong in the epithelium of skin. Some additional signal was seen in the papillary dermis following burn injury, and in inflammatory cells and around sweat glands and blood vessels in healing skin.

Vince Hascall (Cleveland, USA) then outlined the aetiology of inflammatory bowel disease which leads to a loss of structural organisation of colonic crypts. Using an assay where leucocyte adhesion to the pericellular coat of smooth muscle cells was measured it was shown that TNF α and Poly I:C (a synthetic dsRNA used as a virus mimic), could increase leucocyte binding. The TNF induced binding could be inhibited by an anti VCAM-1 antibody indicating a probable VCAM - integrin binding. The Poly I:C induced binding was not inhibited by this antibody, but was reduced with a hyaluronidase digestion. This binding was probably mediated through CD44 (leucocyte) and increased by upregulating the HAS-2 system (smooth muscle cells). A separate investigation of mouse cumulus oocyte cells using quantitative PCR demonstrated that the copy number of HA-synthases was comparatively low at 50 per cell reaching a maximum of 350 per cell during expansion of cumulus cell layer matrix.

The role of inter-cellular HA in the epidermal compartment was then discussed by **Markku Tammi** (Kuopio, Finland) using results from a human keratinocyte culture model. The half-life of HA in the epidermis was shown to be comparatively short at 1 day. Displacement of the HA network could be achieved by incubating tissue with HA decamers but not hexamers, although the maximum displacement was only 50%, and 2 pools of HA were proposed. Quantitative microscopy showed that some anti-CD44 antibodies could reduce the proportion of bound HA in the epidermis, suggesting a role for this receptor in stabilising hyaluronan pericellular networks.

The first afternoon session focused on the biophysical aspects of hyaluronan behaviour. **Tony Day** (Oxford, UK) gave an overview of the hyaladherin family and its common structural domain, the link binding module. Data from isothermal scanning calorimetry demonstrated that HA oligosaccharides of octamer size or larger bind recombinant link module with a K_d of 0.3 μ M. Mutation studies showed 4 amino acids were critical to this binding and that the fully folded protein gave maximal binding efficiency. Further NMR structural studies showed the binding surface to be conserved. Binding was also shown to be pH sensitive and mechanisms for the inhibition of aggrecan aggregation during the inflammatory response were discussed.

John Sheehan (Manchester, UK) then reminded the audience of fibre X-ray crystallography studies which showed the counterion dependence of HA helical structural

arrangements. He then presented a new computational molecular modelling approach to understanding the role of intra-chain hydrogen bonding and the interaction of water molecules with hyaluronan and demonstrated that although stiff, hyaluronan has comparatively high intra-chain mobility over short time scales.

The hydrodynamic and network properties of higher molecular weight HA and aggrecan, measured using the confocal-FRAP technique, were then compared by **Tim Hardingham** (Manchester, UK) and related to their structural properties. The relative contributions of electrostatic repulsion and hydrogen bonding to the intrinsic chain stiffness of hyaluronan was measured in experiments at high pH and the impact of hydrogen bonding was shown to be more important. Divalent counterions, especially Ca^{2+} , were also able to significantly further de-stiffen hyaluronan.

In the special lecture that followed **Andre Balazs** (New Jersey, USA) introduced the audience to some of the medical applications of hyaluronan and cross-linked hyaluronan gels, including viscosupplementation, viscoprotection and viscoaugmentation. The importance of using non-inflammatory material with the minimum proportion of protein contaminants was emphasised, as was ensuring that the molecular weight chosen matched the property required of the gel or solution. The pharmaceutical action of hyaluronan in viscosupplementation studies was then postulated to arise from the temporary return of correct joint articulation which gives an opportunity for the joint to restore homeostasis.

Andrew Pitsillides (London, UK) discussed the role of HA in joint cavitation using embryonic chick diarthrodial joints as a model system. Data were presented demonstrating that effective joint cavitation involved co-ordinated changes in synthesis of HA, CD44 expression and molecules involved in cytoskeletal assembly. Manipulation of joint cavity development, using HA oligosaccharides or immobilisation, disrupted joint formation, decreased HA synthesis and expression of CD44 and actin filaments. Movement-induced stimuli played an essential role in both formation and maintenance of the joint cavity.

Gary Douthwaite (Cardiff, UK) gave the first of four short presentations. He continued with the theme of the role of movement-induced stimuli in joint cavitation, showing changes in the phosphorylation status of CD44 and cytoskeletal organisers when developing joints were placed under strain.

Paul Noble (Yale, USA) described the differential biological properties of high and low molecular weight HA on the regulation of mouse alveolar macrophage activation. Low molecular weight HA (<200,000 Da), but not high molecular weight HA, induced the expression of several genes including TNF α , IL-1 β , VEGF, IL-12 and iNOS. Anti-CD44 monoclonals abrogated HA fragment-induced gene expression.

David Jackson (Oxford, UK) described a novel 80 kDa HA receptor on human lymph vessel endothelium (LYVE-1) which shares 30% sequence identity with CD44. Discovered by homology searching expressed sequence tag databases, recombinant LYVE-1 was shown to bind HA in a solid phase plate assay. A polyclonal antiserum was prepared and used to screen tissue for LYVE-1 distribution. LYVE-1 was expressed in large amounts on lymphatic vessels suggesting a role for this HA receptor in the transport and clearance of HA from the lymphatic system.

Rod Levick (St. Georges, London, UK) discussed the function of HA in the synovial cavity and its role in determining the flow and diffusion of fluids and solutes. In-vivo kinetic studies showed hyaluronan was selectively maintained in the joint cavity, probably through the action of a molecular sieving mechanism. Hyaluronan also acted as a flow limiter, resisting the efflux of water through the synovial membrane, probably because of concentration polarisation. A mathematical model was presented which appeared to accurately describe many of the experimental observations.

Eva Turley (Toronto, Canada) concentrated her presentation on HA binding molecules that do not contain the link module binding motif, in particular the molecule RHAMM. The complicated splice variant pattern encoding multiple protein forms of RHAMM could vary greatly with cell culture conditions. Two receptors were found for RHAMM isoforms that encode an alternatively spliced exon 4; RHAMM and RHAMMv4. RHAMM occurs both on the cell surface and in the cytoplasm. Epitope tagging experiments showed that RHAMMv4 occurs only in the cytoplasm. RHAMM and RHAMMv4 are involved in the regulation of extracellular-regulated kinase (ERK) activity. Over expression of RHAMMv4 enhances expression of ERK.

Cheryl Knudson (Chicago, USA) described the role of HA in chondrocyte pericellular matrix assembly. HA hexamers or CD44 antibodies could inhibit chondrocyte matrix assembly. Pericellular HA, but not CD44, could be removed using *Streptomyces* hyaluronidase. CD44 was shown to

be localised predominantly to the cytoskeleton suggesting a role for intracellular signalling in chondrocyte-matrix interactions.

Hans-Georg Wisniewski (New York, USA) gave an update on the structure and functions of TNF-stimulated gene 6 (TSG-6). The TSG-6 gene encodes a 28 kDa polypeptide which is secreted as a 35 kDa glycoprotein. There are two structural domains; an N-terminal link module with HA binding activity and a C-terminal CUB domain. The TSG-6 gene can be induced 3-4 h after exposed to inflammatory cytokines such as IL-1b or TNFa. Recombinant TSG-6 exerted anti-inflammatory effects in a murine air pouch model of inflammation. Link module mutants, prepared using site-directed mutagenesis, abrogated anti-inflammatory properties of TSG-6. TSG-6 forms a stable complex with the plasma protein inter- α -inhibitor (I α). The function of TSG-6/I α remains unclear.

Helmut Ponta (Karlsruhe, Germany) described the complex variants of CD44 and how CD44 / HA interactions may be important in haematopoietic differentiation. Long term bone marrow culture experiments suggested that HA was important for myelogenesis in this system, possibly as a result of HA stimulated production of IL-6 by bone marrow macrophages, although it was still effective in CD44 knockout mice.

Report on the XVIth Meeting of the Federation of the European Connective Tissue Societies (FECTS) 1st-6th August 1998, University of Uppsala, Sweden by Alison Reith

The recent XVIth FECTS meeting was held in the picturesque university town of Uppsala in Sweden. The opening lecture and plenary lectures were given in the Main University Building, which was located within the heart of Uppsala, while the poster sessions and workshops were conducted in the new Biomedical Centre on the edge of the city.

The meeting opened in the majestic main auditorium of the Main University Building, with its high domed gilded painted ceilings dating from 1887. We were given a historical introduction to the University of Uppsala which, since its foundation in 1477, has produced an environment for the advancement of science through people such as Carolus Linnaeus, Anders Celsius and Anders

Ångström and has been the home of several Nobel Prize winners.

The opening lecture was given by **Erikki Ruoslahti (USA)** who addressed the question of how normal cells find their specific location in the body, while tumour cells locate less specifically. Phage techniques were used to isolate peptides which were capable of homing to the vasculature of a range of normal tissues. These peptide motifs, displayed on phage, were injected into mice and their tumour homing abilities assessed. The peptide motifs RGD, NGR and GSL directed the phage to the tumour. The process of tumour homing was shown to be independent of the tumour origin but dependant upon the angiogenic characteristics of the tumour vasculature. These therapeutically important findings showed that coupling the drug doxorubicin to the tumour homing peptides, RGD and NGR, enhanced anti-tumour activity while decreasing toxicity of this drug in mice.

The main programme commenced on Sunday 2nd August with a series of lectures relating to Glycobiology. **Colin Hughes' (UK)** presentation on the Galectin family, described a group of galactose binding proteins. Galectin-3, the focus of the presentation, was detected in macrophages, branching epithelia and kidney tubules. It has an involvement in establishing epithelial polarity and regulating matrix biosynthesis in kidney development. Functioning as both an antagonist and promoter of cell adhesion, galectin-3 bound to integrins, laminin, fibronectin and tenascin. In the kidney, galectin-3 was regulated during development. A polarised distribution was observed which switched from an apical to basal location on epithelial cell maturation. In renal cysts, galectin-3 localised to the luminal apical surface where it activated other adhesion systems via cadherins.

Henric Clausen (Denmark) discussed the biosynthesis of glycoconjugates via glycosyltransferases. The formation of several glycosidic linkages are now known to be formed by multiple homologous glycosyltransferases with multiple enzymes forming the same linkage. Studies focussed on the polypeptide GalNac-transferases which initiate mucin-type O-glycosylation by attaching GalNAc to selected serine or threonine residues. Kinetic studies demonstrated these transferases have specific, as well as overlapping functions. Mucin-type O-glycosylation was shown to have a complex regulation which had a large genetic back up suggesting partial redundancy of the genes.

Markku Jalkanen (Finland) described structural variations of the proteoglycan syndican-1 which was shown to be dependant upon the location of the side chains along the core protein. These side chains also modulated syndican's role as a co-receptor. The syndicans were shown to undergo signal transduction following complex formation with cell surface receptors. The expression of syndican was controlled within developing tissues and differentiating cells. The recently identified FGF-inducible response element (FiRE) was described, which was shown to be activated by FGF in mesenchymal cells. FiRE was also implicated as responsible for upregulating syndican-1 in keratinocytes during wound healing and tissue regeneration

The proteoglycan theme was continued by **Arthur Lander (USA)** who discussed the role of the glypican heparin sulphate carrier proteins in the developing brain. Glypican 1 and 2, and syndican-3 were expressed in the brain neuron in both developing and adult tissue. Conserved protein domains within glypican-1 regulated glycosylation and directed the polymerisation of heparin sulphate but not chondroitin sulphate chains. Data from a slice culture system indicated that chondroitin sulphate proteoglycan (CSPG) could have either an inhibitory or stimulatory effect on axon growth and cell adhesion. This effect was dependant upon the location within the brain. CSPG was proposed as responsible for organising the presentation of molecules by the ECM in the developing brain.

The first day of the meeting concluded with poster sessions and workshops on Proteoglycans, Cell-cell and cell matrix adhesion and Tissue regeneration and repair.

Monday 3rd August began with a series of lectures associated with Developmental biology.

Liselotte Fessler (USA) introduced us to the ECM proteins tigrin and papilin found in the *Drosophila*, which are not detected in vertebrates. ECM was detected early on in *Drosophila* development, prior to tissue specification. Laminin, papilin and tigrin, in association with integrins, separated the mesoderm and ectoderm. These proteins regulated cell migration and differentiation. The basement membrane in *Drosophila* acts as a covering by surrounding nerve cord, brain, gut and muscles and is not functional in determining which cells form specific organs.

Charles Streuli (UK) discussed the importance of the basement membrane (BM) in mouse mammary epithelial cells for maintaining cellular phenotype, regulating differentiation and apoptosis and controlling cell signalling pathways.

Association of prolactin with its receptor, induced Jak2 protein tyrosine kinase activity and activated the Stat5 transcription factor in this mammary epithelial cell system. BM was shown to be required for prolactin-dependant transcription of milk protein genes via the activation of Stat5 and directly affected growth factor signalling and differentiation at the plasma membrane. Apoptosis in cultured mammary epithelial cells was inhibited in the presence of BM mediated through the $\alpha6$ and $\beta1$ integrin subunits. Insulin signalling was shown to be regulated by cell-matrix interactions in mammary epithelia.

The *in vivo* function of the stable complex formed between laminin and nidogen-1 in basement membrane assembly and in embryogenesis was the topic of the presentation by **Ulrike Mayer (Germany)**. Nidogen-1 was shown to bind to the laminin $\gamma1$ chain via a single module $\gamma1III4$. Knockout mice, in which the nidogen-binding $\gamma1III4$ module of the LAMC1 gene had been deleted, did not survive at birth. The majority of these animals had no kidneys and those which had one or two kidneys present were pathologically abnormal. Small lung size and reduced epithelial branching was observed in the knockout mice. Further characterisation of this mouse model is ongoing but the preliminary studies have shown that laminin requires nidogen-1 binding for both organ development and survival.

Doris Wedlich (Germany) discussed the role of β -catenin in *Xenopus* development. β -catenin binds to cadherins and mediates cell adhesion. It can also transduce the Wnt/Wg signal to target genes. The Wnt/Wg signal modified the mesoderm in *Xenopus* development. Prior to mid blastula transition it had a dorsal function whereas after mid blastula transition it had a ventral function. The mesenchymal genes cadherin-11, $\alpha3\beta1$ and fibronectin were also regulated by the Wnt/Wg signal. Wnt/Wg appears to regulate cross-talk during cell-cell and cell-substrate adhesion.

The day concluded with poster sessions and workshops on Collagens, Cartilage, and ECM contacts and cell signalling.

Tuesday 4th August got underway with a series of lectures on Matrix Receptors. **Michael Henry (USA)** described the role of dystroglycan in early development. Dystroglycan has the ability to bind to numerous sites in laminin leading to a wide variety of potential complexes. The dystroglycan knockout mouse was found to die at the embryonic stage. Studies were also performed using dystroglycan null embryonic stem cells.

Dystroglycan was found to be essential for early development beyond the egg cylinder stage, being required for kidney morphogenesis, laminin binding and basement membrane formation. Collagen IV, perlecan, laminin and nidogen all showed disrupted localisation patterns.

Tero Pihlajaniemi (Finland) reported findings which showed that type XIII collagen was located at the focal adhesions in cultured fibroblasts and at the myotendinous junctions and intercalated disc on human and mouse tissue. Mutant type XIII collagen in transgenic mice was embryonically lethal. It was postulated that type XIII collagen may be involved in cell adhesion to the ECM or act as a receptor for soluble ligands.

Spliced variants of the $\beta 1$ integrin were shown by **Guido Tarone (Italy)** to have important functional properties. The $\beta 1B$ isoform prevented cell adhesion, cell spreading, focal adhesion, fibronectin matrix assembly and epithelial cell organisation while the $\beta 1D$ isoform was a potent adhesive receptor which associated with the actin cytoskeleton and was actively involved in ECM assembly. Tyrosine phosphorylation was also identified as important for the organisation of adhesion molecules. Antibodies against integrin disrupted actin organisation in endothelial cells. Integrin mediated signalling pathways resulted in actin cytoskeleton organisation and regulated matrix-cytoskeleton linkage.

Arnoud Sonnenberg (The Netherlands) continued the theme of integrins in his presentation which discussed the role of the $\alpha 6\beta 4$ integrin in hemidesmosomes, which adhere epithelial cells to the basement membrane. The $\alpha 6\beta 4$ binds to laminin-5 in the ECM and to plectin, which binds directly to the keratin 5 and 14 within the cell cytoskeleton. $\beta 4$ knockout mice showed blistering and skin detachment and lacked hemidesmosomes. $\beta 4$ was shown to be responsible for the localisation of HD1/plectin and BP180 in hemidesmosomes. Studies involving the yeast hybrid system showed the actin binding region of plectin to be involved in binding $\beta 4$ as well as direct binding between BP180 and $\beta 4$. Parallel studies on patients with junctional epidermolysis bullosa showed a decreased number of hemidesmosomes and a lack of $\beta 4$ integrin.

Delegates were given the rest of the day at leisure to explore the delights of Uppsala and its surroundings.

Wednesday 5th August commenced with a series of lectures associated with Genetic Disorders. The presentations commenced with **Jacky Bonaventure (France)** who described

skeletal disorders caused by mutations in FGF receptors. Crouzon syndrome, which results in facial and skeletal deformations, showed abnormalities in both the FGFR1 and FGFR2. Dwarfism was caused by mutations in the FGFR3 gene while craniosynostosis syndromes were caused by mutations in FGFR1, 2 and 3. FGFR1 and 2 were detected at the onset of bud formation in the foetus while FGFR3 was expressed later in development in mature chondrocytes. Thanatophoric dwarfism chondrocytes showed increased apoptosis with both MAPK and STAT pathways activated in these cells.

Eva Engvall (USA) gave a presentation on *in vitro* models for muscular dystrophy. Congenital muscular dystrophy shows an abnormal basement membrane as an effect of defects in the laminin $\alpha 2$ subunit. Laminin $\alpha 2$ has a role in cell survival and provides structural support for contracting myocytes in differentiating embryonic stem cells. Transgenic mice showed abnormal muscle histology and increased paralysis in their hind legs and essentially showed muscular dystrophy and extensive fibrosis. The parallels between the mouse model and the human disease make it suitable for assessment of future treatments for human muscular dystrophy.

Karl Tryggvason (Sweden) told us about the newly identified transmembrane protein nephrin, which has been identified in the glomerular basement membrane in the kidney. The leakage of proteins across the glomerular basement membrane into the silt diaphragm is documented in renal diseases such as nephrotic syndrome. Nephrin was immunolocalised to the podocytes in the silt membrane and is a major constituent of the glomerular basement membrane. It appears to play an important role in the glomerular size-selective filtration barrier and it is likely that it is present in other diseases which have proteinuria.

The fourth afternoon comprised poster sessions and workshops on Diseases of the ECM, Basement membranes and Metalloproteinases and their inhibitors. The conference banquet was held in the main hall of Uppsala Castle where we enjoyed an evening of traditional Scandinavian food and entertainment.

The final day of the meeting began with a series of lectures on Pathophysiology.

Harald Burkhardt (Germany) presented data on a collagen-induced arthritis as a model for rheumatoid arthritis. Collagen-induced arthritis was shown to be associated with T-cell recognition of collagen II peptides. This model was dependent upon collagen II existing in its native conformation

at immunisation and upon the presence of functionally active B cells. A panel of recombinant chimeras of collagen II were used to identify the epitopes which reacted in arthritic mice. Immunodominant domains were numerous, highly organised and spread along the length of the collagen molecule. These studies provided an insight into the autoimmune response to collagen II in joints.

Rolf Reed (Norway) described the exchange of fluid across the capillary epithelial and the resultant pressure created across the ECM. This interstitial fluid pressure (Pif) was studied during edema formation in burn injuries and in an experimental rat model where dextran was injected. In both these situations the Pif decreased. Blocking cellular responses to ECM using antibodies against $\beta 1$ integrin or $\alpha 2\beta 1$ integrin also decreased the Pif. Tissue swelling and the contractile force exerted by the ECM were in equilibrium, when the contraction process was blocked by integrin antibodies, swelling occurred and this was balanced by a further decrease in Pif. Incorporation of PDGF into this system also decreased the Pif.

The penultimate presentation by **Christer Betsholtz (Sweden)** described the role of PDGF in development. PDGF activates cell signalling via receptors PDGFR α and β . Knockout mice for PDGF-A, PDGF-B, PDGFR α and PDGFR β were lethal at either the embryonic or early neonatal stages of development due to defects associated with the mesenchymal cells. PDGF-B and its receptor were required for the development of smooth muscle and vascular endothelial cells and played an important role in cell migration. PDGF-A and its receptor were required for the development of smooth muscle cells associated with epithelial cells in the lung and intestine.

Gerard Karsenty (USA) spoke on osteoblast differentiation during bone remodeling. Osf2, a recently identified protein which binds OSE2 in the osteocalcin promoter, was shown to be expressed in the developing skeleton. During mesenchymal condensation it was restricted to osteoblastic cells. Osf2 regulated the expression of many genes expressed by osteoblasts. Osf2 was found to act as an osteoblast-specific transcription factor and as a regulator of osteoblast differentiation.

The meeting concluded with poster sessions and workshops on Inflammatory diseases of the joints, Structure and function of ECM and Development.

The FECTS meeting was well supported with 594 participants, comprising 50 delegates from the UK.

The Business Meeting the FECTS was held on Wednesday 5th August which discussed the successes and failures of the current meeting. The only criticism was that posters were displayed for only one afternoon, on the same day as the appropriate workshop, giving insufficient time for viewing as poster sessions were held in a separate location from the plenary lectures. It was felt that meeting had been well organised and had been a success. Presentations were heard from representatives from the Italian and Greek Connective Tissue Societies respectively as possible venues for the next FECTS meeting in 2000. This was put to a vote and the venue in Greece was duly selected.