

Connective Issues



BSMB Newsletter

Committee:

Prof. Ray Boot-Handford (Chairman), Prof. Andrew Pitsillides (Secretary),
Dr. David Young (Treasurer), Prof Jo Adams, Dr Emma Blain, Dr. Sarah Howat,
Dr Philippa Hulley, Ms. Eleanor Jones, Dr Chris Murphy, Dr. Mandy Plumb and
Dr Tonia Vincent

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Editorial

Dear BSMB Members

Welcome to the 79th Connective Issues and my second as Honorary Secretary. Having indulged myself in the last Newsletter with some dictionary definitions of 'honorary', I will keep the messages herein simple. There are essentially three.

First, there are nominations required. So on your behalf, I would like to thank our Chairman, Prof. Ray Boot-Handford, for the last 3 years of faultless service to the BSMB. He has been a model leader of the BSMB and our Committee throughout his tenure, so much so that it seems premature to be thanking him in this way. He has shown great care in leading the BSMB but in accord with our procedures; consider the Chairman position of the BSMB executive Committee vacant for nominations. In double-quick time, however, I am extremely happy on behalf of the Committee to nominate Ray to serve for a 2nd term. I am, of course, happy to receive alternative nominations from members.

I'd also like to say a big thank you to Dr. Mandy Plumb for the 3 years she has given the BSMB Committee and for her full role in organising our forthcoming Meeting in Oxford 2012. Thanks are also extended to Eleanor Jones, our outgoing PhD Committee member for giving us an informed and invaluable young perspective. Nominations (below) for these Committee vacancies are also sought.

Second, we organise great meetings. I'd like to thank Dr David Young and Prof Drew Rowan for hosting a fantastic Autumn BSMB 2011 meeting. They constructed a vibrant programme with clear focus in each of the sessions and at the same time managed to create sufficient opportunity for our younger members to show that the future of the BSMB is extremely bright.

Third, register - if you haven't already - for the autumn 2012 meeting in Oxford. Philippa Hulley and Mandy Plumb have, with Liliana Schaefer and John Couchman, put together a smashing programme for a joint meeting with our colleagues from the German Connective Tissue Society – one not to miss.

Andy Pitsillides,
Honorary Secretary

Chairman's Letter

Dear Fellow Matrix Biologists,

Firstly let me wish you a happy, prosperous and scientifically productive New Year. 2012 is an exciting year for Matrix Biology and the Society. In April, we hold our Spring meeting in Oxford (see below for details). This year, the meeting is jointly organised with the German Connective Tissue Society which will be celebrating its 25th Anniversary. The programme is a day longer than normal to allow for both greater interactions between attendees from the two Societies and the sheer number of outstanding presenters assembled. The Spring meeting also includes the BSMB Fell-Muir Award for 2012 which will be presented to Professor Roger Mason. We all look forward to Roger's lecture and the celebrations that will undoubtedly follow! Later in 2012, FECTS will be held in Poland; it's normally held in July but, due to a clash with the European Football Championships, it has been moved this year to late August. A knock-on consequence was our Committee's decision that we would not hold our usual early September meeting and instead, we would throw our full support behind FECTS. I remind all members that we have substantial bursaries available for the Oxford BSMB meeting and up to 10 more of £500 to support members presenting at FECTS.

As always at this time of year, our thoughts turn to the AGM which will be held at Oxford. I will have completed 3 years as Chairman of the Society. It has been an honour to serve as Chairman and I thank the Committee for their nomination for me to serve a second term. Of course this does not preclude any other member from also standing. Committee members completing their terms include Mandy Plumb, Chris Murphy and Tonia Vincent. Chris and Tonia will be co-opted back to the Committee to enable them to fulfil their commitments to organising future BSMB meetings. Mandy completes her term with the Oxford meeting and in this regard we thank her for her efforts and for looking after bursaries for the past 2 years. Finally, we have lost our postgraduate representative, Eleanor Jones, currently finishing her PhD at University of East Anglia. Since January 2010, Eleanor has played an active role on the Committee representing research

students' views. We thank Eleanor for all her efforts and look forward to welcoming her replacement. Nominations are called for later in the newsletter.

With best wishes for the New Year and hoping to be greeting as many members as possible at the Spring meeting.

Ray Boot-Handford

BSMB News

Call for Nominations

BSMB Chairman

BSMB Committee member

BSMB PhD student member

The current BSMB Chairman, Professor Ray Boot-Hanford, completes his term of office at the April 2012 BSMB. It has been the BSMB's Committee decision to ask Prof Boot-Handford to stand for re-election to serve a 2nd term. In accord with our procedures, however, the Chairman position is vacant and we welcome nominations.

The BSMB is also seeking to fill one place on its Committee that will become vacant in April 2012. The usual term of office is three years.

We are also seeking a representative from our PhD student members.

For all of the above please see below for more details.

Fell-Muir Award

BSMB Fell-Muir Award for 2012 will be presented to Professor Roger Mason (sponsored by the International Journal of Experimental Pathology).



Dr Rachel Oldershaw receives 2011 Young Investigator Award from Prof. Ray Boot-Handford (left) and Dr Graham Riley after her lecture at the Newcastle meeting 2011.

2011 BSMB Young Investigator Award to Dr Rachel Oldershaw

Dr Rachel Oldershaw (Newcastle University) receives the Prize at the autumn 2011, meeting. The prize is awarded to a Young BSMB member for outstanding contributions to matrix biology

Bristol BSMB Abstracts 2011 are now published in International Journal of Experimental Pathology (Vol 92, Issue 6) who we acknowledge for continued support. Link: <http://onlinelibrary.wiley.com/doi/10.1111/j.1365-2613.2011.00780.x/abstract>

Welcome to New Members!

We welcome the following to the BSMB:

Full members

Dr Marta Radwan (Newcastle), Dr Cecile Bascoul-Colombo (Cardiff), Dr Anastasios Chanalaris (Oxford), Dr Sheona Drummond (Manchester), Dr Afsie Sabokbar (Oxford), Miss Tarangini Sathyamoorthy (Imperial College London), Miss Arundhati Dongre (Oxford), Dr Mittal Shah (Royal Veterinary College London), Dr Andreea Pataki (Copenhagen),

Student members

David Wilkinson (Newcastle), Basim Alshammari (Imperial College London), Clare Thompson (Queen Mary, London), Livia Henderson (Glasgow), Lorna Mullan (Manchester), Chloe Lallyett (Manchester), Sarah Farrar (University of Manchester), Emma Hagberg (BMRC), Anneke Kramm (Oxford), Hooi Ching Lim (Copenhagen), Ms Anette Høye (Copenhagen), Sadia Haq (Manchester), Betina Fogh (Copenhagen), Marie Morgan-Fisher (Copenhagen).

We hope membership is fun and rewarding.

Subscription reminder

Please keep up to date with subscriptions. An up to date membership list are clearly helpful in our budgeting for the future - so please pay your subscriptions if they are due.

If you have any news, comments or events that you'd like shared with members please send details to apitsill@rvc.ac.uk.

Mark your diary!

- * BSMB Meeting - Oxford, 2nd - 4th April 2012
(with German Connective Tissue Society)
Matrix Molecular Biology:
Integrator of Tissue Function and Disease
Registration now open www.bsmb.ac.uk
 - * University of East Anglia, 3 - 4th Sept 2012,
Tendinopathy: basic science to treatment
 - * 23rd FECTS and ISMB Joint Meeting,
Katowice, Poland August 25th - 29th, 2012
 - * ASMB 2012 - San Diego, USA. 11th - 14th
November, 2012
 - * BSMB Meeting - Oxford, 25th - 26th 2013
Epigenetics in Matrix Biology and Disease
-

Request for Nominations

BSMB Chairman

The Chairman (Chair) is responsible for the organisation and running of the BSMB, and together with the Committee formulates policy, represents the Society as appropriate in interactions with other bodies, and promotes our mission and objectives. The Chair shall not normally hold that office for a term of more than three years. In any case, the Chair shall not hold that office for more than six consecutive years, but they shall be eligible for election to any other office in the Society. The term of office will begin at the April 2012 BSMB meeting. It is expected that the Chair will attend a very high proportion of BSMB Committee meetings and conferences through his/her term of office and the April 2012 meeting to take office.

Nominations for Chairman of the BSMB can be made by any 2 members of the BSMB, or members of the current BSMB Committee. Nominations, together with written consent of the proposed nominee, should be forwarded to the Honorary Secretary, Professor Andrew Pitsillides, by **Wednesday 29th February 2012** by e-mail to apistill@rvc.ac.uk

In the event of more than one nomination, a ballot of membership will be held by e-mail. To this end, it would be helpful if nominees can send a brief (one page) CV together with a short statement outlining their aspirations

for BSMB. In the event that no nominations other than the Committee's (Prof Ray Boot-Handford) are received, the Committee will make this appointment to the Chair.

BSMB Committee member

Any current member in good standing is eligible for nomination. Committee members formulate policy for the Society and have a major responsibility for organising BSMB meetings. It is expected that each Committee member will take a major part in planning and organising one BSMB meeting during their tenure. Committee members should be available to attend most Committee meetings that coincide with our conferences, but also attend occasional further meetings.

We encourage Society members to become involved in the future of BSMB. Should we receive more than one nomination, an election, in line with our Constitution, may be held. Nominees should send a CV and supporting statement to the Secretary, Andy Pitsillides. Appointees become BSMB Trustees and it is recommended that those interested read the BSMB Constitution and trustees responsibilities (www.bsmb.ac.uk).

BSMB PhD student member

BSMB Committee includes representation from PhD student members. Past student Committee members have made valuable contributions. As our current representative prepares to move to the next stage of her career, we now seek their replacement. Any PhD student member of BSMB is eligible for a one year term, from April 2012. The successful applicant will normally attend BSMB Committee meetings and occasional meetings held at other times. Nominees should send a short statement and CV to the Secretary, together with a statement from the student's supervisor agreeing to the student's participation on the Committee. To avoid an election, the first appropriately qualified nominee to e-mail the required information will be appointed.

Closing date for all posts is 29th Feb 2012.

Informal enquiries to the Secretary, Andy Pitsillides by e-mail (apitsill@rvc.ac.uk) or phone (207 468 5245).

Andrew Pitsillides, BSMB Sec., Jan 2012

BSMB 2012 Spring Meeting
2nd - 4th April 2012

**Joint meeting of the British Society
for Matrix Biology and the 25th
Anniversary Meeting of the German
Connective Tissue Society (DGBF)**

***'Matrix Molecular Biology:
Integrator of Tissue Function and
Disease'***

St Catherine's College, Oxford

Organised by

**Philippa Hulley, Mandy Plumb, Liliana
Schaefer and John Couchman**

Our 3-day Spring 2012 meeting is the only meeting of the BSMB in 2012 and covers the broad field of current fundamental research in matrix & connective tissue biology. Speakers are drawn from UK, Germany and beyond with a wide range of interests including vascular biology, stem cells, musculoskeletal sciences and disease processes in cancer, inflammation and fibrosis.

Highlight of the meeting will include the Fell-Muir Award and the German Society Young Investigator award. The conference dinner is on Tuesday 3rd April at St. Catherine's with an after-dinner jazz band in the College Pub and Common Room. Additional oral presentations in all sessions will be selected from submitted abstracts

Monday 2nd April

Session 1: Mechanobiology of the Matrix
Paddy Pendergast (Dublin)

Session 2: Nanotechnology & Adhesion
Clare Waterman (NIH)
Joachim Spatz (Stuttgart)

Fell Muir Award – Professor Roger Mason

Tuesday 3rd April

Session 3: Stem Cells & their Niche
Francesco dell'Accio (London)
Cathy Merry (Manchester)

Session 4: Matrix & Cancer
Kebs Hodivala-Dilke (London)
Yoshifumi Itoh (London)

Session 5: Tissue Injury & Repair
DGBF Young Investigator Award
Dietmar Vestweber (Munster)

Session 6: Wound Healing/Inflammation
Liliana Schaefer (Frankfurt)
Paul Martin (Bristol)

Wednesday 4th April

Session 7: Fibrosis
Oliver Eickelberg (Munich)
George Bou-Gharios (London)

Session 8: Cell Adhesion & Signalling
Reinhard Fässler (Munich)
John Couchman (Copenhagen)

Registration opens on **9th Jan 2012** via the BSMB website for both Societies
www.bsmb.ac.uk

Important: Please note that this is an Oxford Residential Conference and the fee includes everything - 2 nights accommodation, all meals, social events and registration.

Key dates:

Registration opens **9 Jan 2012**
DGBF Young Investigator Bursary deadline
23 Jan 2012
BSMB Presenter and Reporter Bursary
deadline **17 Feb 2012**
Early Bird Registration closes **2 March 2012**
Abstracts close **16 March 2012**
Registration closes **16 March 2012**

Bursaries are available to young investigators from their own society - please visit your website for details.

Fees:

Early registration before 2 March:
£270 for members, £300 for non-members
£210 for student members, £260 for student
non-members
Late registration until 16 March;
£310 for members, £340 for non-members
£250 for student members, £300 for student
non-members

Costs include accommodation and all meals for the duration of the meeting.

Queries to philippa.hulley@ndorms.ox.ac.uk
or mplumb@brookes.ac.uk

We look forward to welcoming you to Oxford

Tendinopathy: from basic science to treatment

(under the auspices of BSMB)

**University of East Anglia, Norwich,
3-4th September 2012**

Organised by Graham Riley

In the absence of an autumn BSMB meeting in 2012, opportunity has been taken to organise a standalone meeting on tendon, to follow on from successful satellite meetings in Cambridge (2006) and Norwich (2010).

The meeting will cover most aspects of tendon biology and pathology, as well as new approaches to treatment, such as gene therapy and the use of stem cells. Confirmed speakers include Professor Chris Evans, Harvard Medical School (gene therapy), Professor Kathe Derwin, Cleveland Clinic (tissue engineering), Professor Paul Ackermann, Karolinska Institute (nerves and tendon repair), and Professor Michael Kjaer, Institute of Sports Medicine, Copenhagen (tendon adaptation to exercise).

The meeting will be held on the UEA campus in Norwich, with a conference dinner in the Sainsbury Centre. Registration fees are likely to be in the region of £80 (£50 for students) and reasonably-priced accommodation will be available on campus. Delegates will be encouraged to submit poster abstracts, some of which will be chosen for short oral presentation.

Further details and a preliminary programme will be presented on the BSMB website early during 2012.

BSMB 2013 Spring Meeting – Oxford, 25-26th March

‘Epigenetics in Matrix Biology and Disease’

Organised by Chris Murphy

Epigenetic regulation, from microRNAs to methylation is of fundamental importance in matrix biology and disease, and this field is set to expand exponentially. Whether actively

involved in epigenetic research or simply keen to learn more, this meeting is for you!

The exciting programme covers diverse aspects of epigenetic regulation relevant to matrix biology and pathology and includes top class international and national speakers. In addition, the distinguished Fell-Muir award and presentation, and the Society's AGM will take place.

Themed sessions include: Arthritis; Musculoskeletal System; Development/Stem Cells; Cancer; in addition to our usual open session.

Speakers include:

Kristian Helin, University of Copenhagen, Denmark; epigenetics and cancer

Karen Steel, Cambridge; microRNAs and hearing loss

Hiroshi Asahara, NRICHD, Japan, and the Scripps Research Institute, USA; microRNAs and skeletal development/disease

Wolf Reik, Babraham Institute, Cambridge; imprinting, epigenetic (re)programming

Ian Clark, University of East Anglia; microRNAs & HDACS in skeletal biology

Patrick Matthias, Basel, Switzerland; Epigenetic networks in development/stem cells

Thomas Pap, Munster, Germany; sumoylation/epigenetics in matrix biology

More details will be made available on the BSMB website shortly.

Conference Bursaries from BSMB

Jo Adams

Young members of BSMB are encouraged to apply for bursaries to assist in expenses associated with attending the Spring 2012 Joint BSMB/DGBF meeting, or other selected meetings, such as those organized by Federation of the European Connective Tissues Societies (next meeting in August 2012), American Society for Matrix Biology (next meeting in November 2012), or other national or international meetings with a matrix biology theme.

Applicants should be non-tenured scientists, preferably at an early stage of their research career and unlikely to have access to travel funds. For this reason, preference is given to graduate students and early post-docs (less than 6 years post-doc). In the case of applications exceeding available bursaries, the quality and impact of the work to be presented together with the previous record of BSMB bursary support will also be taken into account.

Several types of Bursaries are offered:

BSMB Meeting Presenter Bursaries

BSMB offer Meeting Presentation bursaries for young researchers who wish to present their research at BSMB meetings. The research must be relevant to BSMB but need not be on the theme of the meeting. Applications must be accompanied by an abstract of the research to be presented. To qualify for a bursary, an applicant must have been a BSMB member for at least 122 days prior to the opening of registration for the meeting. Successful applicants will be expected to display the BSMB logo on their poster or acknowledgements slide.

Applicants must have joined BSMB before Sept. 9, 2011 to be eligible for a bursary at the Spring 2012 BSMB meeting.

BSMB Meeting Reporter Bursaries

The BSMB offers Meeting Reporter bursaries for BSMB meetings. Applicants undertake to write a report on the talks at the conference for publication on the BSMB website and subsequently in the International Journal of Experimental Pathology. This is an excellent opportunity to develop scientific writing and reporting skills. If desired, reports can be attributed to the individual responsible, enabling the resulting publication to be cited by the author as an example of professional writing. Applicants need not present a poster at the meeting. *To qualify, an applicant must have been a member of the BSMB for at least 4 months (122 days) prior to the opening of registration for the meeting. Applicants must have joined BSMB before Sept. 9, 2011 to be eligible for a bursary at the Spring 2012 BSMB meeting.*

Both BSMB bursaries are for up to £200 to cover conference expenses (e.g. registration, accommodation, conference dinner) and contribute towards travel costs.

Closing date for Bursary applications for the Spring 2012 Joint BSMB/DGBF meeting will be 17th February 2012.

FECTS and ASMB Bursaries

An applicant must have been a member of the BSMB for at least 6 calendar months prior to the start of the meeting and must be contributing a poster or a talk. The work described in the abstract must be novel and of a quality that would reflect well as a BSMB-supported contribution. Successful applicants will be expected to display the BSMB logo on their poster or acknowledgements slide. Funded applicants must participate (ideally with other bursary recipients) in producing a written conference report for posting on the BSMB website. Examples of previous reports are available on the Articles and Reports Pages of BSMB website. *Applications should be made at least 6 months before the start of the meeting. The amount of the award depends on the location of the meeting, and will be up to £500 for FECTS meetings and £600 for ASMB meeting.*

Bursaries to attend other meetings

Bursaries are also available to provide financial support for BSMB members at the beginning of their research careers to attend any meeting relevant to matrix biology. An applicant must have been an active member of the BSMB for at least one year prior to the meeting and have attended one or more BSMB meetings. Applicants must be contributing a poster or a talk. The work described in the abstract must be novel and of a quality that would reflect well as a BSMB-supported contribution. Successful applicants will be expected to display the BSMB logo on their poster or acknowledgements slide. *Applications are considered quarterly. The amount of the award depends on the location of the meeting, and will be up to £150 for UK meetings and £300 for meetings elsewhere in the world.*

Application procedure

Full details on eligibility, application process and necessary forms for each type of Bursary are provided at www.bsmb.ac.uk/bursaries/
Application packs must be sent by e-mail to Prof. Jo Adams, Bursary Committee, jo.adams@bristol.ac.uk

Recently awarded bursaries:

BSMB awarded eight bursaries for the Autumn 2011 Meeting in Newcastle.

Reporter Bursaries were awarded to Dr Sally Dreger (Barts Cancer Institute), Dr Nuor Jamil (University of Edinburgh) and Mr Christos Gavriilidis (Newcastle University).

Presenter bursaries were awarded to Joanna Sherwood (QMUL), Dr Giovanna Nalesso (QMUL), Sandeep Karat (BRIC, Copenhagen), Douglas Dyer (University of Manchester) and Gehan Gibrel (Sheffield Hallam University).

Other meetings:

A bursary was awarded in Oct. 2011 to support attendance of Dr. Chavaunne Thorpe at the Dec. 2011 International Conference on the Mechanics of Biomaterials and Tissues, Hawaii, USA.

BSMB Poster Prizes

Ching-Yan Chloé Yeung (University of Manchester), Youn-Jung Kang (University of Manchester) and Lydia Murray (University of Glasgow) were awarded Poster Prizes, sponsored by the International Journal of Experimental Pathology and judged by the plenary speakers.

- [Poster prize information](#)
-

British Society for Matrix Biology - Spring 2012 Meeting Report

by Sally Dreger, Nuor Jamil & Christos Gavriilidis (reporter bursary recipients)

Matrix signals! Cell-matrix interactions in health and disease

**Organised by Dr David Young and Prof Drew Rowan
hosted by Newcastle University**

The meeting comprised 5 sessions, with 9 plenary lectures, 11 short selected talks (from abstracts) and 4 poster sessions. The BSMB also presented a Young investigator Award to Dr Rachel Oldershaw for her work on "Directed differentiation of human embryonic stem cells toward chondrocytes".

Ching-Yan Chloé Yeung (University of Manchester), Youn-Jung Kang (University of Manchester) and Lydia Murray (University of Glasgow) were awarded Poster Prizes, judged by the plenary speakers and sponsored by International Journal of Experimental Pathology.



The meeting was generously supported by: Nuffield Foundation (sponsored travel and accommodation for Prof Tony Poole and Prof Wim van den Berg along and funding for a large number of attendees); The Company of Biologists and associated journals, Biology Open and Journal of Cell Science (travel and accommodation funds for plenary speakers) and four sponsoring exhibitors Miltenyi Biotec, vhbio, LONZA and Promega.

Session 1 - Matrix mechanical forces

Chairs: Dr Mark Birch and Dr Emma Blain

The session was opened by **Dr Akiko Mammoto** (Harvard Medical School) with an examination of the role of mechanical forces in cell growth, fate and morphogenesis. Dr Mammoto suggested that the cytoskeleton plays key roles during normal developmental and disease by generating mechanical tension that is transmitted across cell-extracellular matrix (ECM) and cell-cell adhesions. She highlighted key roles for the Rho small G protein pathways in such mechanotransduction. She showed that p190RhoGap inhibits Rho activation in strained cells and binds TF-II in the cytoplasm. She then demonstrated that GATA2 breaks this hold and sequesters TF-II to the nucleus in order to activate VEGFR2 expression to control angiogenesis. These transcription factors also antagonise cell migration and tube formation thus regulating angiogenesis via regulating ECM elasticity. To support a role for matrix stiffness in controlling vascular permeability, Dr Mammoto demonstrated that TFII inhibits vascular leakiness in vitro and therefore decreases vascular permeability. She also showed that VEGFR2 does not mediate TFII-dependent control of vascular permeability but that p190RhoGAP does. She concluded by showing a new Lysyl oxidase (LOX) mouse model for studying vascular permeability and ECM structure in the lung.



The first short selected talk (from abstracts) was presented by **Dr Kristian Franze** (Oxford University). His talk addressed the hypothesis that local changes in nerve tissue stiffness influence cell function, migration and growth. Using a *Xenopus* eyeball model system he showed that high tissue stiffness repels nerve axon expansion, via interaction of actin and myosin and intracellular calcium signalling. He demonstrated that high stress and high calcium levels force neuronal retraction. Using atomic force microscopy Dr Franze also showed that retinal ganglion cell axons select to grow along much softer

pathways. After pointing out that chondroitin sulphate disrupts axon pathfinding, he concluded that softening of the local ECM might provide cues for neuron growth in an otherwise stiff environment.

Dr Martin Knight (Queen Mary, London) introduced the idea that primary cilia are involved in ATP and calcium signalling during mechanotransduction in various cell types, and on the basis that these processes regulate anabolic chondrocyte behaviour, he hypothesised that primary cilia are required for chondrocyte mechanotransduction. Dr Knight used cells from wildtype (WT) and Tg737 Oak Ridge Polycystic Kidney (Tg737orpk) mice, a mutation that halts ciliogenesis, and showed that mechanical loading upregulated ATP release, calcium levels, aggrecan expression and sGAG synthesis in WT cells, but only ATP release by Tg737orpk cells. He concluded his talk by suggesting that primary cilia are involved in mechanotransduction but are not just sensors as they control the release of calcium from intracellular stores.



This session was concluded by **Prof C. Anthony (Tony) Poole** (University of Otago) with a historical account of evidence supporting a role for primary cilia in chondrocyte mechanotransduction. He dealt with their localization, structure and interaction with ECM and centrosomes and also described a tomographic model of the chondrocyte matrix-cilium-Golgi continuum. He then highlighted their importance in 3 pathways; Hedgehog, chemosensation and Wnt (with reference to regulation of fluid shear stress and calcium signalling by polycystin 1 and 2 complex) and described a study showing that prolonged cyclic compression results in ciliary shortening and

resorption. The second part of this talk concentrated on an ovine model of Meckel-Gruber syndrome (a human ciliopathy) in which the matrix-cilium-Golgi continuum is disrupted; mutation affects *MKS3* that codes for Meckelin, a protein that localizes in primary cilia and cell membranes and interacts with other MKS proteins to form a complex at the base of the cilium. Amongst other phenotypes *MKS3* mutant chondrocytes exhibited both disrupted cilia and collagen. He concluded by suggesting that chondrocyte primary cilia are complex sensory organelles that interact with their microenvironment like centrosomes and the golgi apparatus to regulate ECM synthesis and secretion in response to physiological loads during all phases of cartilage development.

Young investigator award lecture

Chair: Prof Ray Boot-Handford

Directed differentiation of human embryonic stem cells toward chondrocytes, *Dr Rachel Oldershaw*

This year's Young Investigator Award was presented to **Dr Rachel Oldershaw** for work done in Prof Tim Hardingham's group at the University of Manchester. She gave a brief summary of cartilage structure and function and highlighted the role of chondrocytes in ECM turnover and imbalance in disease. She assessed use of adult and mesenchymal stem cells and the problems associated with their use e.g. delay of removal of tissue and replacement, number of operations required, limited proliferation and bone formation. Subsequently, Dr Oldershaw described studies with human embryonic stem (hES) cells, using feeder-free culture supplemented with growth factors to drive chondrogenesis, in which she monitored gene expression during differentiation to show that pluripotent hES cells progressed through mesendoderm (that can lead to angioblast differentiation) to mesoderm before acquiring a chondrogenic phenotype (SOX9, cartilage specific Collagen II, cell surface aggrecan and matrix sGAG deposition). To conclude, Dr Oldershaw highlighted that this method is more efficient, scalable and can be carried out under fully defined culture conditions but more work has to be carried out in terms of safety and efficacy.

Session 2 - Matrix receptors

Chairs: Prof Drew Rowan & Dr Matt Barter

The afternoon session was opened by **Prof Attila Aszodi** (University of Munich) with an excellent lecture entitled 'Chondrocyte polarity and cartilage function: the role of cell matrix interactions'. Prof. Aszodi highlighted the importance of $\beta 1$ integrin in skeletal morphogenesis and in diseases like chondrodysplasia and osteoarthritis. Cell shape anisotropy maintained by $\beta 1$ integrin provides a default guiding cue for spindle and division positioning in chondrocytes. He showed that 2 polarity levels exist in cartilage growth plate and described how these are established and maintained. The supposed mechanisms behind this are: 1) transcription factors, 2) matrix molecules, 3) soluble factors, 4) cilia and 5) cytoskeletal structure of chondrocytes. $\beta 1$ integrin is pivotal for the flattening and orientation of proliferative chondrocytes with $\beta 1$ -null chondrocytes being rounded, non-oriented, displaying random spindle orientation and division axes and failing to intercalate into columns. In articular cartilage, $\beta 1$ integrin appears not to impact on homeostasis, however, its lack leads to structural changes, which soften the AC surface and increase OA severity. He concluded by exploring links between ECM and cell shape and emphasised the importance of type VI collagen.

The second presentation was by **Dr Jo Sherwood** (Queen Mary, London), about ELR+ CXC chemokine signalling and phenotypic stability of human articular chondrocytes. Healthy articular chondrocytes express their own chemokine receptors and ligands although the functions are poorly understood. CXCR1/2 expression was shown to be lost following differentiation of human articular chondrocytes suggesting a role in phenotypic stability. Other chemokines, CXCL6 and CXCL8, have a distinct ECM localisations in healthy vs. osteoarthritic tissue. Loss of CXCL8 from territorial matrix following heparitinase, but not chondroitinase ABC digestion suggests that it is retained in articular cartilage by interaction with heparan sulphate proteoglycans; which may impact on new treatment.

Dr Susan Taylor (University of Manchester) reported on the role of collagen IV in preventing tendon adhesions. To investigate this, engineered tendons were embedded into fibrin gel and cell migration monitored by time lapse microscopy. Cells from these engineered tendons migrated well, while ex vivo tendon cells did not. She also showed that these cells possess tight junctions composed mainly of collagen IV, laminin, perlecan and nidogen. Using a surgical model to induce adhesion formation in mice she demonstrated that the surface of tendons possessed a layer of laminin, leading her to conclude that tendon is covered by basement membrane epithelium in order to retain cells and to preventing adhesion formation.

Dr Christoph Ballestrem (University of Manchester) concluded this session by sharing work on Vinculin. He described how a large network of focal adhesion (FA) proteins is essential for cells to identify their environment and to regulate bidirectional



outside-in and inside out signals. How these signals are coordinated is unclear but Dr Ballestrem gave us his hypothesis that vinculin was key to these processes. Vinculin is present in two states: folded and unfolded, and using cyclic mechanical stretching he highlighted that vinculin, through its links to the talin-integrin complex and actin, controls the transmission of mechanical forces in cells. Vinculin was shown to regulate integrin activation status and to direct the recruitment and release of the large network of FA components. Mutating vinculin affected the cellular orientation and its speed. Since FAs do not disassemble if vinculin is maintained in an active conformation, it seems that vinculin acts as a mechanotransducer, regulating activity of integrin and other FA proteins.



Session 3 - matrix receptors/sensing

Chairs: Prof Tim Cawston and Dr Gary Litherland

Dr Birgit Leitinger (Imperial college London) spoke about discoidin domain receptors (DDR). DDR1 and DDR2 constitute a subfamily of receptor tyrosine kinases, activated by a number of different collagen types, which are involved in development and different diseases. She described DDR1 structure and identified its specific collagen interaction motif, which differs from the metal ion-dependent integrin binding motif in discoidin. She presented a model for interaction between DDR1, collagen and integrin, and its likely impact on cell processes, such as adhesion, spreading and migration. She concluded that DDR1 functions are not in cell spreading - but that it can promote integrin-mediated cell adhesion - and that a conserved patch in DDR is required for cell adhesion and important in modulating different cellular processes.

Dr Paul Huang (Inst. of Cancer Research, London) also reflected on DDRs but focussed on downstream signalling, highlighting their distinct activation kinetics (delayed and sustained receptor phosphorylation) over several hours after collagen engagement. Mass spec-based phosphoproteomics was used to study signalling downstream of DDR activation by fibrillar collagen and identified >150 phosphorylation sites over 7 time points, one on SHP-2. He demonstrated the importance of DDR2 signalling by showing that SHP-2 is a potent negative regulator of DDR2 receptor phosphorylation, and stated that phosphoproteomics is an exciting method for cell-matrix signalling and signalling in general.

The third talk was given by **Giovanna Nalesso** (Queen Mary, London) on the subject of WNT signalling in chondrocytes. She described Wnt/canonical signalling in osteoarthritis (OA) and its importance in pathology; gain- and loss-of-function both induce OA. WNT-3A participates in chondrocyte differentiation, with low doses triggering intracellular Ca²⁺ accumulation, and high dose activating Wnt/ β -catenin canonical signalling. These pathways were shown to be mutually inhibitory and led to the overall conclusion that their selective modulation could represent a new therapy in OA.

The final talk of this session came from **Dr. Nicola Hunt** (Newcastle University, UK) on ECM control of neural crest cell (NCC) behavior. Congenital heart defects account for approximately 1% of UK births with abnormalities in the cardiac outflow accounting for 30% of these. In this study, Dr. Hunt explained how collagen I and fibronectin affect NCC behaviour. She introduced the talk by discussing the role of collagen I and FN in migration, which is important in acquiring congenital heart diseases. Mouse embryos (E9.5-E14.5) showed abundant collagen I and FN expression and their co-localisation with NCC. In vitro analysis showed that cell behaviour on poly-L-lysine and fibronectin was very similar, whereas behavior on collagen I was markedly different. Migration and smooth muscle differentiation were decreased on collagen I compared with the other two coatings. Expression patterns of collagen I and II by NCCs were also dramatically different on collagen I. Altered composition of the NCC ECM environment may account for some of the outflow tract defects seen in congenital heart disease.

Session 4 – Matrix signalling – fragments

Chairs: Prof Andy Pitsillides & Dr Rose Davidson

Session 4 began with Prof **Suneel Apte** (Cleveland Clinic Lerner Research Institute, USA) describing the structural and functional aspects of the ECM with emphasis on the proteoglycan versican. Versican (four variants, V0, V1, V2, V3), plays an important role in hydration and network formation with fibrillin 1 and fibulin, and is also essential for

matrix development during morphogenesis. He described the ADAMTS group of enzymes (proteoglycanases); a major mechanism for versican clearance in limb, cardiac, vascular and craniofacial development. He showed how a single *Adamts20* mutation produces a white spotting phenotype in mice (belted) and if combined with *Adamts9* or *Adamts1* mutants, a severe spotting phenotype develops with a reduced accumulation of versican in skin. *Adamts5*, *9*, *20* knockout mice have webbed digits, cleft palate and an accumulation of versican respectively. When a recombinant ADAMTS was added to these knockouts, there was an increase in versican proteolysis and reduced phenotype. He elaborated on how the pulmonary artery valve fails to remodel during development due to decrease versican remodelling in *Adamts5*^{-/-} mice; addition of a recombinant ADAMTS again partially rescued the sculpting defect. He also showed ADAMTS5 deficiency in dermal fibroblasts promoting a myofibroblast phenotype due to decreased versican turnover. He concluded by saying ADAMTS proteolysis works in co-operation with versican clearance, and the cleft palate and webbed digit phenotypes suggest that products of versican proteolysis are bioactive. While in the pulmonary artery and in dermal fibroblasts, versican clearance alone is required for these to develop.

Luke Bonser (University of Manchester) introduced polymeric mucins, MUC5AC and 5B, which are intracellular components of the airway, highly condensed in secretory organelles. He described how previous studies reported that MUC5B linearizes post secretion; shown to swell and expands once secreted by high resolution EM. He then asked: 'does MUC5AC unpack in a similar way'. Using conditioned media and agonist-(Anamycin)-stimulated secretions from HT-29 cells, mucin expansion was assessed using rate zonal centrifugation whereby rate of sedimentation is due to mucin architecture. He showed that freshly-secreted mucins are branched and cross-linked but accumulated mucins were linear, suggesting that mucin morphology changes post secretion. He concluded that these findings may have implications for the formation of mucus in chronic airway disease.

Dr. Douglas Dyer (University of Manchester) began by introducing the TSG-6 (TNF stimulated gene 6) which is induced in response to inflammatory mediators and growth factors to act as a protective factor against inflammation. He described how TSG-6 increases in synovial fluid of OA and rheumatoid arthritis patients, resulting in decreased aggrecanase and MMP activity and hence less inflammatory damage. TSG-6 is also a potent inhibitor of neutrophil migration and to demonstrate this, he used the HL-60 cell line in transwell migration assay, to assess the effect of TSG-6 on CXCL8 pro-inflammatory activity. An increase in migration was observed with CXCL8 alone but there was a decrease with TSG-6 alone or with CXCL8. These results prompted studies into the binding mechanism of TSG-6 to CXCL8 via its link module domain (Link_TSG6). Both recombinant TSG-6 and Link_TSG6 inhibited CXCL8-induced neutrophil migration and the link_TSG6 mutants with a reduced heparin binding function enhanced this effect. He concluded his talk by illustrating that TSG-6 binds directly to CXCL8 and inhibits its interaction with heparin and that this is mediated by link_TSG6. This in turn inhibits migration of neutrophils partly due to decreased association with endothelial cell GAGs, limiting pro-migratory activity. This demonstrates how TSG-6 may protect tissue from the damaging effects of inflammation.

The final talk of the session was given by **Dr Kim Midwood** (Kennedy Institute of Rheumatology, Oxford University) about two important ECM glycoproteins; fibronectin and tenascin-C. She described fibronectin structure, its cryptic sites and importance in development, angiogenesis and cell orientation. She highlighted how fibronectin and tenascin-C interact to modulate cellular responses; tenascin-C was injected into embryos and found to quickly localize with fibronectin. She went on to illustrate another study which used a solid phase binding assay to assess fibronectin and tenascin-C interaction. This revealed the ability of tenascin-C fibronectin type III (TNfn) domains 1-8, 1-5 and 3-5 to immobilize binding to plasma fibronectin. Another study used NIH3T3 fibroblasts and cellular fibronectin, to illustrate the TNfn domains had variable

binding affinities to fibronectin. This led Dr. Midwood to ask, whether tenascin-C functionally regulated fibronectin assembly? To address this, she assessed fibroblasts incubated with TNfn domains, c-terminal fibrinogen like globe (FBG) or a 70kDa-N-terminal domain of fibronectin by immunocytochemistry or a DOC soluble assay. She found that tenascin fragments (TNfn) 1-8 interferes with fibronectin-fibronectin/cell interactions while fibrinogen promotes fibronectin disassembly via a cytoskeletal reorganization. She also discussed how full length tenascin-C did not inhibit fibronectin fibrillogenesis but that TNfn domains do, and proposed that tenascin-C cryptic sites can reduce levels of fibronectin assembly and prevent de novo fibril formation. She finished by highlighting the importance of elucidating the complexity of fibronectin:tenascin-C interactions.

Session 5 – Matrix signalling in disease

Chairs: Dr Tonia Vincent & Dr Rachel Oldershaw

The final session of the conference began with a talk by **Prof Wim van den Berg** (RUNMC, The Netherlands), the “Godfather of Alarmins”. He described how the alarmins S100 belong to the DAMPs family and lead a double life; intracellularly they act as a Ca²⁺ shuttle and extracellularly under symptoms of stress, promote inflammation. Importantly, S100A8 and A9 are inflammatory markers involved on neutrophil, monocyte and activated macrophage function which show serum levels that correlate with joint damage in rheumatoid. He explained how alarmins (injections of recombinant S100A8 and polymixin) induced arthritis in FcγR1^{-/-} versus wild type mice by inciting inflammation with an increase in cell infiltrate, enhanced proteoglycan depletion and up-regulation of MMPs (MMP-3, -9, -13, -14) and ADAMTS (-4, -5). He then describes studies which showed that S100A8 and A9 have a catabolic effect on isolated chondrocytes and explants and that this involved a TLR4-dependent pathway; S100A8 stimulated bone resorption was abrogated in TLR4^{-/-} mice. He then spoke about additional studies whereby S100A8 and A9 mediate cartilage destruction in OA. This was demonstrated by comparing two mouse OA models with different

mechanisms of synovial activation. The first model, collagenase-induced OA showed increased mRNA levels of S100A8 and A9 for a prolonged period of time while IL-1 β was only expressed in the early stages of synovial activation. In the S100A8/A9 -/- mice collagenase-induced OA resulted in a marked suppression in synovial activation and cartilage damage compared to the wild type. The second model DMM (destabilized medial meniscus) had lower levels of both synovial activation and up-regulation of S100A8/A9. He concluded his presentation by suggesting levels of S100A8 appear to be predictive marker of erosive OA.

This was followed by a short talk from **Youn-Jung Kang** (Universities of Manchester and Oxford) on interaction of the integrin $\alpha\beta3$ and osteopontin (OPN) mediating embryo attachment in the first stage of implantation. Using implantation assays (either embryos or embryo-sized beads carrying various protein ligands) and siRNA knockdown of $\alpha\beta3$ of human uterine epithelial (Ishikawa) cells, she demonstrated that embryos stably attached onto the cells after 48 hours. Of the protein coated beads, an increase in attachment was observed on HB-EGF>OPN>IGF-1. She explained how there was a local increase in $\alpha\beta3$ expression adjacent to either the attached embryos or beads with IGF-1, HB-EGF and OPN treatment. Knockdown of $\alpha\beta3$ in the Ishikawa cells demonstrated a decreased attachment of beads carrying IGF-1 or HB-EGF. She also demonstrated how treatment with soluble IGF-1 and not HB-EGF cleaved OPN to produce an adhesion active fragment. In the IGF-1 treated cells, $\alpha\beta3$ levels and cleavage of endogenous OPN were suppressed by soluble OPN. She concluded from these studies there maybe two independent pathways, in which $\alpha\beta3$ mediates implantation: $\alpha\beta3$ -OPN-IGF-1 pathway and the $\alpha\beta3$ -HB-EGF pathway.

Peter Bell's (University of Manchester) presentation focused on matrilins, of which there are four. He focused on matrilin-3 which is expressed only in cartilage, binds to collagen and leucine rich proteoglycans and forms heterotetrameric oligomers with matrilin-1 and then described multiple epiphyseal dysplasia (MED); a disease with irregular endochondral bone growth and early

onset of OA which can be due to mutations in matrilin-3. Using a MED mouse model with a Matn3 pV194D mutation, he observed that the majority of mutant matrilin-3 was retained in chondrocytes while some was secreted into the ECM, a phenomena due possibly to the formation of heteroligomers with matrilin-1. He hypothesized that deleting matrilin-1 would inhibit matrilin 1/3 heterotetramers, inhibit matrilin-3 secretion into the ECM and reduce the severity of chondrodysplasia. They crossed a Matn3 pV194D mouse line with Matn1 null mice to generate a matilin-1 null, homozygous pV194D mouse. Characterizing these animals, he observed that the mutant matilin-3 remained in the ECM and the amount of matrilin -3 in chondrocytes was unaffected. Chondrocytes from the wild type and mutant mice formed disulphide bond aggregates for matrilin-3 as demonstrated by western blots. He then went on to characterize the folding and secretion properties of the mutant A domains (pV194D and pV245M). The Matn3 mutation pV194D was not secreted into the ECM whereas the pV245M mutation was, and this secreted protein folded identically to wild type. His talk finished by him concluding a part of mutant matrilin-3 is correctly folded, its secretion does not require matrilin-1 heteroligomerization and that its ablation does not affect MED disease severity.

The final talk and conclusion of the meeting was by **Prof Ben Alman** (University of Toronto, Canada). He began with an overview of limb, bone and cartilage development and their role in OA. Using knockout mice, he demonstrated how hedgehog (Hh) signalling via transcription factors Ptch, Smo and Gli can promote cartilage degeneration due to mechanical trauma and knocking out some of the key players, Kif7 and Sufu (suppressor of fused), enhanced Hh signalling. He also demonstrated how Kif7 is a positive regulator of Hh signaling; Kif7-/- mice showed increase Sufu levels (a Gli activator) compared to wild type mice. Kif7 was found to localize at the tip of primary cilia in chondrocytes with Sufu and Gli located at the base. He then went on to explain how PTHrP regulates Hh signaling by using Sufu knockout mice with PTHrP treatment. He observed an increase in collagen type X but detected a decreased

expression in untreated chondrocytes, to demonstrate that PTHrP normally suppresses Sufu. However, Gli2 over-expressing transgenic mouse chondrocytes resembled those from OA, with decreased proteoglycan expression and increased collagen type X levels, suggesting Hh signaling plays a role. He also investigated Hh signaling in human knee replacement explants by microarray and ChIP-sequencing assays. His results showed increased collagen X, MMP13, Ptch1, Gli1, Hhip1 and interestingly genes involved in cholesterol biosynthesis, the genes for which are important Hh regulated genes in human OA. To demonstrate these finding, he used a conditional knockout of Insig genes in mouse chondrocytes which developed severe OA which was delayed by blocking cholesterol metabolism. He concluded that there was a novel downstream target of Hh signaling and that statins may possibly be a new therapeutic treatment OA.

Meeting report:

Chavaunne Thorpe (Queen Mary, London)

The 4th International Conference on the Mechanics of Biomaterials and Tissues was held at Waikoloa Beach Resort, Hawaii from 11th – 15th December 2011. This biennial meeting was attended by over 200 delegates from many institutions with diverse areas of expertise, making it an excellent forum for presenting work related to the mechanics of biological materials.

With 14 keynote talks and over 100 oral presentations as well as 130 poster presentations, the ICMOBT covered a diverse selection of topics, ranging from the mechanics of hard and soft tissues to biomimetic and bioinspired materials. The meeting began with a keynote address from Prof. Peter Fratzl, from Max Planck Institute of Colloids and Interfaces, entitled “Multiple scale interfaces and mechanical adaptation of biological material”. This fascinating talk addressed the important role the hierarchical organisation of biological matrices plays in determining the mechanical properties of a variety of tissues. The “Soft Tissues” sessions were also of particular interest, and included presentations on the mechanical properties of various soft tissue components.

Highlights included a talk by Prof. Anthony Weiss, describing how tropoelastin can be used to generate functional elastic scaffolds that promote cell attachment, spreading and proliferation. Dr Rene Svensson spoke about the failure properties of collagen fibrils and highlighted the importance of cross-links in the fibril mechanical response. Other presentations in this session covered a wide variety of topics, including the effect of diabetes-related glycation on the properties of the Achilles tendon, the distinct visco-elastic response of tendons with different functions, regulation of articular cartilage tensile properties by IGF-1 and TGF- β 1 during in vitro growth and the effect of smoking of the structure and function of skin.

Another theme covered during the meeting was the effect of matrix mechanics and cell matrix interactions on cell properties. Prof. Huajian Gao from Brown University gave a keynote talk entitled “Probing mechanical properties of cell-material interactions” in which he explored how cells sense the mechanical properties of their matrix and how they actively control adhesion via cytoskeletal contractile machinery. Another keynote on this topic, entitled “Mechanopathology of human diseases – why mechanics matter” was presented by Prof Chwee Lim from the National University of Singapore. Prof Lin explained how the mechanical properties of cells are altered with disease, and how these alterations may be used as biomarkers for early diagnosis. Other presentations followed this theme, with several talks on the effects of matrix stiffness and stress relaxation on cell phenotype and stem cell differentiation.

ICMOBT was a very useful and interesting meeting to attend, particularly as delegates were from backgrounds including materials, engineering, cell biology and chemistry. This resulted in interesting discussions as topics were addressed from a number of different perspectives, which highlighted how a multi-disciplinary approach can be used with great effect in order to understand the structure function relationships in biological materials. The 5th ICMOBT will be held in December 2013, venue TBA, and I would urge any matrix biologists with an interest in tissue mechanics to attend!

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 **Oxford 2012 Joint Meeting** 
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“Matrix Molecular Biology: Integrator of Tissue Function and Disease”
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Local Organisers: Mandy Plumb and Philippa Hulley
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Fell Muir award Lecture: Roger Mason

Confirmed speakers:
Reinhard Fässler, Valerie Weaver, Clare Waterman, Joachim Spatz, Liliana Schaefer, Dietmar Vestweber, John Couchman, Oliver Eickelberg, Paddy Prendergast, Yoshifumi Itoh, Kebs Hodivala-Dilke, Cathy Merry, Francesco dell’Accio, George Bou-Gharios, Paul Martin

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