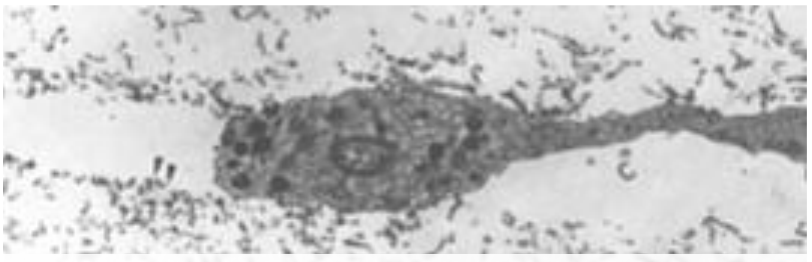

A fibroblast within a tethered lattice, after 13 days, with a cavity surrounding the cell and areas of packed collagen fibrils *(Porter et al. 1998 Wound Rep. Reg. 6: 157-166)*

ISMB NEWSLETTER 30 October 2018
Editor: Sylvie Ricard-Blum

FROM THE PRESIDENT'S DESK

Dear ISMB members,

Dear ISMB members:

Welcome to the 2018 fall issue of the ISMB Newsletter.
This October, our Society celebrates 26 years of successful activities. The Society continues to grow and currently has more than 330 members. It is my pleasure to welcome all new ISMB members hailing from Australia, Denmark, Germany, Ireland, Spain, and the United Kingdom, who are listed in this issue.
With the constant growth of the ISMB, we are able to offer a higher number of travel awards for the up-and-comers in our field, helping them attend matrix biology meetings around the world and present their work. The ISMB travel awards allowed Jennifer Ashworth (UK), Elena Carava (Italy), Heather Davies (France), Laura Dupont (Belgium), Andras Kiss (Hungary), Chun-Yu Lin (Sweden), Marion Marchand (France), Madalina V. Nastase (Germany), and Jazmin Ozsvar (Australia) to attend the Matrix Biology Europe 2018 in Manchester and various Gordon Research Conferences. The winners of the July and October 2018 ISMB travel award competition, Rocco Bernasconi (Germany), Joan Chang (UK), Wing Ying Chow (Germany), Ioanna Kalograiaki (Spain), Essak Khan (Germany), Ayşe Koçak (Turkey) were able to attend the FEBS Advanced Lecture Course, in Patras, Greece from September 27 - October 2, 2018 or the American Society for Matrix Biology meeting in Las Vegas, NV, USA, from October 14-17, 2018. In this 2018 fall issue of the ISMB Newsletter, we include awardees' enthusiastic reports about their experience presenting their own data and discussing this data with established scientists in the matrix biology field. The ISMB Council is pleased to announce the winners of the October 2018 ISMB travel award competition: Yasmene F. Alanazi (UK), Vishal Chaturvedi (Australia), and Chieh Yu (Australia).These winners will attend the 11th Asian and Pan Pacific

Connective Tissue Society Symposium & the 3rd National Conference of CSMB, in Hangzhou, China, November 16 - 20, 2018, or the Matrix Biology Society of Australia and New Zealand, in Auckland, New Zealand, December 4-7, 2018.

As you can see, the summer of 2018 was incredibly successful for the field of matrix biology. Congratulations to the organizers of the 50th Anniversary of Matrix Biology Europe (formerly FECTS), held in Manchester from July 21-24, 2018. At this meeting, ISMB had the pleasure of presenting the Rupert Timpl award 2018 to Alexandra Naba. ISMB also honored David Hulmes, current Secretary and Treasurer of the ISMB, and past President, for his many years of service to our Society. David Hulmes's term as ISMB secretary/treasurer expires at the end of 2018. We are excited to announce that beginning in January 2019, Jo Adams (UK) will serve as secretary of the Society, and Ruud Bank (The Netherlands) will serve as treasurer.

Last month we completed a very successful meeting of the American Society for Matrix Biology in Las Vegas, NV, USA, October 14-17, 2018. The ISMB presented the ISMB Distinguished Investigator award to Billy Hudson at the ASMB meeting, and on Billy's birthday nonetheless! It was my honor and pleasure to present Billy with this well-deserved award. The audience recognized his spectacular lecture with a standing ovation.

As the field of matrix biology continues to grow, so too do our journals. During the ASMB meeting in Las Vegas, birth of a new journal in the field of matrix biology was officially announced. Based on the success of the Matrix Biology (IF 2017 8.1) and growing number of submissions to the journal, Matrix Biology Plus, the newest journal, is now open for submissions. The details about the journal and differences in the scope are presented in this issue of the ISMB Newsletter. Please submit your papers to the Matrix Biology and the Matrix Biology Plus journals!

Finally, I would like to wish successful grant applications and fascinating scientific discoveries to all of our members and readers. Enjoy the golden fall and celebrate Thanksgiving and the Christmas holidays with your families and friends.

Kind regards,

Liliana Schaefer
ISMB President

COMPOSITION OF ISMB COUNCIL SUBCOMMITTEES

Communication

Jo Adams (UK)
Danny Chan (Hong-Kong)
Julia Etich (Germany)
Wei Kong (China)
Sylvie Ricard-Blum (France, chair)

Membership

Jamie Fitzgerald (Australia, chair)
Hide Watanabe (Japan)
Suneel Apte (USA)

Meetings and travel grants

Anthony Day (UK)
Ruud Bank (The Netherlands, chair)
Barbara Smith (USA)
Gerhard Sengle (Germany)
Hide Watanabe (Japan)
Chloé Yeung (Denmark)

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ISMB correspondents of National Societies for Matrix Biology for Facebook & Twitter

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The British Society for Matrix Biology
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The Finnish Connective Tissue Society
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The German Society for Matrix Biology
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Matrix Biology Society of Australia and New Zealand (MBSANZ)
Luise Kung, Murdoch Childrens Research Institute, Melbourne (Australia)
louise.kung@mcri.edu.au

MEETING REPORTS

Matrix Biology Europe 2018, Celebrating 50 years of FECTS Meetings, The University of Manchester (UK) 21st - 24th July 2018

I am a third year PhD student in the lab of Professor Alberto Passi at the Università degli Studi dell'Insubria of Varese (Italy). Matrix biology is the focus of my research group. We are interested in the involvement of hyaluronan and syndecans in the atherosclerosis disease because on them depend the matrix reorganization happening during the onset of the atherosclerotic plaque.
I was really honoured that ISMB awarded me a travel grant because it facilitated my attendance at the Matrix Biology Europe 2018 congress that took place on 21-24 July 2018 in Manchester, UK.
MBE is the most important European conference dealing with Matrix Biology. I participated for the first in Athens in 2016 and I was very excited to attendance the conference for a second time because it would have been an unmissable opportunity to hear about the most exciting and new aspects of the research performed on the extracellular matrix. During the congress I had the possibility to follow very interesting plenary sessions and to choose the workshops more related to my research topic.
On this occasion I presented a poster on the role of PCSK9 (a new therapeutic target for atherosclerosis disease) in remodelling of matrix components such as hyaluronan and syndecan in vascular cells. I had very interesting discussions about this. Furthermore, it was the occasion to discuss with the greatest connoisseurs about the role of proteoglycans in endothelial permeability and the hyaluronan modifications during inflammation, which are the topics of my thesis project.
During the meeting we also celebrated the 50 years of Federation of European Connective Tissue Societies (FECTS) foundation and the first meeting held in Cambridge, UK (June 30th to July 2nd, 1968). The celebration took place in the Manchester Museum where John Couchman gave a beautiful and fascinating talk in an amazing location.
MBE 2018 gathered together world-class speakers with young scientists. The days were very full of sessions, but I had time for visiting the city of Manchester with other PhD students like me. This social aspect is important in a meeting because it gave me the opportunity to enjoy the free time and to create contacts with colleagues in a less formal manner. To conclude, the congress expanded my knowledge on matrix biology, allowed me to search collaborations and gave me enthusiasm for future working perspectives.
Elena Caravà, Insubria University of Varese (Italy)

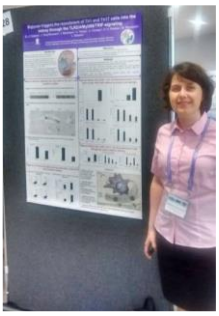
I am a first year postdoc in the lab of Dr. Alain Colige at University of Liege in Belgium where I mainly study the function of ADAMTS2, 3 and 14. My first Matrix Biology meeting was in 2016 in Athens, Greece. For the first time, my abstract was selected for a talk. I was very excited to present our work on "ADAMTS3 activity is mandatory for embryonic lymphangiogenesis and regulates placental angiogenesis". The Matrix Biology meeting was an interesting experience; not only could I interact with prominent researchers who are doing outstanding research, but I also learned much more about development, homeostasis and (genetic) disorders of the extracellular matrix.
Finally, I was honored to get an ISMB Travel Award so I could attend the MBE2018 meeting in Manchester, United Kingdom. This meeting marked 50 years since the very first conference of European matrix biology societies in Cambridge (30th June-2nd July 1968). The conference grew out of a need for young researchers to have a forum for their research and to meet experts in the field. The organizers have planned a series

of international plenary and workshop presentations from leaders in the field and every workshop will be included at least 4 presentations selected from abstracts submitted. There will be plenty of opportunities to network with colleagues at various receptions and poster sessions.
During the meeting I was very happy to present my poster on recently published work: “Spontaneous atopic dermatitis due to immune dysregulation in mice lacking Adamts2 and 14”. The results presented aroused the interest of several participants of the congress by their innovative aspect and the widening of the dogma concerning the amino procollagen peptidases that are the ADAMTS2, 3 and 14 and which are key actors of the extracellular matrix. The discussions related to this work mainly concerned the techniques used or related to certain aspects also dealt with by other participants in the congress. By attending some of the oral presentations of the MBE2018 or by chatting with other participants, some ideas came to my mind regarding the continuation of the project, which would not necessarily have been the case without my participation in this conference.
Participation in this congress was intended to broaden my knowledge in the field of extracellular matrix. It was also intended to bring new ideas to the fore for my project, drawing on what is being done elsewhere, and to discuss with the participants in order to establish contact with them and to establish future collaborations. I believe that the MBE2018 conference has fully met these objectives as it brought together world-renowned researchers in the field of extracellular matrix and collagens; it allowed me to chat with other researchers to get some details about particular techniques, or about some aspects of my project. The congress was, in my opinion, well organized. The oral presentations were of good quality and the researchers were generally available for discussion during the various "poster sessions". This congress was a great opportunity to present our work and represent the laboratory internationally.
Laura Dupont, University of Liège (Belgium)

First of all, I would like to thank the ISMB for granting me a travel grant, which enabled me to participate in the MBE 2018 Conference in Manchester. I am studying at the University of Szeged as a last year PhD student, and I deal with collagen IV collagen COL4A1 mutations, and this conference gave me a fantastic opportunity to present my results in a poster presentation to the most experienced researchers in this field. It has helped my work so much that I can see many unpublished data that have given me new ideas for my future plans. It was especially useful that I could talk casually with foreign researchers, professors and PhD students from all over the world. I have received many inspirational opinions about my current research, which will surely help my work in the future. The atmosphere was excellent at the conference, and I got many new friends. It was a pleasure for me to build up many new cooperative relationships. I liked the common programs where we had open discussions all over the world. The Manchester Museum was beautiful, and it was a pleasure for me to get to the Old Trafford stadium I was looking forward to. Many people have come to the Manchester Conference, and it was a pleasure to see that so many people deal with the extracellular matrix in the world. It was a good feeling that I could meet both current and future researchers in the profession. I feel amazed at the many helpful advice and approaches. It is my pleasure to know that I have chosen an excellent research area and how much potential is in this topic. The number of options is endless, and I know that I can seek advice from all over the world, as many people are working in this field.
Overall, I've had very positive experiences at this conference. It was very good at organising, I met a lot of inspirational colleagues, and I managed to build new friendships and collaborations. I'm sure I chose a right way.
András A. Kiss, PhD student, University of Szeged (Hungary)

In July 2018, I had the great pleasure to receive a travel grant from the ISMB to attend the Matrix Biology Europe meeting held in Manchester, UK. The meeting was organized by the British Society for Matrix Biology together with the Wellcome Trust Centre for Cell-Matrix Research and was hosted at The University of Manchester. With the help of this grant, I was able to travel to Manchester and to give a talk on my recent work which is about ECM scaffolding and regulation ofangiogenesis. More precisely, my PhD project is focused on vascular ECM remodeling by the lysyl oxidase like-2 (LOXL2) during angiogenesis. This protein regulates the cross-linking of collagens and elastin in the extracellular matrix of blood vessels and I am now focused on the regulation of mechanotransduction in endothelial cells, as a direct consequence of ECM organization by LOXL2. As this meeting is held every two years, I really felt lucky to participate this year, as this may be the only possibility for me to attend. This was also the opportunity to celebrate 50 years of FECTS meetings. The meeting was great, as the program was covering a large range of very exciting topics from fundamental to applied research. I appreciated that the schedule was leaving time to discuss and socialize during poster sessions. The general atmosphere of the meeting was really friendly and welcoming, and it was nice to meet other graduate students and postdocs working on different aspects of matrix biology. I learned a lot on different topics during the meeting, as the atmosphere was really stimulating. I especially liked the 'matrix mechanobiology' session that is directly linked to my project. The talk by Alexandra Naba on the matrisome project was absolutely stunning and gave me lots of ideas for my future research. I was also really pleased to attend the talk from Viola Vogel on mechanobiology of ECM fibers in vitro and in vivo. She shared very exciting and amazing data on fibers mechanics and this topic is particularly interesting for me. The conference was also a way to open my mind about new questions linked to my points of interests, especially ECM proteomics and cell/matrix interactions. I would like to strongly thank the ISMB committee for their support, together with the conference organizers and I am looking forward to attend the next MBE in Florence!

Marion Marchand, CIRB, Collège de France (Paris)



I attended the Matrix Biology Europe 2018 Conference hosted at The University of Manchester on the 21-24th of July as a postdoc, being awarded by ISMB with an international travel grant. This was a big opportunity as well as an honor and I sincerely thank to the ISMB Committee for consideration.

The conference impressed me from day one as the organizers gave us the occasion as a start up to gather all together in the spectacular building of Manchester Museum which was fascinating to explore.

The scientific research communicated at the conference covered a diversity of aspects of the extracellular matrix components, their function as structural components as well as matrix component dynamics and signaling with translation in physiology and pathology. The interesting ideas and concepts as well as the newly advanced methodologies impacted consistently my overview on the progress of the projects I am working with.

Moreover, I appreciated very much the sessions where both seniors in the field as well as young investigators starting their own independent groups were awarded for their important contribution to the extracellular matrix field. The sessions were very interesting and dynamic.

The presentation of our laboratory research including the talk held by my PI and a poster presented by me was focused on the key signaling pathways promoted by the matrix-derived component biglycan in acute and chronic renal inflammation. I found our work complementary with the research of other scientists from where we had nice feedbacks and suggestions.

Finally, the social events including the coffee breaks, meals, gathering together at the Manchester Museum or the dinner held at Manchester United Football Club were very good occasions to group together in very nice locations and in a friendly atmosphere. It was a great pleasure and honor for me to meet so many experts in the matrix field. I was really enjoying to participate at this event of the matrix scientific community which is putting the scientific skills, passion for science, creativity and human qualities at the service of science and mankind needs.

Madalina-Viviana Nastase, PostDoc, Institute of General Pharmacology and Toxicology Frankfurt am Main (Germany)

I was given the honour of receiving an ISMB Travel Award to attend Matrix Biology Europe 2018 (MBE2018), held in Manchester, UK. MBE2018 was directly relevant to my own project, which revolves around the elastin fibre precursor, tropoelastin. This was my first MBE conference, and it was a pleasure to attend. Attending MBE2018 gave me the chance to attend a wide range of talks, encompassing topics such as stem cells, matrix mechanobiology, cell fate and mechanisms of matrix diseases. These talks were not only given by renowned research leaders, but also by PhD students and early career postdoctoral researchers. Of particular relevance to me was Professor Viola Vogel's presentation on mechanobiology of matrix fibres, during which I drew inspiration from some of her previous molecular dynamics approaches to apply to my own future studies. Additionally, I had the opportunity to listen to presentations for the prestigious Dick Heinegard Young Investigator Award, where five early career researchers pitched their research topics to a panel of judges. I also greatly enjoyed Professor Ray Boot-Handford's talk on "the trials and tribulations of a life with collagen" during his acceptance of the Fell Muir Award. By receiving the Travel Award, I was able to present a poster on my own PhD work, which involves computational modelling of tropoelastin. I thoroughly enjoyed answering questions about my work and was grateful for the feedback and interest I received. There was also plenty of time to walk around and visit other posters, several of which I found relevant to my own work. This also ended up sparking a number of interesting discussions and networking opportunities. I would like to convey my sincere thank you to the ISMB for selecting me for the prize, and to the British Society for Matrix Biology for organising an exciting, interactive conference. I sincerely hope to attend another MBE conference in the future.

Jazmin Ozsvar, School of Life and Environmental Sciences, The University of Sydney (Australia)

The Rupert Timpl Award presented to Alexandra Naba (University of Illinois at Chicago, USA) by the President of the ISMB, Liliana Schaefer, and by Valerie Teng-Broug (Publisher, Matrix Biology Elsevier, which sponsored the award) during the Matrix Biology Europe meeting in Manchester (UK)

Gordon Research Seminar and Conference on Proteoglycans, Proctor Academy, Andover, NH (USA)
July 7th-13th, 2018

The Proteoglycans Gordon Research Conference (GRC) is a small meeting of around 200 researchers, making it the ideal environment for networking with other researchers in the field. It takes place at a beautiful academy in New Hampshire, with lots of opportunities for activities such as sports, swimming and even wine tasting, meaning that the environment is very relaxed and ideal for meeting new people. This, in combination with the very high quality of research presented at the conference, has given me many new ideas for collaborations, new experiments and fellowship applications.
The GRC is preceded by the GRS, or Gordon Research Seminar, which is aimed at early career researchers. I was selected to give an oral presentation at the GRS, which led to some very useful feedback on my work and has already inspired some new experiments. The GRS was also a great chance to meet researchers at my own level, as well as to hear advice from and talk with mentors such as Glenn Prestwich and Liliana Schaefer. The arrival of many very well-established and famous researchers at the start of the GRC could easily be a daunting experience, but the GRS acts as a brilliant introduction and really helps with the sense of community, which is one of the nicest things about the whole conference.
At the GRC itself, I presented a poster, and I was very grateful to receive a poster award from the conference organisers. Discussions at my poster helped me to form a plan for an initial publication, by identifying the focus that would be of most interest to other researchers. My postdoc position is based on developing a fully-defined in vitro model of breast cancer progression, and my main interest in attending this conference was to understand how I might be able to use this model to study cell-matrix interactions. I found many of the talks highly relevant for this application, in particular several of the talks that focussed on the influence of hyaluronan on mechanobiology, and the role of ECM on fibroblast activation and contractility. I am very grateful to ISMB for providing financial support to allow me to attend the GRC, and would highly recommend the proteoglycans GRC to anyone in the field.
Jennifer Ashworth, Nottingham (UK)

Thank you to the ISMB for the Travel Award to attend and present at the Gordon Research Conference (GRC) on Proteoglycans in 2018. I'm a post-doc working in University Grenoble Alpes, France, mentored by Dr. Ralf Richter (University of Leeds, UK) investigating the physical mechanisms by which immune cells home to the blood vessel wall, with a particular focus on CD44+ immune cells binding to hyaluronan in the glycocalyx. I was very keen to attend my second GRC on Proteoglycans after thoroughly enjoying my first in 2016.

Before the GRC, I attended the Gordon Research Seminar (GRS), where it was great to meet up with people who I had met in 2016, and meet new faces too. The atmosphere was friendly and approachable and there were very interesting scientific discussions both after the oral presentations and at the poster sessions. A new session that was held this year was a panel discussion with Glenn Prestwich (Symic Bio, USA), Liliana Schaefer (University of Frankfurt, Germany) and the GRS chairs Aaron Petrey (Cleveland Clinic, USA) and Rogier Reijmers (Leiden University Medical Center, The Netherlands). This interactive session was a great addition, in which advice was given to the GRS audience of young researchers on how to progress in academic science.

Following the GRS, the GRC attendees arrived to bring the total number of attendees to 200. Tony Day (University of Manchester, UK) and Carol de la Motte (Cleveland Clinic, USA) did a fantastic job and

organised excellent scientific sessions, particularly in terms of the variety of disciplines covered, quality of research and level of discussion after each talk. Masters students, PhD students, post-docs and principal investigators all contributed to the oral presentations and were actively encouraged to take part in the discussions. From the first day until the last, the science was stimulating and exciting. For instance, in the first session of 'Late-breaking topics', we heard from Spencer Freeman (The Hospital for Sick Children, Canada) about how CD44 has a newly discovered role in co-ordinating the activity of other receptors by forming transmembrane pickets. On the last day, Cathy Merry (University of Nottingham, UK) gave a fantastic talk about developing well-defined 3D biomaterials for growing cell cultures *in vitro* for improved models of disease and development. Such talks were particularly inspiring and I have taken away a wealth of knowledge and new ideas for future endeavors.

During the conference, I presented a poster and presented a short talk in the session 'Proteoglycan-based technologies and treatments'. I received interesting comments from the audience in the discussion session immediately after my talk, as well as afterwards during the social events. Such discussions have highlighted that different aspects of my project have potentially interesting applications to other contexts that I hadn't realised before attending this conference, and so could serve to progress this work beyond my post-doctoral project. In addition to the excellent opportunities to discuss scientific details, what I especially like about the GRS/GRC on Proteoglycans is the way it brings together industry and academia, with prestigious players from both being active participants in the meeting. In addition, this conference goes all out with social activities, having dedicated social chairs who organised a talent show, bird-watching and a whole range of events to take part in. Overall the GRS/GRC provided a fun, relaxed atmosphere for superb scientific discussion.

Dr. Heather Davies, University Grenoble Alpes (France)

It is my great honor to be funded by the International Society for Matrix Biology (ISMB) to participate in this 2018 GRC proteoglycans conference. I am impressed by the conference because the organization committee included so many fields related to proteoglycan research, such as the fields of infectious diseases, inflammatory diseases, and fibrosis.

The conference emphasized sharing of unpublished data by the speakers. Therefore, I learned a lot about novel fields of proteoglycan studies, such as cell surface hyaluronidase, TMEM2. There were also discussions about novel technics and tools to analyze the tissue samples, although there was still a gap to go for the analysis of the human plasma or serum. In the poster sessions, I appreciated to have the chance to discuss the details of experiments with researchers who are working in close fields and perform similar experiments as I do. For me, an important part of this conference was that I participated in some discussions about the roles of proteoglycan molecules and pathway modulations. It is thought-provoking that we can learn much from addressing a question from different perspectives.

As an inexperienced participant, throughout this conference, around the dining tables, walking aside the lake, on the football game court, and even in the living room of our accommodation, I was relishing the talks with senior investigators. We had discussed about scientific issues, practical problems, and even their opinions and experiences based on their careers. This will be great advantage for me to go further in my own career.

Out of these kinds of feedback during this conference, I generated several ideas which will be crucial in my future research. First of all, to elucidate the role of hyaluronan on macrophages surface during the process of flaviviral entrance and infection. It would be valuable to find out what kind of effects over-produced hyaluronan has on the susceptible macrophages and other immune cells. A second idea is to perform the

systemic biology analyses with plasma samples from those patients with different kinds of infectious diseases. A third idea is to elucidate the role of different kinds of hyaluronan binding proteins, such as neurocan, among infectious diseases.

As a clinician-backgrounded PhD student, I believe that the translation of proteoglycan research into clinical implications (such as discoveries of diagnostic biomarkers, and therapeutics molecules and materials innovations) is very important. Therefore, this conference perfectly fitted with my research interests. Finally, I would like to thank the committee of ISMB again for supporting me to participate this excellent and meaningful conference.

Chun-Yu Lin Uppsala (Sweden)/Kaohsiung (Taiwan)

I really appreciate that ISMB awarded me a travel grant to attend the GRS and GRC meeting on proteoglycans 7-13 July, 2018, at Proctor Academy in Andover NH, US. This was my second GRS/GRC on proteoglycan meeting, and this meeting has always been one of my favourite meetings to attend. This year was no exceptional. The conference started with the 2 day GRS, where only PhD students and post-doc attended. This was a great opportunity to network with the young scientists.

The GRC was straight after the GRS, where more senior researchers joined. The talks were excellent and the research was cutting-edge. Although some of them were not directly related to my research, I still learned a lot. My favourite talk was from Catherine Merry (University of Nottingham), where she talked about their chemically defined and serum free 3D cell culture environment that supports iPS and stem cell culture and differentiation.

I gave a Poster Preview talk, which was 90 seconds long. It was quite a challenge but good experience to summarise my poster content into 90 seconds, which also need to attract people's attention as well. The poster session was very helpful and interesting. The great thing about GRC is that people are very open to discuss their projects and are also willing to listen and give advices and suggestions. I was able to get some great suggestions and comments about my project during the poster session.

Overall, I thoroughly enjoyed this year's GRS/GRC proteoglycan conference, and I would highly recommend this conference. I would like to thank the ISMB again for the financial support.

Fengying Tang, University of New South Wales, Sydney (Australia)

The FEBS Advanced Lecture Course on Extracellular Matrix: Cell Regulation, Epigenetics and Modeling, University of Patras (Greece), September 27th - October 2nd 2018

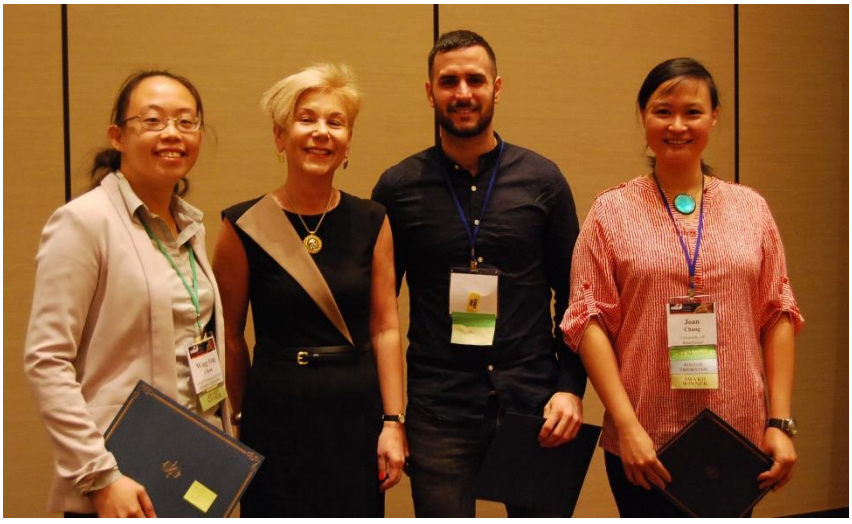
I would like to thank International Society for Matrix Biology (ISMB) travel grant to attend the FEBS Advanced Lecture Course on Extracellular Matrix: Cell Regulation, Epigenetics and Modeling, held in the Conference & Cultural Center of the University of Patras, 27th September to 2nd October 2018.

I am currently a Postdoc in the laboratory on Dokuz Eylul University and work on scleroderma and fibrosis. Also, My PhD thesis about fibrosis mechanism and scleroderma pathogenesis. Also, I presented my preliminary data and this course was the perfect place for it. This advanced course was an excellent opportunity to give me new perspectives. Also, all participants served the conference well in terms of the breadth of research presented. Lectures were so powerful and informative. Especially the new techniques were very useful for me and our researches. Also, I learned a lot of new things. My poster and oral presentation was about on the meprins on scleroderma pathogenesis. I had very interesting discussions. The course program was very impressive. The program started at 9 am, and finished about 7 pm. Lectures, selected talks and flash talks were so interesting. I attended to all lectures where worldwide experts, in that varied range of research areas, presented their research. The poster time after lunch provided a great

opportunity to spend the time with other participants and lecturers. There were also career development sessions designed for PhD students presented by representatives of the FEBS Education Committee.
The social program was so enjoyable. Thanks to all organizing committee. For me, this was an extremely stimulating and impressive experience. Also, I was very chuffed to be part of such an amazing scientific event and I am very grateful to the ISMB for their support.
Ayşe Koçak, PhD, Dokuz Eylul University, Izmir (Turkey)

ASMB Biennial Meeting, October 14-17, 2018, Las Vegas, NV (USA)

The ISMB Distinguished Investigator award 2018 has been presented to Billy Hudson (Vanderbilt University Medical Center, Nashville, USA) by the President of the ISMB, Liliana Schaefer, at the ASMB meeting in Las Vegas (USA)



ISMB travel awardees at the ASMB meeting 2018. From the left: Wing Ying Chow, Rocco Bernasconi and Joan Chang.

POSITIONS AVAILABLE on ISMB website (http://ismb.org/career/)

Post-doctoral positions in vascular development and disease at the Cleveland Clinic Lerner Research Institute, OH (USA)

Post-doctoral fellowships are available in Suneel Apte's laboratory at the Cleveland Clinic. The project area will be extracellular matrix in vascular development and disease. The positions will suit PhDs, MD/PhDs or MDs with an interest in proteases, extracellular matrix, smooth muscle cell biology, mouse genetics, human vascular disease and proteomics. Applicants should send their curriculum vitae, a statement of research/career goals and the contact information for three references with an intimate knowledge of their work to **aptes@ccf.org**.
The Cleveland Clinic is an equal opportunity employer

Post-doctoral position available at the University of Illinois at Chicago (USA)

The Naba Lab in the Department of Physiology and Biophysics in the College of Medicine at the University of Illinois at Chicago is looking for an outstanding postdoctoral fellow. The successful candidate will join a young, dynamic and collaborative research team to study the role of the extracellular matrix in cancer metastasis and developmental biology. Our laboratory employs a wide range of techniques and approaches, including mouse genetics, cell biology, proteomics, and bioinformatics to study how the extracellular matrix governs normal developmental processes (craniofacial morphogenesis and bone development) and disease progression (cancer and fibrosis). Our current research particularly focuses on a novel ECM protein SNED1, identified by Dr. Naba as a breast cancer metastasis promoter (eLife, 2014). More information on the Naba lab can be found here: http://nabalab.uic.edu.

Applicants are required to hold a Ph.D. in biology or a related field and have a solid background in cell biology and at least one of the following fields: cancer biology, developmental biology, cell adhesion, signal transduction, live-cell imaging. Previous experience working with mouse models of cancer or to study development is preferable. Applicants must be highly motivated and able to work independently in a very collaborative environment. Applicants are expected to have demonstrated excellence in research through publications and presentations, have a strong work ethic, and have excellent writing and oral communication skills.

By joining our team, the successful candidate will experience working with a broad range of techniques on an exciting and challenging research project. He/she will get opportunity to be involved in mentoring and benefit from interactions with a network of collaborators in the U.S.A and Europe.

Interested applicants should submit 1) a cover letter outlining research interests, relevant work experience and career goals, 2) a CV including complete bibliography, and 3) the contact information for 3 references. Applications and inquiries should be sent to Dr. Naba anaba@uic.edu.

The University of Illinois at Chicago is an Equal Opportunity, Affirmative Action employer. Minorities, women, veterans and individuals with disabilities are encouraged to apply. The University of Illinois may conduct background checks on all job candidates upon acceptance of a contingent offer. Background checks will be performed in compliance with the Fair Credit Reporting Act.

German Research Council-funded postdoctoral fellowship in the laboratory of Liliana Schaefer, Frankfurt (Germany) January 1st, 2019

The positions require a Ph.D. in biochemistry or biology and a post-graduate (PhD) academic education and experience in autophagy or ubiquitination. The fellows will join an exciting and energetic team of researchers using state-of-the-art techniques in an innovative environment. We are scientifically anchored in the Collaborative Research Centre CRC815, CRC1039, CRC1177, and Cluster of excellence. Our scientific work is focused on innate immunity receptors and their corresponding signaling pathways. Innate immune responses are involved in a broad spectrum of acute and chronic inflammatory processes, e.g. sepsis, malignant, fibrotic and auto-inflammatory diseases, whereby so far, no curative therapeutic approaches have been discovered for these various disease entities (*unmet medical need*).
If you are interested, please submit your C.V. with photo, list of publications, copies of certificates etc. to: **Schaefer@med.uni-frankfurt.de**

Faculty Position Available Immediately

ASSOCIATE DIRECTOR The Joan and Joel Rosenbloom Research Center for Fibrotic Diseases
Academic researcher/manager faculty position at **The Sidney Kimmel Medical College at Thomas Jefferson University, Philadelphia, PA (USA)**

Candidates with strong interest and track record in fibrosis focused research will be considered for a professorial appointment at the level dependent on experience and track-record.
For application, please forward your *Curriculum Vitae*, or for inquiries, please contact:

Joel Rosenbloom, MD, PhD
Director, The Joan and Joel Rosenbloom Research Center for Fibrotic Diseases
Joel.Rosenbloom@jefferson.edu
Telephone: 215-503-8661

or

Jouni Uitto, MD, PhD
Professor and Chair
Department of Dermatology and Cutaneous Biology
Director, Jefferson Institute of Molecular Medicine
Jouni.Uitto@jefferson.edu
Telephone: 215-503-5785

NEWS

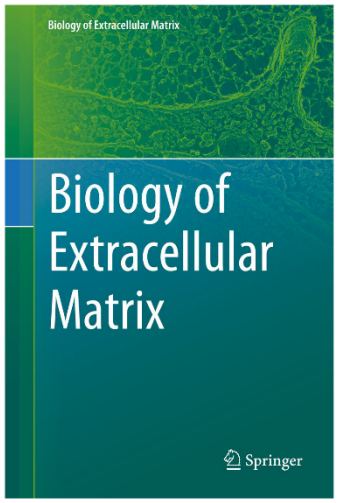
The birth of **MATRIX BIOLOGY PLUS** was officially announced during the ASMB meeting held in Las Vegas (USA) in October.

AWARDS

Professor Anthony Weiss has been given this year the following awards Medal of the Order of Australia, Eureka Prize for Innovation in Medical Research, Fellow of Tissue Engineering and Regenerative Medicine (TERMIS), elected Chair of TERMIS Asia-Pacific, and Vice-Chancellor's Award for Excellence for his work on tropoelastin and synthetic elastin. He is the McCaughey Chair in Biochemistry at the University of Sydney (Australia). His lab's research (http://www.weisslab.net/) focuses on the use of human tropoelastin in elastic tissue assembly and wound repair. This encompasses a blend of biochemistry and cell biology, as well as the tissue engineering and regenerative medicine opportunities provided by this elastic protein

BIOLOGY OF EXTRACELLULAR MATRIX SERIES

Professor Nikos Karamanos (University of Patras, Greece) is the Series editor of *Biology of Extracellular Matrix* series published by Springer (https://www.springer.com/series/842



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Biology of Extracellular Matrix

Series Ed.: N.K. Karamanos

Extracellular matrix (ECM) biology, which includes the functional complexities of ECM molecules, is an important area of cell biology. Individual ECM protein components are unique in terms of their structure, composition and function, and each class of ECM macromolecule is designed to interact with other macromolecules to produce the unique physical and signaling properties that support tissue structure and function. ECM ties everything together into a dynamic biomaterial that provides strength and elasticity, interacts with cell-surface receptors, and controls the availability of growth factors. Topics in this series include cellular differentiation, tissue development and tissue remodeling. Each volume provides an in-depth overview of a particular topic, and offers a reliable source of information for post-graduates and researchers alike. "Biology of Extracellular Matrix" is published in collaboration with the American Society for Matrix Biology.

Recently published:

R.A. Brekken, D. Stupack (Eds.)
Extracellular Matrix in Tumor Biology

D.W. DeSimone, R. Mecham (Eds.)
Extracellular Matrix in Development

F.W. Keeley, R. Mecham (Eds.)
Evolution of Extracellular Matrix

RECENT PAPERS

Kiss AA, Popovics N, Boldogkői Z, Csiszár K, Mink M. 4-Hydroxy-2-nonenal Alkylated and Peroxynitrite Nitrated Proteins Localize to the Fused Mitochondria in Malpighian Epithelial Cells of Type IV Collagen *Drosophila* Mutants. Biomed Res Int. (2018) Jan 30;2018:3502401.

Corresponding author: mink@bio.u-szeged.hu

Abstract: *Background. Human type IV collagenopathy is associated with mutations within the COL4A1 and to a less extent the COL4A2 genes. The proteins encoded by these genes form heterotrimers and are the highest molar ratio components of the ubiquitous basement membrane. The clinical manifestations of the COL4A1/A2 mutations are systemic affecting many tissues and organs among these kidneys. In order to uncover the cellular and biochemical alterations associated with aberrant type IV collagen, we have explored the phenotype of the Malpighian tubules, the secretory organ and insect kidney model, in col4a1 collagen gene mutants of the fruit fly Drosophila melanogaster. In Malpighian epithelial cells of col4a1 mutants, robust mitochondrial fusion indicated mutation-induced stress. Immunohistochemistry detected proteins nitrated by peroxynitrite that localized to the enlarged mitochondria and increased level of membrane peroxidation, assessed by the amount of proteins alkylated by 4-hydroxy-2-nonenal that similarly localized to the fused mitochondria. Nuclei within the Malpighian epithelium showed TUNEL-positivity suggesting cell degradation. The results demonstrated that col4a1 mutations affect the epithelia*

and, consequently, secretory function of the Malpighian tubules and provide mechanistic insight into col4a1 mutation-associated functional impairments not yet reported in human patients and in mouse models with mutant COL4A1.

Karamanos NK, Piperigkou Z, Theocharis AD, Watanabe H, Franchi M, Baud S, Brézillon S, Götte M, Passi A, Vigetti D, Ricard-Blum S, Sanderson RD, Neill T, Iozzo RV. Proteoglycan Chemical Diversity Drives Multifunctional Cell Regulation and Therapeutics. Chem Rev. 2018 118: 9152–9232
Corresponding author: n.k.karamanos@upatras.gr
Abstract: *The extracellular matrix (ECM) constitutes a highly dynamic three-dimensional structural network comprised of macromolecules, such as proteoglycans/glycosaminoglycans (PGs/GAGs), collagens, laminins, fibronectin, elastin, other glycoproteins and proteinases. In recent years, the field of PGs has expanded rapidly. Due to their high structural complexity and heterogeneity, PGs mediate several homeostatic and pathological processes. PGs consist of a protein core and one or more covalently attached GAG chains, which provide the protein cores with the ability to interact with several proteins. The GAG building blocks of PGs significantly influence the chemical and functional properties of PGs. The primary goal of this comprehensive review is to summarize major achievements and paradigm-shifting discoveries made on the PG/GAG chemistry-biology axis, focusing on structural variability, structure-function relationships, metabolic, molecular, and epigenetic mechanisms underlying their synthesis. Recent insights related to exosome biogenesis, degradation, and cell signaling, their status as diagnostic tools and potential pharmacological targets in diseases as well as current applications in nanotechnology and biotechnology are addressed. Moreover, issues related to docking studies, molecular modeling, GAG/PG interaction networks, and their integration are discussed.*

A pipeline to translate glycosaminoglycan sequences into 3D models. Application to the exploration of glycosaminoglycan conformational space. Glycobiology 2018 Sep 18. doi: 10.1093/glycob/cwy084. [Epub ahead of print]
Clerc O, Mariethoz J, Rivet A, Lisacek F, Pérez S, Ricard-Blum S.
Corresponding author: sylvie.ricard-blum@univ-lyon1.fr
Abstract: *Mammalian glycosaminoglycans are linear complex polysaccharides comprising heparan sulfate, heparin, dermatan sulfate, chondroitin sulfate, keratan sulfate and hyaluronic acid. They bind to numerous proteins and these interactions mediate their biological activities. GAG-protein interaction data reported in the literature are curated mostly in MatrixDB database (http://matrixdb.univ-lyon1.fr/). However, a standard nomenclature and a machine-readable format of GAGs together with bioinformatics tools for mining these interaction data are lacking. We report here the building of an automated pipeline to (i) standardize the format of GAG sequences interacting with proteins manually curated from the literature, (ii) translate them into the machine-readable GlycoCT format and into SNFG (Symbol Nomenclature For Glycan) images and (iii) convert their sequences into a format processed by a builder generating three-dimensional structures of polysaccharides based on a repertoire of conformations experimentally validated by data extracted from crystallized GAG-protein complexes. We have developed for this purpose a converter (the CT23D converter) to automatically translate the GlycoCT code of a GAG sequence into the input file required to construct a three-dimensional model.*

Vallet SD, Miele AE, Uciechowska-Kaczmarzyk U, Liwo A, Duclos B, Samsonov SA, Ricard-Blum S. Insights into the structure and dynamics of lysyl oxidase propeptide, a flexible protein with numerous partners. Sci Rep. (2018) 8: 11768.

Corresponding author: sylvie.ricard-blum@univ-lyon1.fr

Abstract: *Lysyl oxidase (LOX) catalyzes the oxidative deamination of lysine and hydroxylysine residues in collagens and elastin, which is the first step of the cross-linking of these extracellular matrix proteins. It is secreted as a proenzyme activated by bone morphogenetic protein-1, which releases the LOX catalytic domain and its bioactive N-terminal propeptide. We characterized the recombinant human propeptide by circular dichroism, dynamic light scattering, and small-angle X-ray scattering (SAXS), and showed that it is elongated, monomeric, disordered and flexible (Dmax: 11.7 nm, Rg: 3.7 nm). We generated 3D models of the propeptide by coarse-grained molecular dynamics simulations restrained by SAXS data, which were used for docking experiments. Furthermore, we have identified 17 new binding partners of the propeptide by label-free assays. They include four glycosaminoglycans (hyaluronan, chondroitin, dermatan and heparan sulfate), collagen I, cross-linking and proteolytic enzymes (lysyl oxidase-like 2, transglutaminase-2, matrix metalloproteinase-2), a proteoglycan (fibromodulin), one growth factor (Epidermal Growth Factor, EGF), and one membrane protein (tumor endothelial marker-8). This suggests new roles for the propeptide in EGF signaling pathway.*

Pulido D, Sharma U, Vadon-Le Goff S, Hussain SA, Cordes S, Mariano N, Bettler E, Moali C, Aghajari N, Hohenester E, Hulmes DJS. Structural Basis for the Acceleration of Procollagen Processing by Procollagen C-Proteinase Enhancer-1. Structure (2018) Jul 12. pii: S0969-2126(18)30243-0.

Corresponding authors: e.hohenester@imperial.ac.uk and david.hulmes@ibcp.fr

Abstract*: Procollagen C-proteinase enhancer-1 (PCPE-1) is a secreted protein that specifically accelerates proteolytic release of the C-propeptides from fibrillar procollagens, a crucial step in fibril assembly. As such, it is a potential therapeutic target to improve tissue repair and prevent fibrosis, a major cause of mortality worldwide. Here we present the crystal structure of the active CUB1CUB2 fragment of PCPE-1 bound to the C-propeptide trimer of procollagen III (CPIII). This shows that the two CUB domains bind to two different chains of CPIII and that the N-terminal region of one CPIII chain, close to the proteolytic cleavage site, lies in the cleft between CUB1 and CUB2. This suggests that enhancing activity involves unraveling of this chain from the rest of the trimer, thus facilitating the action of the proteinase involved. Support for this hypothesis comes from site-directed mutagenesis, enzyme assays, binding studies, and molecular modeling.*

Hiebert P, Wietecha MS, Cangkrama M, Haertel E, Mavrogonatou E, Stumpe M, Steenbock H, Grossi S, Beer HD, Angel P, Brinckmann J, Kletsas D, Dengjel J, Werner S. Nrf2-Mediated Fibroblast Reprogramming Drives Cellular Senescence by Targeting the Matrisome. Dev Cell. (2018) 46: 145-161.e10.

Corresponding authors: paul.hiebert@biol.ethz.ch and sabine.werner@biol.ethz.ch

Abstract: *Nrf2 is a key regulator of the antioxidant defense system, and pharmacological Nrf2 activation is a promising strategy for cancer prevention and promotion of tissue repair. Here we show, however, that activation of Nrf2 in fibroblasts induces cellular senescence. Using a combination of transcriptomics, matrix proteomics, chromatin immunoprecipitation and bioinformatics we demonstrate that fibroblasts with activated Nrf2 deposit a senescence-promoting matrix, with plasminogen activator inhibitor-1 being a key inducer of the senescence program. In vivo, activation of Nrf2 in fibroblasts promoted re-epithelialization of skin wounds, but also skin tumorigenesis. The pro-tumorigenic activity is of general relevance, since Nrf2*

activation in skin fibroblasts induced the expression of genes characteristic for cancer-associated fibroblasts from different mouse and human tumors. Therefore, activated Nrf2 qualifies as a marker of the cancer-associated fibroblast phenotype. These data highlight the bright and the dark sides of Nrf2 and the need for time-controlled activation of this transcription factor.

Zoppi N, Chiarelli N, Binetti S, Ritelli M, Colombi M. Dermal fibroblast-to-myofibroblast transition sustained by αvß3 integrin-ILK-Snail1/Slug signaling is a common feature for hypermobile Ehlers-Danlos syndrome and hypermobility spectrum disorders. Biochim Biophys Acta Mol Basis Dis. (2018) 1864: 1010-1023.

Corresponding author: nicoletta.zoppi@unibs.it

Abstract: *Hypermobile Ehlers-Danlos syndrome (hEDS) is a heritable connective tissue disorder with unknown molecular basis mainly characterized by generalized joint hypermobility, joint instability complications, and minor skin changes. The phenotypic spectrum is broad and includes multiple associated symptoms shared with chronic inflammatory systemic diseases. The stricter criteria defined in the 2017 EDS nosology leave without an identity many individuals with symptomatic joint hypermobility and/or features of hEDS; for these patients, the term Hypermobility Spectrum Disorders (HSD) was introduced. We previously reported that in vitro cultured hEDS and HSD patients' skin fibroblasts show a disarray of several extracellular matrix (ECM) components and dysregulated expression of genes involved in connective tissue homeostasis and inflammatory/pain/immune responses. Herein, we report that hEDS and HSD skin fibroblasts exhibit in vitro a similar myofibroblast-like phenotype characterized by the organization of α-smooth muscle actin cytoskeleton, expression of OB-cadherin/cadherin-11, enhanced migratory capability associated with augmented levels of the ECM-degrading metalloproteinase-9, and altered expression of the inflammation mediators CCN1/CYR61 and CCN2/CTGF. We demonstrate that in hEDS and HSD cells this fibroblast-to-myofibroblast transition is triggered by a signal transduction pathway that involves αvβ3 integrin-ILK complexes, organized in focal adhesions, and the Snail1/Slug transcription factor, thus providing insights into the molecular mechanisms related to the pathophysiology of these protean disorders. The indistinguishable phenotype identified in hEDS and HSD cells resembles an inflammatory-like condition, which correlates well with the systemic phenotype of patients, and suggests that these multisystemic disorders might be part of a phenotypic continuum rather than representing distinct clinical entities.*

Zoppi N, Chiarelli N, Ritelli M, Colombi M. Multifaced Roles of the αvβ3 Integrin in Ehlers-Danlos and Arterial Tortuosity Syndromes' Dermal Fibroblasts. Int J Mol Sci. 2018 19:pii: E982.

Corresponding author: nicoletta.zoppi@unibs.it

Abstract*: The αvβ3 integrin, an endothelial cells' receptor-binding fibronectin (FN) in the extracellular matrix (ECM) of blood vessels, regulates ECM remodeling during migration, invasion, angiogenesis, wound healing and inflammation, and is also involved in the epithelial mesenchymal transition. In vitro-grown human control fibroblasts organize a fibrillar network of FN, which is preferentially bound on the entire cell surface to its canonical α5β1 integrin receptor, whereas the αvβ3 integrin is present only in rare patches in focal contacts. We report on the preferential recruitment of the αvβ3 integrin, due to the lack of FN-ECM and its canonical integrin receptor, in dermal fibroblasts from Ehlers-Danlos syndromes (EDS) and arterial tortuosity syndrome (ATS), which are rare multisystem connective tissue disorders. We review our previous findings that unraveled different biological mechanisms elicited by the αvβ3 integrin in fibroblasts derived from patients affected with classical (cEDS), vascular (vEDS), hypermobile EDS (hEDS), hypermobility spectrum disorders (HSD), and ATS. In cEDS and vEDS, respectively, due to defective type V and type III*

collagens, αvβ3 rescues patients' fibroblasts from anoikis through a paxillin-p60Src-mediated cross-talk with the EGF receptor. In hEDS and HSD, without a defined molecular basis, the αvβ3 integrin transduces to the ILK-Snail1-axis inducing a fibroblast-to-myofibroblast-transition. In ATS cells, the deficiency of the dehydroascorbic acid transporter GLUT10 leads to redox imbalance, ECM disarray together with the activation of a non-canonical αvβ3 integrin-TGFBRII signaling, involving p125FAK/p60Src/p38MAPK. The characterization of these different biological functions triggered by αvβ3 provides insights into the multifaced nature of this integrin, at least in cultured dermal fibroblasts, offering future perspectives for research in this field.

Chiarelli N, Carini G, Zoppi N, Ritelli M, Colombi M. Transcriptome analysis of skin fibroblasts with dominant negative COL3A1 mutations provides molecular insights into the etiopathology of vascular Ehlers-Danlos syndrome. PLoS One (2018) 13: e0191220.
Corresponding author: nicoletta.zoppi@unibs.it
Abstract*: Vascular Ehlers-Danlos syndrome (vEDS) is a dominantly inherited connective tissue disorder caused by mutations in the COL3A1 gene that encodes type III collagen (COLLIII), which is the major expressed collagen in blood vessels and hollow organs. The majority of disease-causing variants in COL3A1 are glycine substitutions and in-frame splice mutations in the triple helix domain that through a dominant negative effect are associated with the severe clinical spectrum potentially lethal of vEDS, characterized by fragility of soft connective tissues with arterial and organ ruptures. To shed lights into molecular mechanisms underlying vEDS, we performed gene expression profiling in cultured skin fibroblasts from three patients with different structural COL3A1 mutations. Transcriptome analysis revealed significant changes in the expression levels of several genes involved in maintenance of cell redox and endoplasmic reticulum (ER) homeostasis, COLLs folding and extracellular matrix (ECM) organization, formation of the proteasome complex, and cell cycle regulation. Protein analyses showed that aberrant COLLIII expression is associated with the disassembly of many structural ECM constituents, such as fibrillins, EMILINs, and elastin, as well as with the reduction of the proteoglycans perlecan, decorin, and versican, all playing an important role in the vascular system. Furthermore, the altered distribution of the ER marker protein disulfide isomerase PDI and the strong reduction of the COLLs-modifying enzyme FKBP22 are consistent with the disturbance of ER-related homeostasis and COLLs biosynthesis and post-translational modifications, indicated by microarray analysis. Our findings add new insights into the pathophysiology of this severe vascular disorder, since they provide a picture of the gene expression changes in vEDS skin fibroblasts and highlight that dominant negative mutations in COL3A1 also affect post-translational modifications and deposition into the ECM of several structural proteins crucial to the integrity of soft connective tissues.*

Beutel B, Song J, Konken CP, Korpos E, Schinor B, Gerwien H, Vidyadharan R, Burmeister M, Li L, Haufe G, Sorokin L., New in vivo compatible matrix metalloproteinase (MMP)-2 and MMP-9 inhibitors. Bioconjug Chem. (2018) Oct 24. doi: 10.1021/acs.bioconjchem.8b00618. [Epub ahead of print]
Corresponding authors: sorokin@uni-muenster.de and haufe@uni.muenster.de
Abstract: *Matrix metalloproteinases (MMPs) are emerging as pivotal fine tuners of cell function in tissue homeostasis and in various pathologies, in particular inflammation. In vivo monitoring of the activity of specific MMPs, therefore, provides high potential for assessing disease progression and tissue function, and manipulation of MMP activity in tissues and whole organisms may further provide a mode of controlling pathological processes. We describe here the synthesis of novel fluorinated and non-fluorinated analogues of a secondary sulfonamide-based lead structure, compound 2, and test their efficacy as in vivo*

inhibitors and tracers of the gelatinases, MMP-2 and MMP-9. Using a murine neuroinflammatory model, we show that compound 2 is a highly effective in vivo inhibitor of both MMP-2 and MMP-9 activity with little or no adverse effects even after long-term daily oral administration. A fluorescein-labelled derivative compound 17 shows direct binding to activated gelatinases surrounding inflammatory cuffs in the neuroinflammation model and to pancreatic β-cells in the islets of Langerhans, colocalizing with MMP-2 and MMP-9 activity as detected using in situ zymography techniques. These results demonstrate that compound 2 derivatives have potential as in vivo imaging tools and for future development for specific MMP-2 versus MMP-9 probes. Our chemical modifications mainly target the residues directed towards the S1' and S2' pockets and, thereby, provide new information on the structure-activity relationships of this inhibitor type.

Brauchle E, Kasper J, Daum R, Schierbaum N, Falch C, Kirschniak A, Schäffer TE, Schenke-Layland K., Biomechanical and biomolecular characterization of extracellular matrix structures in human colon carcinomas. Matrix Biol. (2018) 68-69: 180-193.
Corresponding author: katja.schenke-layland@med.uni-tuebingen.de
Abstract: *The extracellular matrix (ECM) is extensively remodeled in tumor tissues. Overproduction of collagens, pathological collagen crosslinking and alignment of fibers are major processes that ultimately result in an increased tissue stiffness. Although it is known that glycosaminoglycans (GAGs) play an important role in tumor signaling, their contribution to the biomechanical properties of tumor ECM is unknown. In this study, ECM structures of human colon carcinoma and normal (control) colon tissues were histologically identified. Using atomic force microscopy (AFM) nanoindentation, we show that the collagen-rich regions within the ECM of colon carcinoma tissues were significantly stiffer than the submucosal collagen-rich layer of control tissues. Screening of these regions with Raman microspectroscopy revealed significantly different molecular fingerprints for collagen fibers in colon carcinoma tissues compared to control tissues. We further showed an increased alignment of collagen fibers and elevated levels of GAG immuno-reactivity within the collagen network of colon carcinoma tissues. GAGs such as heparan sulfate and chondroitin sulfate were detected in significantly elevated levels in collagen fibers of carcinoma tissues. Moreover, immunodetection of the collagen-associated proteoglycan decorin was significantly decreased in carcinomas tissues of individual patients when compared with the corresponding control tissues. Overall a strong patient-to-patient variability was evident in the ECM composition, structure and biomechanics of individual colon carcinoma tissues. Although, biomechanical characteristics of tumor ECM were not directly impacted by GAG content, GAGs might play an important role during the mechanical and structural remodeling of pathological tumor ECM. To manipulate GAG expression and deposition in tumor microenvironments could represent a novel potential therapeutic strategy.*

Do NN, Willenborg S, Eckes B, Jüngst C, Sengle G, Zaucke F, Eming SA., Myeloid Cell-Restricted STAT3 Signaling Controls a Cell-Autonomous Antifibrotic Repair Program. J Immunol. (2018) 201: 663-674.
Corresponding author: sabine.eming@uni-koeln.de
Abstract: *Myeloid cells can be beneficial as well as harmful in tissue regenerative responses. The molecular mechanisms by which myeloid cells control this critical decision of the immune system are not well understood. Using two different models of physiological acute or pathological chronic skin damage, in this study we identified myeloid cell-restricted STAT3 signaling as important and an injury context-dependent regulator of skin fibrosis. Targeted disruption of STAT3 signaling in myeloid cells significantly accelerated development of pathological skin fibrosis in a model of chronic bleomycin-induced tissue injury, whereas the impact on wound closure dynamics and quality of healing after acute excision skin injury was minor.*

Chronic bleomycin-mediated tissue damage in control mice provoked an antifibrotic gene signature in macrophages that was characterized by upregulated expression of IL-10, SOCS3, and decorin. In contrast, in STAT3-deficient macrophages this antifibrotic repair program was abolished whereas TGF-β1 expression was increased. Notably, TGF-β1 synthesis in cultured control bone marrow-derived macrophages (BMDMs) was suppressed after IL-10 exposure, and this suppressive effect was alleviated by STAT3 deficiency. Accordingly, coculture of IL-10-stimulated control BMDMs with fibroblasts suppressed expression of the TGF-β1 downstream target connective tissue growth factor in fibroblasts, whereas this suppressive effect was lost by STAT3 deficiency in BMDMs. Our findings highlight a previously unrecognized protective role of myeloid cell-specific STAT3 signaling in immune cell-mediated skin fibrosis, and its regulatory pathway could be a potential target for therapy.

Firner S, Willwacher S, de Marées M, Bleuel J, Zaucke F, Brüggemann GP, Niehoff A., Effect of increased mechanical knee joint loading during running on the serum concentration of cartilage oligomeric matrix protein (COMP). J Orthop Res. (2018) 36: 1937-1946.
Corresponding author: niehoff@dshs-koeln.de
Abstract: *The purpose of the study was to investigate the effect of an increase in mechanical knee joint loading during running on the serum COMP level. On two different test days, 20 healthy men ran with knee orthoses for 30 min on a treadmill (v = 2.2 m/s). On day 1, the orthoses were passive, whereas on day 2 they were pneumatically driven (active) and thus increased the external knee flexion moments (+30.9 Nm) during stance phase. Lower-limb mechanics and serum COMP levels (baseline; 0, 0.5, 1, 2 h post running) were analyzed. COMP levels increased immediately after running with passive (+35%; pre: 7.5 U/l, 95%CI: 6.4, 8.7, post: 9.8 U/l, 95%CI: 8.8, 10.8, p < 0.001) and active orthoses (+45%; pre: 7.6 U/l; 95%CI: 6.4, 8.8, post: 10.3 U/l, 95%CI: 9.2, 11.5, p < 0.001), but they did not differ between interventions. While running with active orthoses, greater ankle dorsiflexion angles, knee flexion angles, and moments occurred (p < 0.05). Comparing both interventions, the Δ COMP pre-post, meaning the difference (Δ) between running with active and passive orthoses in pre to post COMP level change (=level after (post) running minus level before (pre) running), correlated negatively with Δ COMP baseline (difference between the baseline COMP level before running with active and passive orthoses, r = -0.616; p = 0.004), and with a positive tendence with the Δ maximum knee flexion (r = 0.388; p = 0.091). Therefore, changes in COMP concentration after physical activity seem to be highly influenced by the COMP baseline level. In addition, correlation analysis indicates that modifications in knee joint kinematics have a greater effect on cartilage metabolism than an increase in joint moments*.

Kastl P, Manikowski D, Steffes G, Schürmann S, Bandari S, Klämbt C, Grobe K., Disrupting Hedgehog Cardin-Weintraub sequence and positioning changes cellular differentiation and compartmentalization *in vivo*. Development (2018) 145(18).
Corresponding author: kgrobe@uni-muenster.de
Abstract: *Metazoan Hedgehog (Hh) morphogens are essential regulators of growth and patterning at significant distances from their source, despite being produced as N-terminally palmitoylated and C-terminally cholesteroylated proteins, which firmly tethers them to the outer plasma membrane leaflet of producing cells and limits their spread. One mechanism to overcome this limitation is proteolytic processing of both lipidated terminal peptides, called shedding, but molecular target site requirements for effective Hh shedding remained undefined. In this work, by using Drosophila melanogaster as a model, we show that mutagenesis of the N-terminal Cardin-Weintraub (CW) motif inactivates recombinant Hh proteins to variable degrees and, if overexpressed in the same compartment, converts them into suppressors of*

endogenous Hh function. In vivo, additional removal of N-palmitate membrane anchors largely restored endogenous Hh function, supporting the hypothesis that proteolytic CW processing controls Hh solubilization. Importantly, we also observed that CW repositioning impairs anterior/posterior compartmental boundary maintenance in the third instar wing disc. This demonstrates that Hh shedding not only controls the differentiation of anterior cells, but also maintains the sharp physical segregation between these receiving cells and posterior Hh-producing cells.

Lima AM, Wegner SV, Martins Cavaco AC, Estevão-Costa MI, Sanz-Soler R, Niland S, Nosov G, Klingauf J, Spatz JP, Eble JA., The spatial molecular pattern of integrin recognition sites and their immobilization to colloidal nanobeads determine α2β1 integrin-dependent platelet activation. Biomaterials (2018) 167: 107-120.

Corresponding author: johannes.eble@uni-muenster.de

Abstract: *Collagen, a strong platelet activator, is recognized by integrin α2β1 and GPVI. It induces aggregation, if added to suspended platelets, or platelet adhesion if immobilized to a surface. The recombinant non-prolylhydroxylated mini-collagen FC3 triple helix containing one α2β1 integrin binding site is a tool to specifically study how α2β1 integrin activates platelet. Whereas soluble FC3 monomers antagonistically block collagen-induced platelet activation, immobilization of several FC3 molecules to an interface or to colloidal nanobeads determines the agonistic action of FC3. Nanopatterning of FC3 reveals that intermolecular distances below 64 nm between α2β1 integrin binding sites trigger signaling through dot-like clusters of α2β1 integrin, which are visible in high resolution microscopy with dSTORM. Upon signaling, these integrin clusters increase in numbers per platelet, but retain their individual size. Immobilization of several FC3 to 100 nm-sized nanobeads identifies α2β1 integrin-triggered signaling in platelets to occur at a twentyfold slower rate than collagen, which activates platelet in a fast integrative signaling via different platelet receptors. As compared to collagen stimulation, FC3-nanobead-triggered signaling cause a significant stronger activation of the protein kinase BTK, a weak and dispensable activation of PDK1, as well as a distinct phosphorylation pattern of PDB/Akt.*

Nastase MV, Zeng-Brouwers J, Beckmann J, Tredup C, Christen U, Radeke HH, Wygrecka M, Schaefer L., Biglycan, a novel trigger of Th1 and Th17 cell recruitment into the kidney. Matrix Biol. (2018) 68-69: 293-317.

Corresponding author: schaefer@med.uni-frankfurt.de

Abstract: *Th1 and Th17 cells, T helper (Th) subtypes, are key inducers of renal fibrosis. The molecular mechanisms of their recruitment into the kidney, however, are not well understood. Here, we show that biglycan, a proteoglycan of the extracellular matrix, acting in its soluble form as a danger signal, stimulates autonomously the production of Th1 and Th17 chemoattractants CXCL10 and CCL20 in macrophages. In the presence of IFNγ, biglycan synergistically stimulates CXCL9. In macrophages deficient for TLR2, TLR4, and their adaptor molecules MyD88 or TRIF, we identified highly selective mechanisms of biglycan-dependent Th1/17 chemoattraction. Thus, the expression of CXCL9 and CXCL10, common chemoattractants for CXCR3-positive Th1 and Th17 cells, is triggered in a biglycan-TLR4/TRIF-dependent manner. By contrast, biglycan induces CCL20 chemokine production, responsible for CCR6-positive Th17 cell recruitment, in a TLR2/4/MyD88-dependent manner. Importantly, at the onset of diabetes mellitus and lupus nephritis we provide evidence for biglycan-dependent recruitment of Th1 and Th17 cells, IFNγ and IL-17 production, and development of albuminuria in mice lacking or overexpressing soluble biglycan. Furthermore, by genetic ablation of Cxcl10 we showed in vivo involvement of this chemokine in biglycan-dependent recruitment of Th1 and Th17 cells into the kidney. Finally, a positive correlation of biglycan and*

CXCL10/CXCL9 levels was detected in plasma from patients with diabetic nephropathy and lupus nephritis. Taken together, we identified biglycan as a novel trigger of Th1 and Th17 cell recruitment into the kidney and we postulate that interfering with biglycan/TLR/TRIF/MyD88-signaling might provide novel therapeutic avenues for renal fibrosis.

Niedermair T, Schirner S, Seebröker R, Straub RH, Grässel S., Substance P modulates bone remodeling properties of murine osteoblasts and osteoclasts. Sci Rep. (2018) 8: 9199.
Corresponding author: susanne.graessel@ukr.de
Abstract: *Clinical observations suggest neuronal control of bone remodeling. Sensory nerve fibers innervating bone, bone marrow and periosteum signal via neurotransmitters including substance P (SP). In previous studies we observed impaired biomechanical and structural bone parameters in tachykinin (Tac) 1-deficient mice lacking SP. Here, we aim to specify effects of SP on metabolic parameters of bone marrow macrophage (BMM)/osteoclast cultures and osteoblasts isolated from Tac1-deficient and wildtype (WT) mice. We demonstrated endogenous SP production and secretion in WT bone cells. Absence of SP reduced bone resorption rate, as we found reduced numbers of precursor cells (BMM) and multinucleated osteoclasts and measured reduced cathepsin K activity in Tac1-/- BMM/osteoclast cultures. However, this might partly be compensated by reduced apoptosis rate and increased fusion potential of Tac1-/- precursor cells to enlarged "super" osteoclasts. Contrarily, increased ALP enzyme activity and apoptosis rate during early osteoblast differentiation accelerated osteogenesis and cell death in the absence of SP together with reduced ALP activity of Tac1-/- osteoblasts during late osteogenic differentiation resulting in reduced bone formation at later stages. Therefore, we suggest that absence of SP presumably results in a slight reduction of bone resorption rate but concomitantly in a critical reduction of bone* formation and mineralization rate.

Probst K, Stermann J, von Bomhard I, Etich J, Pitzler L, Niehoff A, Bluhm B, Xu HC, Lang PA, Chmielewski M, Abken H, Blissenbach B, Machova A, Papadopoulou N, Brachvogel B., Depletion of Collagen IX Alpha1 Impairs Myeloid Cell Function. Stem Cells. (2018) Jul 31. doi: 10.1002/stem.2892. [Epub ahead of print]
Corresponding author: bent.brachvogel@uni-koeln.de
Abstract: *The trabecular extracellular matrix (ECM) forms a three-dimensional scaffold to stabilize the bone marrow, provide substrates for cell-matrix interactions and retain, present or release signals to modulate hematopoietic stem and progenitor cell development. However, the impact of trabecular ECM components on hematopoiesis has been poorly studied. Using collagen IX alpha1 - deficient (Col9a1(-/-)) mice, we revealed that a lack of collagen IX alpha1 results in a disorganized trabecular network enriched in fibronectin, and in a reduction in myeloid cells, which was accompanied by a decrease in colony-stimulating factor 1 receptor expression on monocytes from the bone marrow. In contrast, B-cell numbers in the bone marrow and T-cell numbers in the thymus remained unchanged. Alterations in the bone marrow microenvironment may not only reduce myeloid cell numbers, but also have long-term implications for myeloid cell function. Mice were infected with Listeria moncytogenes to analyze the function of myeloid cells. In this case, an inadequate macrophage-dependent clearance of bacterial infections was observed in Col9a1(-/-) mice in vivo. This was mainly caused by an impaired interferon-gamma/tumor necrosis factor-alpha-mediated activation of macrophages. The loss of collagen IX alpha1 therefore destabilizes the trabecular bone network, impairs myeloid cell differentiation, and affects the innate immune response against Listeria.*

Ruthard J, Hermes G, Hartmann U, Sengle G, Pongratz G, Ostendorf B, Schneider M, Höllriegl S, Zaucke F, Wagener R, Streichert T, Klatt AR., Identification of antibodies against extracellular matrix proteins in human osteoarthritis. Biochem Biophys Res Commun. (2018) 503: 1273-1277.

Corresponding author: andreas.klatt@uk-koeln.de

Abstract: *We investigated the presence of autoantibodies against the extracellular matrix proteins thrombospondin-4 (TSP-4), cartilage oligomeric matrix protein (COMP), C-type lectin domain family 3 member A (CLEC3A), collagen II, collagen VI, matrilin-3, and fibrillin-2 in the serum of osteoarthritis (OA) patients. We compared those results with the presence of such antibodies in rheumatoid arthritis (RA) patients and in healthy donors (HD). Our study examines whether antibodies against extracellular proteins can be used as potential biomarkers to support the clinical diagnosis of OA. 10 OA, 10 RA patients and 10 HD were enrolled in this explorative cross-sectional study. SDS-PAGE and immunoblot were used to investigate the presence of antibodies against extracellular matrix proteins. The serum of 5/10 OA patients but 0/10 HD exhibited TSP-4 IgG isotype antibodies (P = 0.033). The serum of 8/10 OA patients but only 1/10 HD exhibited IgG isotype antibodies against TSP-4 or COMP (P = 0.005). The serum of 9/10 OA patients but only 1/10 HD exhibited IgG isotype antibodies against TSP-4, COMP or CLEC3A (P = 0.005). We found strong evidence for the presence of IgG isotype autoantibodies against the cartilage extracellular matrix proteins TSP-4, COMP and CLEC3A in OA. The detection of IgG isotype autoantibodies against TSP-4, COMP and CLEC3A may support the clinical diagnosis of OA. OA with autoantibodies against cartilage extracellular matrix proteins defines a new OA subgroup suggesting that patients with high concentrations of autoantibodies may benefit from an immune suppressive therapy.*

Üstün Y, Reibetanz M, Brachvogel B, Nischt R, Eckes B, Zigrino P, Krieg T., Dual role of laminin-511 in regulating melanocyte migration and differentiation. Matrix Biol. (2018) Sep 29. pii: S0945-053X(18)30341-X. doi: 10.1016/j.matbio.2018.09.006. [Epub ahead of print]

Corresponding authors: yasemin.uestuen@uk-koeln.de and thomas.krieg@uni-koeln.de

Abstract*: Laminins are the major basement membrane (BM) components and are heterotrimers composed of an α, a β and a γ chain. In skin, laminins are present in basement membranes surrounding vascular structures, nerves, adipose tissue and in the specialized junctional BM between the epidermis and dermis. The main laminin isoforms in the dermo-epidermal BM are laminin-332, laminin-511 and laminin-211, the latter being restricted to hair follicles (HFs). The laminin γ1 chain is the most abundant γ chain; its global ablation in mice leads to early embryonic lethality at E5.5. To elucidate the cellular function of the γ1 chain in skin, we generated mice with keratinocyte-specific deletion of this chain (Lamc1EKO) by using the keratin (K)14-Cre/loxP system. These mice showed delayed coat pigmentation despite normal melanocyte counts in the skin. However, levels of differentiation-specific melanocyte enzymes TRP-1, TRP-2 and tyrosinase were reduced in Lamc1EKO mice, and melanocytes failed to migrate to their differentiation niche in HFs and accumulated in the IFE. These results suggested that the pigmentation defect results from impaired melanocyte migration. The impaired migratory capacity of melanocytes is due to the altered composition of laminins in the BM of Lamc1EKO mice: Loss of keratinocyte-derived pro-migratory laminin-511 is not compensated by ectopically deposited fibroblast-derived laminin-211. Furthermore, contact of melanocytes with recombinant laminin-511, but not with laminin-211, induces the expression of the chemokine receptor CXCR4 on melanocytes, needed for SDF-1 (stromal cell-derived factor-1)-mediated migration into HFs. We here demonstrate that laminin-511 controls the differentiation of melanocytes by regulating their migration from the epidermis into HFs and by activating CXCR4 expression on melanocytes required for their recruitment into HFs in an SDF-1-dependent manner.*

ISMB MEMBERSHIP: BECOME A MEMBER OF ISMB!

ISMB is dedicated to promoting matrix biology research on a global scale and to facilitating communication among matrix-related organizations and researchers from different countries. Members are eligible for discounted registration fees at matrix meetings supported by ISMB. The Society sends out newsletters highlighting recent research advances, descriptions of matrix biology resources, new appointments and awards, together with announcements of relevant meetings.

Every two years, the Society presents the Rupert Timpl Award to a young scientist (<40 years old) for the best paper related to matrix biology published in the previous two years and gives the Distinguished Investigator Award for lifetime achievement in the field of matrix biology. ISMB sponsors travel grants for young scientists to attend international matrix meetings. If you work in the matrix biology area, consider becoming a member of ISMB to support the international matrix community and give your input on ways to improve interactions and communication. See the website www.ismb.org to join, and for recent job postings and newsletters.

Welcome to new members of ISMB since May 2018

PhD students
Morten Jensen, University of Southern Denmark, Odense, DENMARK
Yazmin Brown, University of Newcastle, Adamstown, AUSTRALIA
Silvia Redondo Garcia, University of Granada, Granada, SPAIN
Ivor Geoghegan, National University of Ireland, Galway, IRELAND
Rocco Bernasconi, Universitat Klinikum Freiburg, Freiburg, GERMANY
Rebecca Dodd, University of Manchester, Manchester, UNITED KINGDOM
Conor Sugden, University of Liverpool, Liverpool, UNITED KINGDOM
Carina Blaker, University of Sydney, St Leonards, AUSTRALIA
Chieh Yu, Inst. of Health and Biomedical Innovation, Brisbane, AUSTRALIA

Post-doctoral fellows
Fiona Kenny, King's College London, London, UNITED KINGDOM
Jessica Llewellyn, University of Pennsylvania, Philadelphia, USA
Jeremy Herrera, University of Manchester, Manchester, UNITED KINGDOM
Franziska Uhl, University of Vermont, Burlington, USA
Vishal Chaturvedi, University of Melbourne, Melbourne, AUSTRALIA

Senior scientists
Mahbub Shihan, Research Assistant, Univeristy of Delaware, Newark, USA
Victor Leung, Assistant Professor, University of Hong Kong, Hong Kong, CHINA
Kim Midwood, Professor, University of Oxford, Oxford, UNITED KINGDOM
Tom Barker, Professor, University of Virginia, Charlottesville, USA
Ida Gjervold Lunde, Group Leader, Oslo University Hospital, Oslo, NORWAY

MATRIX MEETING ANNOUNCEMENTS

Matrix Biology Ireland (MBI) 2018 meeting will be held from the 21st to the 23rd of November 2018 at Galway (Ireland). The theme of the meeting is 'Matrix Pathophysiologies and Reparative Therapies'.

To submit your abstract, please download, complete and submit the attached form to mbi@nuigalway.ie
http://www.mbi.ie/meeting-2018/home

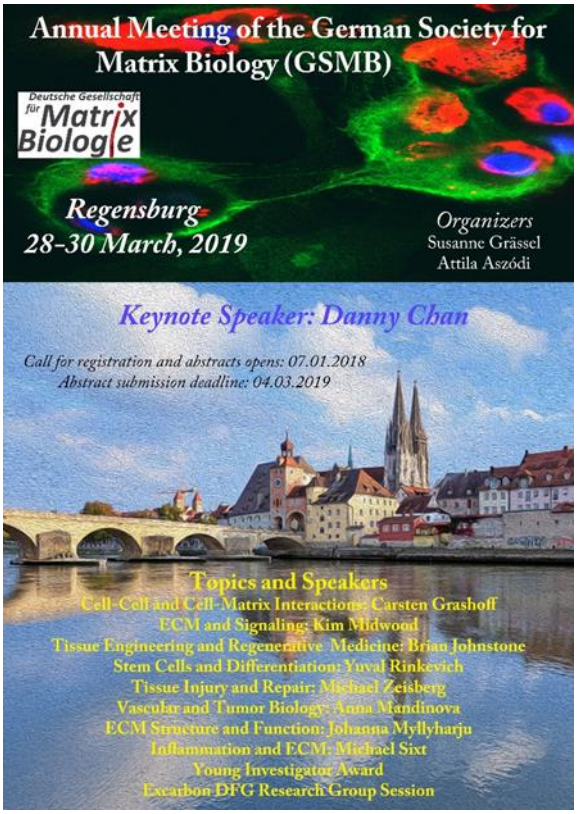
Matrix Biology Society of Australia and New Zealand 2018 Annual Meeting, December 4-7, Auckland (New Zealand)

Returning to New Zealand after nearly 20 years, it is our great pleasure to invite you to attend MBSANZ18 in Auckland. Program themes include Matrix Mechanobiology, Neuroscience, Matrix in Disease, Imaging the Matrix, Therapeutics, and Biomaterials.

Being held in tandem with the University of Auckland 10th Annual Mechanobiology Symposium and the 11th Annual Australasian Biomechanics Conference, this year's meeting will bring a mix of engineers, clinicians and biologists together for a highly interdisciplinary scientific exchange.

This year we will also be hosting one day of the conference amongst the vines of the stunning Goldie Estate winery on Waiheke Island, nestled in the Hauraki Gulf 40 minutes from downtown Auckland. This will also be the venue for the conference dinner.
For a full list of plenary and invited speakers, as well as registration details, please visit http://mbsanz18.nz

Annual meeting of the German Society for Matrix Biology (GSMB), 28-30 March, 2019, Regensburg (Germany)
http://www.matrixbiologie.de/JahrestagungRegensburg_2019/side_docs/TopicsDGMB2019.pdf
http://www.matrixbiologie.de/JahrestagungRegensburg_2019/side_docs/PrelimProg0918.pdf



Gordon Research Conference on Cartilage Biology and Pathology
Chairs: Yingzi Yang and John Bateman
March 17-22, 2019, Galveston, TX (USA)
The GRC will be the ninth in a series of highly successful biennial conferences, and it will be preceded for the third time by a GRS, a pre-meeting exclusively dedicated to and organized by trainees. We expect approximately 200 attendees from North America, Europe, Asia, and Australia

Session Topics and major speakers
Skeletal Growth *Discussion Leader* Andrea Vortkamp (University of Duisburg-Essen, Germany)
Speakers
Kim Cooper (University of California, San Diego, USA)
Elazar Zelzer (Weizmann Institute of Science, Israel)
Oliver Pourquie (Harvard Medical School, USA)

Cartilage Development
Discussion Leader Henry Kronenberg (Massachusetts General Hospital / Harvard Medical School, USA)
Speakers
David Ornitz (Washington University in St. Louis, USA)
Karen Lyons (University of California, Los Angeles, USA)
Veronique Lefebvre (Lerner Research Institute, Cleveland Clinic, USA)
Ernestina Schipani (University of Michigan, USA)

Genetic and Epigenetic Programming
Discussion Leader Andrew Lassar (Harvard Medical School, USA)
Speakers
Regis O'Keefe (Washington University in St. Louis, USA)
Shinsuke Ohba (University of Tokyo, Japan)

Mechanobiology of Cartilage and Joints
Discussion Leader Farshid Guilak (Washington University in St. Louis, USA)
Speakers
Tamara Alliston (University of California, San Francisco, USA)
Kate Poole (University of New South Wales, Australia)
Robert Mauck (University of Pennsylvania, USA)

Cartilage Matrix
Discussion Leader Danny Chan (The University of Hong Kong, Hong Kong SAR China)
Speakers
Martin Lotz (The Scripps Research Institute, USA)
Andrew Rowan (Newcastle University, United Kingdom)

Genetic Cartilage Disease
Discussion Leader
Michael Briggs (Newcastle University, United Kingdom)

Speakers
Brendan Lee (Baylor College of Medicine, USA)
Maurizio Pacifici (The Children's Hospital of Philadelphia, USA)
Matthew Warman (Boston Children's Hospital / Harvard University, USA)

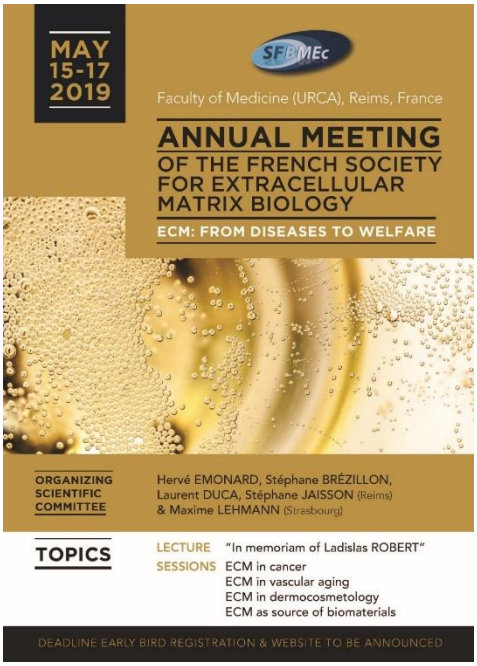
Cartilage Degeneration
Discussion Leader Benjamin Alman (Duke University, USA)
Speakers
Frank Beier (University of Western Ontario, Canada)
Joyce van Meurs (Erasmus MC, The Netherlands)

Stem Cells and Cartilage Regeneration
Discussion Leader Christine Hartmann (University of Münster, Germany)
Speakers
Hiroshi Asahara (Tokyo Medical and Dental University, Japan / The Scripps Research Institute, USA)
Deneen Wellik (University of Michigan Medical School, USA)
Xu Cao (Johns Hopkins University, USA)

New Approaches to Cartilage Therapies
Discussion Leader Deborah Krakow (University of California, Los Angeles, USA)
Speakers
Raymond Boot-Handford (Wellcome Trust Centre for Cell-Matrix Research, United Kingdom)
Jacqueline Hecht (The University of Texas Health Science Center at Houston, USA)

Additional short talks in each session will be selected from abstracts.
To apply for registration and further details – visit https://www.grc.org/cartilage-biology-and-pathology-grs-conference/2019/
International travel grants for young scientists may be available from ISMB to attend this meeting. Applications should be made directly to ISMB at www.ismb.org

Annual meeting of the French Society for Matrix Biology, May 15-17, 2019, Reims (France)
www.meeting-sfbmec.fr



We are pleased to announce the **Tissue Engineering & Regenerative Medicine International Society (TERMIS) EU Chapter 2019 Meeting, 27th to 31st of May 2019, Rhodes (Greece)**.
The TERMIS EU 2019 meeting will be held at Rhodes, Greece. Rhodes, the Island of Knights, is a place with spectacular natural beauty and abundant history and culture. Rhodes hosts the mythical Colossus of Rhodes, one of the Seven Wonders of the Ancient World. The Medieval Old Town of the City of Rhodes is listed as a UNESCO World Heritage site.
TERMIS EU 2019 invites symposia proposals in all areas of tissue engineering and regenerative medicine research, development and clinical translation. Symposia proposals with educational, career development and societal impact are welcomed. If you are interested in submitting a symposium proposal, please complete the attached form and submit it to termis@nuigalway.ie by the **28th of September 2018**. TERMIS EU 2019 also offers various sponsoring and exhibiting opportunities. For further information, please contact us at termis@nuigalway.ie.
We are honoured to host the TERMIS EU 2019 meeting and we are looking forward to welcoming you at Rhodes.

Sincerely,
Dimitrios I. Zeugolis, PhD and Maria Chatzinikolaidou, PhD

ASMB 2019 Workshop on Fibroblasts, The Arbiters of Extracellular Matrix Remodeling
June 23-25, 2019, University of Virginia, Charlottesville, VA (USA)

Co-organized by Tom Barker (University of Virginia, USA) and Merry Lindsey (University of Nebraska Medical Center, USA)

Fibroblasts are an enigmatic and diverse population of cells with stromal origins. It is believed that their primary function is the maintenance and turnover of the extracellular matrix (ECM). Through the coordinated degradation and assembly of ECM they maintain normal ECM homeostasis, which in turn impacts cell and tissue homeostasis through direct support and signaling of resident cells. It has become increasingly clear that fibroblasts are extremely diverse and heterogeneous even within a single microenvironment and that their functions are far more diverse than simply maintaining ECM tone. In response to a single stimulus, a single population of fibroblasts may diverge into distinct subpopulations, often through unknown mechanisms. This diversity and emergence of subpopulations likely has a tremendous impact on wound healing and ECM-centric pathologies, such as cancer, cardiovascular disease, and fibrosis. Yet our understanding of fibroblasts, their classifications, their origins, and how those origins impact tissue biology is in its infancy. This program will bring together the fibroblast and the extracellular matrix biology communities to explore current research and theoretical concepts in fibroblast biology.

For logistics and event information: Samantha Clarke (snc8uh@virginia.edu)
For information about the conference content: Tom Barker (thomas.barker@virginia.edu)

ASMB Workshop on Basement Membranes
July 10-12, 2019, Vanderbilt University Medical Center, Nashville, TN (USA)

Gordon Research Seminar on Collagen, July 13-14, 2019
Colby-Sawyer College, New London, NH (USA)

Chairs: Wing Ying Chow, Charlotte A Gistelinck
Decoding Collagen: From Molecular Ensembles to Tissue Scaffolds in Physiology and Disease
Weblink: https://www.grc.org/collagen-grs-conference/2019/

The 5th Collagen Gordon Research Seminar (GRS) is an opportunity for graduate students, postdocs, and other scientists of comparable experience to present their work and exchange ideas in a supportive setting prior to the Conference. Apart from a keynote speaker and talks selected from abstract submissions, the GRS will also have a mentorship component in the form of a discussion panel focusing on career development. We look forward to meeting the next generation of collagen researchers, as well as nurturing multidisciplinary collaborations. For enquiries, feel free to contact the chairs via email (chow@fmp-berlin.de, charlink@uw.edu) or Twitter (@wingyingchow, @char2link, #collagenGRS2019).

Gordon Research Conference on Collagen, July 14-19, 2019
Colby-Sawyer College, New London, NH (USA)
Chair: Marion K. Gordon, Vice Chair: Taina Pihlajaniemi
Collagens and Associated Molecules Supporting Organ Structure and Function
There are several funding schemes of particular interest to underrepresented minorities, attendees from certain countries, and predominantly undergraduate institutions. Please see https://www.grc.org/about/grc-diversity-initiatives/ for more information

FASEB Science Research Conference Series
Matricellular Proteins in Tissue Remodeling and Inflammation, July 14 - 19, 2019, Lisbon (Portugal)
CONFERENCE ORGANIZERS: Olga Stenina-Adognravi, Cleveland Clinic (USA) and Joanne Murphy-Ullrich, University of Alabama at Birmingham (USA)
http://faseb.org/Science-Research-Conferences/About-FASEB-SRC/Upcoming-Conferences.aspx

11th International Conference on Proteoglycans, September 29 - October 3, 2019, Kanazawa (Japan)

Dear ISMB members,

On behalf of the Organizing Committee, we would like to invite you to the 11th International Conference on Proteoglycans, scheduled on September 29 (Sun) - October 3 (Thurs) 2019 in Kanazawa (Japan). The Conference is a biennial world meeting held in different continents, which gives the opportunity to share new results. It is an informal meeting where experienced and young researchers can interact with each other. We believe your participation will develop the research field of proteoglycans.

For further information, please visit www.proteoglycans2019.com

Hideto Watanabe and Shuhei Yamada
Chairs of the 11th International Conference on Proteoglycans