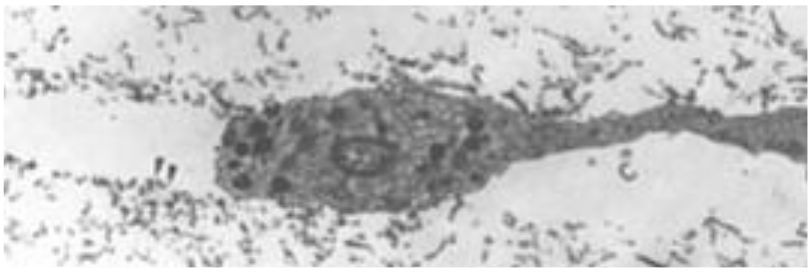


A fibroblast within a tethered lattice, after 13 days, with a cavity surrounding the cell and areas of packed collagen fibrils *(Porter et al. 1998 Wound Rep. Reg. 6: 157-166)*

ISMB NEWSLETTER 45 October 2023 Editor: Sylvie Ricard-Blum

FROM THE PRESIDENT'S DESK

Dear ISMB members, friends, and colleagues,

With heavy hearts, we open this edition of our ISMB newsletter, bearing somber news of the untimely passing of our esteemed colleague and dear friend, Ulrich auf dem Keller. Our thoughts and condolences are with his family during this difficult time.

The fundamental mission of ISMB remains steadfast: to foster global research in extracellular matrix biology and to support the professional growth of early-career researchers. It's heartening to note that one-third of our members are junior researchers, and our society continues to thrive, having welcomed over 40 new members since 2022. We kindly encourage you to share this newsletter with your students, post-doctoral fellows, and fellow matrix biology colleagues, helping us extend the reach of ISMB and further enrich our vibrant and dynamic community.

We are pleased to announce the formation of a new Outreach Committee, focused on brainstorming initiatives to engage our younger members. We eagerly await the innovative ideas and projects that will emerge from this committee's efforts. As such, we invite motivated junior matrix researchers interested in enhancing ISMB's visibility and presence on social media and our website to reach out to the team coordinator, Valerio Izzi (valerio.izzi@oulu.fi).

In support of junior scientists attending international meetings relevant to matrix biology, ISMB offers international travel grants for ISMB members. Applications can be submitted directly through our website (https://www.ismb.org/copy-of-travel-grants). Please mark your calendars for the upcoming application deadline on January 1st, 2024. A support to graduate students and early career scientists is offered by the Triple Helical Peptides Ltd, which launches Collagen Ligands competition 2023 and will offer Collagen Ligand plates for collagens II and III to the winner. Please participate!

Within this final issue of 2023, we are thrilled to highlight the matrix-related conferences scheduled for the year and provide a glimpse of the exceptional events lined up for 2024. One notable event is the Matrix Biology Europe conference, organized by the French Society for Matrix Biology, scheduled for September 24-27, 2024, in Lyon.

This conference will also feature the presentation of the Rupert Timpl Award for outstanding contributions in matrix biology research and the Dick Heinegård Young Investigator Award. Keep an eye out for nomination calls! A new addition to the program is a pre-meeting organized by junior researchers for their peers, offering an invaluable opportunity for networking, reconnecting with old friends, and forging new connections. We encourage all our friends to visit the conference's website for further details (https://mbe2024.sciencesconf.org/).

As you dive into this edition of the ISMB newsletter, we extend our gratitude to our newsletter editor, Sylvie Ricard-Blum, for consistently maintaining the high quality and success of our newsletters.

Nikos Karamanos, ISMB President (n.k.karamanos@upatras.gr), and
Julia Etich, ISMB Secretary (julia.etich@uni-koeln.de)

Content

Obituary - Ulrich auf dem Keller (1974-2023)

With great sadness, we received the sudden, incomprehensible and saddening announcement of the passing of Professor Dr. Ulrich auf dem Keller in Copenhagen this August. Ulrich's untimely death came as a shock to his family, friends, and colleagues, and we will all miss him beyond words. We accompanied Ulrich through the different stages of his life and career as a mentee, collaborator, colleague, teacher, and mentor – but most importantly as a friend.

Ulrich was a world leader in the fields of wound healing and proteinase biology and a pioneer in mass spectrometry-based proteomics technologies. He has published over 100 highly cited papers on the discovery of matrix metalloproteinase (MMP) substrates and global analysis of proteolysis in healing skin wounds. From 2017, Ulrich was a board member of the European Tissue Repair Society (ETRS), a supporting society of Wound Repair and Regeneration that he served as editorial board member. From 2019-2022, he elegantly and efficiently maneuvered the ETRS through the most difficult period of the COVID-19 pandemic as the president of the society. He was also a member of the SKINTEGRITY.CH research consortium, which he supported through multiple collaborations and contributions to scientific events, and served as the president of the International Proteolysis Society from 2017-2019. His sudden death prematurely stopped his remarkable career on a steep upward trajectory, and we can only imagine what he would have achieved in the upcoming years. Ulrich auf dem Keller's research work will continue to inspire scientists to study wound healing, protease biology, and proteomics for generations to come.

Ulrich was raised in Mülheim an der Ruhr, Germany. He spoke Latin and ancient Greek. Following his passion for science, he studied Biochemistry in Tübingen, Germany, and received his Diploma in Biochemistry in 2000. During that period, he spent one year at the Max-Planck-Institute of Biochemistry in Martinsried outside Munich to perform internships in several labs, including the lab of Sabine Werner. Their first encounter was the beginning of a long-term friendship and collaboration. Ulrich subsequently performed his Ph.D. studies in Sabine's lab, which by then had moved to the Swiss Federal Institute of Technology (ETH) Zurich, Switzerland. As a Ph.D. student, he discovered a key function of the cytoprotective transcription factor NRF2 in the prevention of skin carcinogenesis and additionally studied the role of NRF2 in wound healing. Ulrich received his Ph.D. in 2005.

During his Ph.D., Ulrich developed an enthusiasm for quantitative systems biology when analyzing the NRF2 transcriptome in keratinocytes. To follow this new passion, he decided to join the laboratory of Christopher Overall in Vancouver, Canada, in 2006 where he learned innovative proteomics technologies and combined them with biomedically relevant questions. In the Overall lab, as the recipient of a research fellowship from the German Research Foundation (DFG), Ulrich studied the role of proteases in cancer and co-developed iTRAQ-TAILS, a proteomics technology that allows the identification of neo-N-termini of proteins, and he used TAILS to address many important biological questions. His Post-Doctoral work led to 25 publications, including his most significant in Science Signalling on protease networks in skin inflammation in wild-type and Mmp2-/- mice. After his return to the ETH in Zurich in 2009, Ulrich established an independent research group, now combining his expertise in wound healing research and proteomics, to identify MMP cleavage events during tissue repair in mice and humans.

In 2017, Ulrich was recruited by the Technical University of Denmark (DTU) Bioengineering in Lyngby. Supported by a Young Investigator Award from the Novo Nordisk Foundation, he established his research program on

proteolytic networks in skin homeostasis, inflammation, and repair with a focus on MMPs. At the DTU, Ulrich quickly became a highly appreciated leader, first with his appointment to Associate and then to Full Professor and head of the Section for Protein Science and Biotherapeutics, Bioengineering, Department of Biotechnology and Biomedicine. The obituary released by his university perfectly summarizes his personal approach to science and leadership: *"[…] with his visionary and yet modest approach to leadership he has set an example for us all. In countless situations he has put his colleagues' needs before his own, and through his kindness, always managed to bring out the best in people. Ulrich had an ability to identify exceptional talent regardless of their career stage, when recruiting highly skilled scientists as Ph.D. students, postdocs, and group leaders*."

With his charisma, Ulrich was a source of contagious inspiration and positive energy to his colleagues and mentees. For six continuous years, he was responsible for impressive growth in his section and recruited numerous talented group leaders all sharing the desire to be mentored by him. The career trajectories of his trainees are, first and foremost, proof that his students and postdocs enormously benefitted from Ulrich`s skills and his excellent mentorship. Through his friendly, modest, and positive personality, he was liked by everybody, and he became a role model for many young investigators to whom he demonstrated that scientific success can be combined with family life and hobbies. This included his passion for classical music; Ulrich was an outstanding viola player who played in different orchestras and music groups. In Vancouver, on arrival for his postdoctoral experience, he instantly was made 1st Viola in the Vancouver Symphony Orchestra. Scandinavians also got to enjoy the tones of his viola. His virtuous performances in the orchestra were matched, if not topped, by his presentations as a gifted speaker at leading conferences in his field. Ulrich was an outstanding presenter who most actively contributed not only to the official discussions but also to networking events and trainee discussion forums.

In addition to his leadership and teaching roles, Ulrich's research group and program within Protease Systems Biology at DTU provided exciting insight into the temporal and spatial regulation of proteases in tissue repair and identified novel regulators of the healing process. His contributions were pivotal in understanding the skin protease network, where he focused on MMPs and kallikreins. He was also leading the international research in understanding the interplay between posttranslational modifications and protease activity in the extracellular space, with hallmark works on glycosylation and phosphorylation. Complementing his interest in basic mechanistic questions, Ulrich became increasingly fascinated by the complex problem of non-healing wounds. He extended his research to translational studies, which he performed in extremely productive and joyful collaborations with Hans Smola and Magnus S. Ågren. Ulrich's generous contributions to collaborations with us and others on a wide variety of different research topics resulted in publications that would not have been possible without his skills, knowledge, and ideas. His most recent work identified innovative and promising biomarkers for normally healing *versus* non-healing human wounds, paving the way for the development of new treatments to restore the healing of chronic wounds. In his pledge to apply for membership on the ETRS board in 2017, he stated, "*By becoming a member of the ETRS board, I want to give something back to the society and help in maintaining and further expanding this outstanding network of researchers dedicated to tackle a major unmet medical need with devastating impact on the quality of life of an increasing number of people*." And he did.

We have all been extremely privileged and fortunate to interact with Ulrich both professionally and in our private lives. It is impossible to fill the void he leaves, and we cannot begin to grasp his loss for his family. Most importantly, Ulrich was a devoted husband, father of two children, Florian and Julia, brother and son, and our thoughts go out to his family in this period of grief.

Magnus S. Ågren
Konstantinos Kalogeropoulos
Hans Smola
Boris Hinz
Christopher M. Overall
Sabine Werner

News from national societies

Chinese Society for Matrix Biology

The 7th National Conference on Matrix Biology was successfully held on August 2-5, 2023 in Lishui City, Zhejiang Province, China. The conference was hosted by the Chinese Society of Matrix Biology of Chinese Association for Physiological Sciences, the Hangzhou Institute for Advanced Study of University of Chinese Academy of Sciences, and the School of Life Sciences of Zhejiang University.

More than 160 scholars and students from all over the country, gathered together to participate in the grand event. The conference focused on the interaction between cell microenvironment and stem cell differentiation, embryonic development, tissue regeneration, transmembrane signal transduction, and biomechanical sensing from multiple perspectives, as well as their applications in tumors, aging, cardiovascular diseases and inflammation, etc. The conference provided an excellent learning and communication platform for participating teachers and students through conference reports, young scientists forums and poster exchanges.To encourage and support the development of young talents, the conference specially established awards for young scientist reports and posters, granting a stage for young PIs, postdoctoral fellows and graduate students to showcase their work.

The conference spanned 4 days, and the venue was fully occupied. The academic atmosphere was warm and friendly, providing a platform for matrix biology researchers and clinical workers to collide ideas, exchange and cooperate. In addition, Chinese Society of Matrix Biology collaborated with the journal "Chemistry of Life" to organize a special issue with the theme of "Cells and Microenvironment" specifically for this conference, including more than 20 high-level reviews focusing on cell sensing of matrix mechanic signaling, the physiological function of the interaction between cell adhesion molecules and extracellular matrix in the microenvironment in stem/progenitor cells, as well as cell microenvironment in cardiovascular diseases, cartilage damage, liver disease and tumors.

The President of the CSMB is Professor Jianfeng Chenjfchen (jfchen@sibcb.ac.cn).

British Society for Matrix Biology (BSMB) and Biochemical Society *89th* Harden *Conference - Proteoglycans: Matrix Master Regulators 2023 report* *by Chloé Yeung*

On 4th September, I travelled from Copenhagen, Denmark to Surrey, United Kingdom for the British Society for Matrix Biology (BSMB) and Biochemical Society *89th* Harden *Conference - Proteoglycans: Matrix Master Regulators 2023 at De Vere Horsley Estate. The special 4-day joint meeting was coordinated by leading proteoglycan and glycosaminoglycan (GAG) researchers from across the UK, led by Jerry Turnbull (University of Liverpool). The meeting program focussed on cutting edge research areas of the field and opened with a keynote lecture from Jeffrey Esko (UCSD). The meeting was friendly, fast paced and covered the role of proteoglycans and GAGs in numerous areas of research, from basic to translational. Highlights from the meeting for me included the translational talk from Fransiska Malfait (Ghent University) on Ehlers Danlos Syndrome and the new technologies talk from Rebecca Miller (University of Copenhagen) on the visualisation of GAG structures.*

I attended the meeting because I was selected by the BSMB to receive this year's BSMB Early Career Researcher Award for my recent work on the human tendon circadian clock. I am really chuffed to have my work recognised by the BSMB and to be given a great opportunity to tell others about it!

L-R: Chloé Yeung, Andrew Pitsillides (Chair of the BSMB; Royal Veterinary College), John Couchman (University of Copenhagen), Linda Troberg (University of East Anglia), James Whiteford (Queen Mary University of London)

As part of the award, I delivered the John Scott lecture "Circadian-Regulated Matrix Homeostasis in Mouse and Human Tendons". My talk covered my work that started at The University of Manchester in the lab of Karl Kadler, and in collaboration with Qing-Jun Meng: the discovery of the tendon circadian clock in mouse tendons (PMID: 24897937), the establishment of the "chronomatrix" and the importance of the daily dynamics of this small pool of matrix on the maintenance of tendons and its function (PMID: 31907414) and role of matrix metalloproteinase 14 in collagen fibrillogenesis and turnover (bioRxiv 2022.04.01.486699). I then discussed the challenges of circadian research on human tendons, primarily the limitation of biopsy frequency due to sustained global gene activation by the biopsy procedure (PMID: 26769953) and the size of the tissue sample. Despite these challenges and in close collaboration with the clinicians at The Institute of Sports Medicine Copenhagen, we established that human patellar tendons exhibit a disrupted circadian rhythm with chronic tendinopathy *(PMID: 36810732). I will lead a clinical study to investigate whether time-of-day of exercise therapy influences clinical outcomes of patients with chronic patellar tendinopathy, funded by the Wu Tsai Human Performance Alliance at the Stanford University and the Joe and Clara Tsai Foundation and the Independent Research Fund Denmark.*

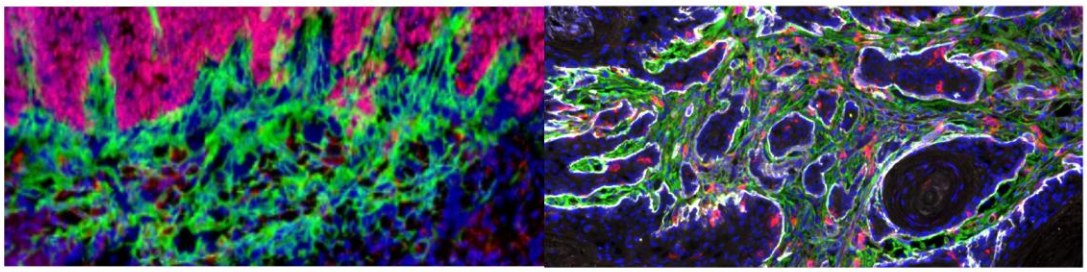
I thank the BSMB for their support since my PhD, and I encourage junior researchers to join local matrix societies to maximize networking opportunities and access travel awards for international conferences. I want to acknowledge the contribution of members of the Kadler lab and Meng lab and our numerous collaborators who contributed to the mouse tendon circadian clock studies. Last but certainly not least, I thank my mentor Michael Kjær and my colleagues at the Institute of Sports Medicine Copenhagen for their support in all my research endeavours.

Chloé Yeung is a Senior Researcher at the Institute of Sports Medicine Copenhagen, Bispebjerg Hospital Denmark and the Treasurer of the Danish Society for Matrix Biology. The DSMB Annual Meeting will take place on 30th-31st October 2023 at The University of Copenhagen. For more information, visit https://tinyurl.com/DSMB2023 Follow her on X: @ccstorytime.

PhD, post-doctoral and other positions

Postdoc Position available

To study Matrix and Immunity in Cancer



In the **INCa PLBIO** funded project **"Matrix-CAR-Targeting"** three research laboratories (**E. Donnadieu**, Institut Cochin, Paris, **C. Ferrand**, EFS, (French Blood Center), Besançon, **G. Orend**, INSERM U1109, Strasbourg) are collaborating in developing and applying novel tools to target the extracellular matrix for reprogramming the immune suppressive tumor microenvironment. Spatial distribution, migration and activation of engineered T cells will be investigated in spheroid cultures, ex vivo tumor tissue slices, and cancer progression models. Tumor immunity, growth and lung metastasis and tumor microenvironment properties will be determined by flow cytometry, tissue staining, cytokine array, RNA seq analysis and other technology in an environment with state of the art facilities.

A **24 months position (postdoc)** starting immediately is available in the **Tumor Microenvironment group** of **Gertraud Orend** (INSERM U1109, Strasbourg). This laboratory (**https://orend-tme-group.com**) is specialized in the analysis of the tumor microenvironment with particular emphasis on the extracellular matrix molecule **tenascin-C** (*Yimaz et al., 2022, **J Cell Sci**, Midwood et al., 2016, **J Cell Sci***). This laboratory has demonstrated pivotal roles of tenascin-C in orchestrating an immune suppressive tumor microenvironment (*Spenlé et al., 2020, **Cancer Immun Res**, Deligne et al., 2020, **Cancer Immun Res**, Spenle et al., 2021, **Front Immunol,** Murdamoothoo et al., 2021, **EMBO Mol Med,** Fonta et al., 2023, **Mat Bio***). In frame of this project the candidate will apply novel CAR T cells and the recently published MAREMO peptides (Loustau et al., 2022, ***Mat Bio***) and nanobodies (Dhaouadi et al., 2021, ***Front Immunol***).

We offer: **a highly dynamic and supportive group of colleagues including researchers, postdocs, PhD and master students and technical support with expertise in extracellular matrix research, murine tumor models and tumor immunity. The salary remuneration follows INSERM guidelines taking into account previous experience.**

We search: **a highly motivated scientist with strong background in tumor biology, mouse tumor models, immunology, immunohistochemistry and cell culture, high team spirit and good English communication skills.**

Interested candidates are invited to send their CV together with a motivation letter and the names of three referees to **Gertraud Orend (gertraud.orend@inserm.fr).**

Spotlights on methods that other ISMB members may find useful

If you would like to feature in the next newsletter, please email Julia Etich (julia.etich@uni-koeln.de)

In Focus

Translating genomic tools to Raman spectroscopy analysis enables high-dimensional tissue characterization on molecular resolution

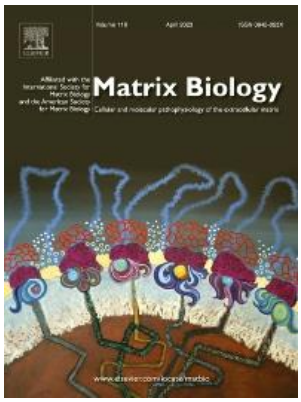
Sigle M, Rohlfing AK, Kenny M, Scheuermann S, Sun N, Graeßner U, Haug V, Sudmann J, Seitz CM, Heinzmann D, Schenke-Layland K, Maguire PB, Walch A, Marzi J, Gawaz MP. Nat Commun. 2023 14:5799.
doi: 10.1038/s41467-023-41417-0.

Corresponding author: Meinrad.Gawaz@med.uni-tuebingen.de

Spatial transcriptomics of histological sections have revolutionized research in life sciences and enabled unprecedented insights into genetic processes involved in tissue reorganization. However, in contrast to genomic analysis, the actual **biomolecular composition of the sample** has fallen behind, leaving a gap of potentially highly valuable information. **Raman microspectroscopy provides untargeted spatiomolecular information at high resolution, capable of filling this gap**. In this study we demonstrate spatially resolved **Raman "spectromics"** to reveal homogeneity, heterogeneity and dynamics of cell matrix on molecular levels by repurposing state-of-the-art bioinformatic analysis tools commonly used for transcriptomic analyses. By exploring sections of murine myocardial infarction and cardiac hypertrophy, we identify myocardial subclusters when spatially approaching the pathology, and define the surrounding metabolic and cellular (immune-) landscape. Our innovative, **label-free, non-invasive "spectromics" approach** could therefore open perspectives for a profound characterization of histological samples, while additionally allowing the combination with consecutive downstream analyses of the very same specimen.

Extracellular Matrix special Issues & journals

Special Issue (SI) Opportunities in Matrix Biology and Matrix Biology Plus



SI Matrix Biology Plus: Intersection of extracellular matrix biology and biomaterials design

This special themed issue will feature articles at the intersection of extracellular matrix biology and biomaterials design. Extracellular matrix based materials provide rich instructive cues for engineering cellular responses and directing tissue regeneration. Conversely, bottom-up approaches using synthetic materials to mimic extracellular matrices can provide deep understanding of mechanisms of ECM-cell interactions in driving complex physiological processes including embryogenesis, repair, and fibrosis. Articles featured in this issue will include both naturally derived ECM materials and ECM-mimetic synthetic scaffolds.

Topics of interest include ECM-based and ECM-mimetic materials for:

- Tissue Engineering and Regenerative Medicine
- Immunoengineering
- Engineering Cellular Microenvironments
- Controlling ECM/Stem Cell Interactions
- Probing mechanisms of tissue repair and/or fibrosis

Guest editors: Dr. Ashley Brown, North Carolina State University and University of North Carolina at Chapel Hill, USA and Dr. Daniel Alge Texas A&M University, USA

The Journal's submission system will be open for submissions to our Special Issue from May 01, 2023. When submitting your manuscript please select the article type "VSI: 'ECM and Biomaterials".

SI Matrix Biology: Fibroblasts and the Microenvironment

The focus of this special call is on the fibroblast and the microenvironment. The tissue microenvironment consists of a dynamic cellular population and non-cellular components, which form a multifaceted regulatory network that helps to maintain the homeostasis of an organ. Under pathological conditions, the

microenvironment undergoes alterations that can either sustain, improve, or worsen the injured/diseased tissues. We welcome submissions of original research articles, reviews, and perspectives on how the fibroblast microenvironment affects the extracellular matrix, cell-cell interactions, soluble factors, oxygen tension, and mechanical forces. Specifically, we are interested in manuscripts that explore the role of the ECM in regulating cell function in health or disease and how this can be targeted as a therapeutic strategy. We also encourage submissions that examine direct and indirect contact and/or signaling molecules that regulate ECM development, homeostasis, or repair in health or disease. We are interested in manuscripts that explore the regulation of cell behavior by secreted growth factors, the effects of oxygen tension on cell survival and function, as well as those that investigate how mechanical forces affect cell behavior and contribute to tissue development and remodeling. The overall aim of this special call is to highlight the importance of the fibroblast microenvironment and its effects on cell behavior during homeostasis, tissue development, and disease progression, in addition to fibroblast emergence as a novel therapeutic target. This special issue has been developed in cooperation with ASMB.

Guest Editors: Jeremy Simpson, University of Guelph; Co-Editors: Kristine Deleon-Pennell, Medical University of South Carolina, and Lisandra de Castro Brás, East Carolina University

Submission is now open! https://www.sciencedirect.com/journal/matrix-biology/about/call-for-papers#fibroblasts-and-the-microenvironment

"Matrix in Medicine" series published in Matrix Biology

Also, please consider submitting a review to the “Matrix in Medicine” series published in Matrix Biology. To date, 6 thought provoking reviews have been published. Check them out!

To highlight the importance of extracellular matrix and cell-matrix interactions in the pathogenesis of human disease, we are introducing a special section in Matrix Biology called “Matrix in Medicine.” Review articles published in the “Matrix in Medicine” series are intended for both scientists and clinicians and should emphasize the relevance of extracellular matrix to fundamental biological processes involved in human diseases. These articles will discuss the clinical aspects of a particular human disease with an emphasis on the underlying extracellular matrix-focused molecular mechanisms in the disease pathogenesis and/or potential for extracellular matrix targeted therapeutic interventions. The intent is to publish at least one “Matrix in Medicine” review in each of the 8 yearly issues of Matrix Biology.

Submissions should highlight the role of extracellular matrix in important clinical problems and will undergo rigorous peer review characteristic of all submissions to Matrix Biology. Articles should be submitted through the “Matrix in Medicine” submission option at the Matrix Biology website: https://www.sciencedirect.com/journal/matrix-biology.

Joanne E. Murphy-Ullrich, Editor-in-Chief of Matrix Biology and Matrix Biology Plus

October 5, 2023

Dear Proteoglycan Researchers,

I hope this finds you well. The journal is proceeding strongly. We have now 11 published papers, see below, and 4 in revision/accepted. I extend my deepest gratitude to the contributors for their unwavering support.

As I mentioned before, *Proteoglycan Research* considers both mechanistic and correlative papers including research articles, research reports, full- length and mini-reviews, perspectives, methods papers, and technical advances. Please view the Guide to Authors for more information. It would be a great opportunity if you could submit research papers from your lab and co-workers. If you are interested in writing a minireview or a perspective focused on emerging fields of research in proteoglycans, please contact me directly with your idea/proposal via e-mail to renato.iozzo@jefferson.edu. Also, ideas about special issues and guest editors are currently welcome.

The centrality of proteoglycan research: Expect the unexpected
Renato V. Iozzo
Proteoglycan Research Volume 1, Issue 1 e2, First Published: 27 March 2023

Extracellular vesicles released during hypoxia transport heparanase and enhance macrophage migration, endothelial tube formation and cancer cell stemness
Kaushlendra Tripathi, Shyam K. Bandari, Ralph D. Sanderson. Proteoglycan Research Volume 1, Issue 1 e1, First published: 28 March 2023

Glypicans, 35 years later
Jorge Filmus. Proteoglycan Research Volume 1, Issue 2, e5, First published: 10 May 2023

Functional organization of extracellular hyaluronan, CD44, and RHAMM
Mary K. Cowman, Eva A. Turley Proteoglycan Research Volume 1, Issue 2 e4, First published: 24 May 2023

Synthesis and optimization of collagen-targeting peptide-glycosaminoglycans for inhibition of platelets following endothelial injury
Michael Nguyen, Tanaya Walimbe, Andrew Woolley, John Paderi, Alyssa Panitch Proteoglycan Research Volume 1, Issue 2 e3, First published: 1 June 2023

Heparanase—A single protein with multiple enzymatic and nonenzymatic functions
Israel Vlodavsky, Yasmin Kayal, Maram Hilwi, Soaad Soboh, Ralph D. Sanderson, Neta Ilan
Proteoglycan Research Volume 1, Issue 3 e6, First published: 9 July 2023

The role of hyperglycemia-evoked intracellular hyaluronan accumulation and its activity on the autophagic and endoplasmic reticulum stress pathways
Andrew Wang, Aimin Wang, Vincent Hascall. Proteoglycan Research Volume 1, Issue 3 e7, First published: 26 July 2023

Sulfated motifs in heparan sulfate inhibit Streptococcus pneumoniae adhesion onto fibronectin and attenuate corneal infection
Atsuko Hayashida, Hajirah N. Saeed, Fuming Zhang, Yuefan Song, Jian Liu, William C. Parks, Paulo J. M. Bispo, Pyong Woo Park
Proteoglycan Research Volume 1, Issue 3 e9, First published: 9 August 2023

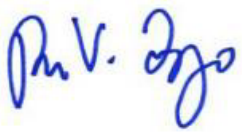
Functional and structural insights into human N-deacetylase/N-sulfotransferase activities
Sylvain D. Vallet, Thibault Annaval, Romain R. Vives, Emeline Richard, Jerome Hénault, Christine Le Narvor, David Bonnaffé, Bernard Priem, Rebekka Wild, Hugues Lortat-Jacob Proteoglycan Research Volume 1, Issue 3 e8, First published: 13 August 2023

The functional network of biglycan: A new frontier in tumor progression
Li Yu, Nako Maishi, Aya Matsuda, Kyoko Hida
Proteoglycan Research Volume 1, Issue 3 e11, First published: 11 September 2023

Interplay of heparan sulfate chains with the core proteins of syndecans 2 and 4
Martyna Maszota-Zieleniak, Adam Liwo, Sylvie Ricard-Blum, Sergey A. Samsonov
Proteoglycan Research Volume 1, Issue 3 e10, First published: 17 September 2023

I wish you a great fall and a warm winter, and I will see many of you soon at the ASMB meeting in Salt Lake City, Utah.

All the best,



Renato V. Iozzo, MD PhD
Editor-In-Chief, Proteoglycan Research

Triple Helical Peptides Ltd Collagen Ligands Competition 2023

Calling all Graduate Students and Early Career Researchers

Do you have a collagen-related research project you'd like to showcase? Would you like the opportunity to win £10,000 worth of the Collagen Ligands Collection to map the binding of your protein onto collagens II and III to elevate your research above the crowd?

The Collagen Ligands Competition is your opportunity to submit a short innovative research plan and in return receive:

- An article and feature bio on the Triple Helical Peptides (THP) website
- £10,000-worth of Collagen Ligand plates for collagens II and III
- Expertise from the scientists at THP who designed and created the Ligands.

Collagens are fundamental components of the extracellular matrix, and are highly interactive, with many partners within the ECM and several sets of receptors on the cell surface. Our new company, Triple Helical Peptides Ltd, makes ligands for these various proteins, and to celebrate our launch, we are offering free access through this competition to our Collagen Ligands Collection. These are synthetic peptides that were formerly distributed to the field as the Collagen Toolkits; libraries of overlapping triple-helical peptides, each containing 27 residues of collagen primary sequence. They come in 96-well plate format and comprise a comprehensive yet technically straightforward tool for mapping the binding of proteins onto collagens II or III. Our objectives are to discover new collagen-binding molecules, to map binding sites of known collagen-binding proteins within the fibrillar collagens, and to establish collaborative links with academic partners. We are inviting Graduate Students and Early Career Researchers***** to submit short collaborative projects that will make best use of our materials. The most exciting project outline will be rewarded with four copies of each Ligand library; each plate containing a complete Ligand set for either collagen II or III.

To enter, and for more information on Collagen Ligands Collection and other collagen-related peptides, please visit https://www.triplehelical.com

The deadline to submit your collaborative proposal is 29th December, 2023.
The winning entrant will be notified in January 2024. The Ligands Collection will be shipped shortly thereafter.

***** within 6 years of obtaining PhD

International travel grants

ISMB provides international travel grants (on average 500 Euros) for young scientists (graduate students or postdocs up to 5 years after Ph.D., with extensions for maternity leave, military service, etc) to allow them to attend major meetings in matrix biology anywhere in the world. While priority will be given to meetings directly supported by ISMB (including Matrix Biology Europe, the American Society for Matrix Biology and the Pan Pacific Connective Tissue Societies Symposium), applications are accepted for any meeting, provided that the scope of the meeting agrees with the aims of the Society.

Candidates should be members of the ISMB (see membership page for details), and a graduate student or postdoc. up to 5 years after Ph.D., with extensions for maternity leave, military service, etc.

Grants will be paid out in the form of reimbursement to grant awardees, after reception of proof of participation. To this aim, users must send a receipt of the expenses (e.g., meeting subscription fees, etc.) within 3 months from the end of the meeting they received support for.

To apply for a travel grant, please fill the the form available on ISMB web site (https://www.ismb.org/copy-of-travel-grants) and append a single pdf file containing:
(1) a letter giving information about the meeting, the amount requested and a detailed justification for support
(2) the abstract of your poster/short talk
(3) your curriculum vitae and list of publications.

Please apply several months in advance of the meeting, before one of the following deadlines: January 1, April 1, July 1, October 1

Meeting reports from ISMB International Travel Awardees

Cartilage Biology, Structure and Function: From Development to Regeneration, March 19 - 24, 2023, Lucca (Italy)
Due to the generous support of the ISMB, I could experience a productive and insightful meeting at the Gordon Cartilage Conference in 2023 in Italy. The idyllic and peaceful location supported the friendly atmosphere among researchers and fostered personal exchanges about various research topics surrounding cartilage.
Especially my role as discussion leader in the Research Seminar for young researchers allowed me to experience also this aspect of research and mediate in a passionate discussion. Furthermore, it was a really good experience to present my own research at a large but not extremely large conference with a neatly defined topic.
Because all the participants were housed at the same location as the conference, plenty of discussion time was also available during meals and in between sessions. I also liked the sessions about soft skill and research-life balance especially because I usually rarely talk about these topics with other researchers I meet at conferences.
I am really glad that I had the opportunity to join this meeting with the support of the ISMB. So thank you very much again for you generous support!

Alice Stephan, Center for Biochemistry, Medical Faculty, University of Cologne, Germany

Gordon Research conference 2023 Physiological and Disease Relevance and the Underlying Molecular Properties of Collagens. New London, NH, USA

I am delighted to share the exciting details of my recent participation in the renowned Gordon Conference on Collagen Biology. As a young postdoctoral researcher specializing in Collagen VI at the University of Padova under the guidance of Prof. Paolo Bonaldo, this conference marked a significant milestone in my scientific journey. It provided an invaluable opportunity for me to present my recent findings and immerse myself in the world of the extracellular matrix (ECM) and collagens.

During the conference, I had the privilege to present my recent results about a new mechanism involved in the turnover of Collagen VI in skeletal muscle. The opportunity to share my work with a distinguished audience of experts in the field was really exciting and the constructive suggestions that I received will undoubtedly propel my research project. The poster sessions were a particularly enriching aspect of the conference. These interactive sessions allowed me to engage in one-on-one conversations with peers, fostering an environment of collaboration and knowledge exchange. Out of all the conferences I've attended, this Gordon Conference stood out for its warm atmosphere of friendship and collaborative spirit.

I received invaluable input that not only refined the scope of my research but also provided me with fresh perspectives to explore. This conference experience has been instrumental in shaping my academic and professional journey. It has increased my passion for ECM and collagen biology and inspired me to continue pushing the boundaries of our understanding in this fascinating field. Moreover, the connections I got during this conference have opened doors to potential collaborations and mentorship opportunities that will be instrumental in my career development.

I look forward to sharing more updates on the exciting developments in my research and the collaborations that have emerged from this conference. I want to extend my gratitude to the International Society for Matrix Biology (ISMB) for making this invaluable experience possible. It wouldn't have been possible without their support and dedication to fostering scientific excellence.

Warm regard,

Samuele Metti, PhD
Department of Molecular Medicine
University of Padova, Italy

Recent publications

Modeling human skeletal development using human pluripotent stem cells.
Lamandé SR, Ng ES, Cameron TL, Kung LHW, Sampurno L, Rowley L, Lilianty J, Patria YN, Stenta T, Hanssen E, Bell KM, Saxena R, Stok KS, Stanley EG, Elefanty AG, Bateman JF. Proc. Natl. Acad. Sci. USA. 2023 May 9;120(19):e2211510120. doi: 10.1073/pnas.2211510120.
Corresponding author: shireen.lamande@mcri.edu.au
Abstract: *Chondrocytes and osteoblasts differentiated from induced pluripotent stem cells (iPSCs) will provide insights into skeletal development and genetic skeletal disorders and will generate cells for regenerative medicine applications. Here, we describe a method that directs iPSC-derived sclerotome to chondroprogenitors in 3D pellet culture then to articular chondrocytes or, alternatively, along the growth plate cartilage pathway to become hypertrophic chondrocytes that can transition to osteoblasts. Osteogenic organoids deposit and mineralize a collagen I extracellular matrix (ECM), mirroring in vivo endochondral bone formation. We have identified gene expression signatures at key developmental stages including chondrocyte maturation, hypertrophy, and transition to osteoblasts and show that this system can be used to model genetic cartilage and bone disorders.*

Secreted ADAMTS-like 2 promotes myoblast differentiation by potentiating WNT signaling
Taye N, Singh M, Baldock C, Hubmacher D. Matrix Biol 2023 120:24-42. PMID: 37187448
Corresponding author: dirk.hubmacher@mssm.edu
Abstract: *Myogenesis is the process that generates multinucleated contractile myofibers from muscle stem cells during skeletal muscle development and regeneration. Myogenesis is governed by myogenic regulatory transcription factors, including MYOD1. Here, we identified the secreted matricellular protein ADAMTS-like 2 (ADAMTSL2) as part of a Wnt-dependent positive feedback loop, which augmented or sustained MYOD1 expression and thus promoted myoblast differentiation. ADAMTSL2 depletion resulted in severe retardation of myoblast differentiation in vitro and its ablation in myogenic precursor cells resulted in aberrant skeletal muscle architecture. Mechanistically, ADAMTSL2 potentiated WNT signaling by binding to WNT ligands and WNT receptors. We identified the WNT-binding ADAMTSL2 peptide, which was sufficient to promote myogenesis in vitro. Since ADAMTSL2 was previously described as a negative regulator of TGFβ signaling in fibroblasts, ADAMTSL2 now emerges as a signaling hub that could integrate WNT, TGFβ and potentially other signaling pathways within the dynamic microenvironment of differentiating myoblasts during skeletal muscle development and regeneration.*

En route towards a personalized medicine approach: Innovative therapeutic modalities for connective tissue disorders
Redhead C, Taye N, Hubmacher D. Matrix Biol 2023 122:46-54. PMID: 37657665
Corresponding author: dirk.hubmacher@mssm.edu
Abstract: *Connective tissue disorders can be caused by pathogenic variants (mutations) in genes encoding extracellular matrix (ECM) proteins. Such disorders typically manifest during development or postnatal growth and result in significant morbidity and mortality. The development of curative treatments for connective tissue disorders is hampered in part by the inability of many mature connective tissues to efficiently regenerate. To be most effective, therapeutic strategies designed to preserve or restore tissue function will likely need to be initiated during phases of significant endogenous connective tissue remodeling and organ sculpting postnatally and directly target the underlying ECM protein mutations. With recent advances in whole exome sequencing, in-vitro and in-vivo disease modeling, and the development of mutation-specific molecular therapeutic modalities, it is now*

feasible to directly correct disease-causing mutations underlying connective tissue disorders and ameliorate their pathogenic consequences. These technological advances may lead to potentially curative personalized medicine approaches for connective tissue disorders that have previously been considered incurable. In this review, we highlight innovative therapeutic modalities including gene replacement, exon skipping, DNA/mRNA editing, and pharmacological approaches that were used to preserve or restore tissue function in the context of connective tissue disorders. Inherent to a successful application of these approaches is the need to deepen the understanding of mechanisms that regulate ECM formation and homeostasis, and to decipher how individual mutations in ECM proteins compromise ECM and connective tissue development and function.

Serine protease 35 regulates the fibroblast matrisome in response to hyperosmotic stress.
Sänger CS, Cernakova M, Wietecha MS, Garau Paganella L, Labouesse C, Dudaryeva OY, Roubaty C, Stumpe M, Mazza E, Tibbitt MW, Dengjel J, Werner S. Sci Adv. 2023 9:eadh9219. doi: 10.1126/sciadv.adh9219.
Corresponding author: sabine.werner@biol.ethz.ch
Abstract: *Hyperosmotic stress occurs in several diseases, but its long-term effects are largely unknown. We used sorbitol-treated human fibroblasts in 3D culture to study the consequences of hyperosmotic stress in the skin. Sorbitol regulated many genes, which help cells cope with the stress condition. The most robustly regulated gene encodes serine protease 35 (PRSS35). Its regulation by hyperosmotic stress was dependent on the kinases p38 and JNK and the transcription factors NFAT5 and ATF2. We identified different collagens and collagen-associated proteins as putative PRSS35 binding partners. This is functionally important because PRSS35 affected the extracellular matrix proteome, which limited cell proliferation. The in vivo relevance of these findings is reflected by the coexpression of PRSS35 and its binding partners in human skin wounds, where hyperosmotic stress occurs as a consequence of excessive water loss. These results identify PRSS35 as a key regulator of the matrisome under hyperosmotic stress conditions.*

A first-in-class pan-lysyl oxidase inhibitor impairs stromal remodeling and enhances gemcitabine response and survival in pancreatic cancer.
Chitty JL, Yam M, Perryman L, Parker AL, Skhinas JN, Setargew YFI, Mok ETY, Tran E, Grant RD, Latham SL, Pereira BA, Ritchie SC, Murphy KJ, Trpceski M, Findlay AD, Melenec P, Filipe EC, Nadalini A, Velayuthar S, Major G, Wyllie K, Papanicolaou M, Ratnaseelan S, Phillips PA, Sharbeen G, Youkhana J, Russo A, Blackwell A, Hastings JF, Lucas MC, Chambers CR, Reed DA, Stoehr J, Vennin C, Pidsley R, Zaratzian A, Da Silva AM, Tayao M, Charlton B, Herrmann D, Nobis M, Clark SJ, Biankin AV, Johns AL, Croucher DR, Nagrial A, Gill AJ, Grimmond SM; Australian Pancreatic Cancer Genome Initiative (APGI); Australian Pancreatic Cancer Matrix Atlas (APMA); Pajic M, Timpson P, Jarolimek W, Cox TR. Nat Cancer 2023 Aug 28. doi: 10.1038/s43018-023-00614-y. Online ahead of print.
Corresponding author: t.cox@garvan.org.au
Abstract: *The lysyl oxidase family represents a promising target in stromal targeting of solid tumors due to the importance of this family in crosslinking and stabilizing fibrillar collagens and its known role in tumor desmoplasia. Using small-molecule drug-design approaches, we generated and validated PXS-5505, a first-in-class highly selective and potent pan-lysyl oxidase inhibitor. We demonstrate in vitro and in vivo that pan-lysyl oxidase inhibition decreases chemotherapy-induced pancreatic tumor desmoplasia and stiffness, reduces cancer cell invasion and metastasis, improves tumor perfusion and enhances the efficacy of chemotherapy in the autochthonous genetically engineered KPC model, while also demonstrating antifibrotic effects in human patient-derived xenograft models of pancreatic cancer. PXS-5505 is orally bioavailable, safe and effective at inhibiting*

lysyl oxidase activity in tissues. Our findings present the rationale for progression of a pan-lysyl oxidase inhibitor aimed at eliciting a reduction in stromal matrix to potentiate chemotherapy in pancreatic ductal adenocarcinoma.

Targeting collagen XVIII improves the efficiency of ErbB inhibitors in breast cancer models
Devarajan R, Izzi V, Peltoketo H, Rask G, Kauppila S, Väisänen MR, Ruotsalainen H, Martínez-Nieto G, Karppinen SM. Väisänen T, Kaur I, Koivunen J, Sasaki T, Winqvist R, Manninen A, Wärnberg F, Sund M, Pihlajaniemi T, Heljasvaara R. J Clin Invest 2023 133:e159181. doi: 10.1172/JCI159181.
Corresponding author: ritva.heljasvaara@oulu.fi
Abstract: *The tumor extracellular matrix (ECM) critically regulates cancer progression and treatment response. Expression of the basement membrane component collagen XVIII (ColXVIII) is induced in solid tumors, but its involvement in tumorigenesis has remained elusive. We show here that ColXVIII was markedly upregulated in human breast cancer (BC) and was closely associated with a poor prognosis in high-grade BCs. We discovered a role for ColXVIII as a modulator of epidermal growth factor receptor tyrosine kinase (ErbB) signaling and show that it forms a complex with ErbB1 and -2 (also known as EGFR and human epidermal growth factor receptor 2 [HER2]) and α6-integrin to promote cancer cell proliferation in a pathway involving its N-terminal portion and the MAPK/ERK1/2 and PI3K/AKT cascades. Studies using Col18a1 mouse models crossed with the mouse mammary tumor virus-polyoma virus middle T antigen (MMTV-PyMT) mammary carcinogenesis model showed that ColXVIII promoted BC growth and metastasis in a tumor cell-autonomous manner. Moreover, the number of mammary cancer stem cells was significantly reduced in the MMTV-PyMT and human cell models upon ColXVIII inhibition. Finally, ablation of ColXVIII substantially improved the efficacy of ErbB-targeting therapies in both preclinical models. In summary, ColXVIII was found to sustain the stemness properties of BC cells and tumor progression and metastasis through ErbB signaling, suggesting that targeting ColXVIII in the tumor milieu may have important therapeutic potential.*

Modeling collagen fibril self-assembly from extracellular medium in embryonic tendon.
Revell CK, Herrera JA, Lawless C, Lu Y, Kadler KE, Chang J, Jensen OE. Biophys J. 2023 Aug 22;122(16):3219-3237. doi: 10.1016/j.bpj.2023.07.001.
Corresponding author: oliver.jensen@manchester.ac.uk
Abstract: *Collagen is a key structural component of multicellular organisms and is arranged in a highly organized manner. In structural tissues such as tendons, collagen forms bundles of parallel fibers between cells, which appear within a 24-h window between embryonic day 13.5 (E13.5) and E14.5 during mouse embryonic development. Current models assume that the organized structure of collagen requires direct cellular control, whereby cells actively lay down collagen fibrils from cell surfaces. However, such models appear incompatible with the time and length scales of fibril formation. We propose a phase-transition model to account for the rapid development of ordered fibrils in embryonic tendon, reducing reliance on active cellular processes. We develop phase-field crystal simulations of collagen fibrillogenesis in domains derived from electron micrographs of inter-cellular spaces in embryonic tendon and compare results qualitatively and quantitatively to observed patterns of fibril formation. To test the prediction of this phase-transition model that free protomeric collagen should exist in the inter-cellular spaces before the formation of observable fibrils, we use laser-capture microdissection, coupled with mass spectrometry, which demonstrates steadily increasing free collagen in inter-cellular spaces up to E13.5, followed by a rapid reduction of free collagen that coincides with the appearance of less-soluble collagen fibrils. The model and measurements together provide evidence for extracellular self-assembly of collagen fibrils in embryonic mouse tendon, supporting an additional mechanism for rapid collagen fibril formation during embryonic development.*

The Role of ADAMTS Proteoglycanases in Thoracic Aortic Disease.
Kemberi M, Salmasi Y, Santamaria S. Int J Mol Sci. 2023 Jul 28;24(15):12135. doi: 10.3390/ijms241512135.
Corresponding author:
Abstract: *Thoracic aortic aneurysm and dissection (TAAD) are complex disease states with high morbidity and mortality that pose significant challenges to early diagnosis. Patients with an aneurysm are asymptomatic and typically present to the emergency department only after the development of a dissection. The extracellular matrix (ECM) plays a crucial role in regulating the aortic structure and function. The histopathologic hallmark termed medial degeneration is characterised by smooth muscle cell (SMC) loss, the degradation of elastic and collagen fibres and proteoglycan (PG) accumulation. Covalently attached to the protein core of PGs are a number of glycosaminoglycan chains, negatively charged molecules that provide flexibility, compressibility, and viscoelasticity to the aorta. PG pooling in the media can produce discontinuities in the aortic wall leading to increased local stress. The accumulation of PGs is likely due to an imbalance between their synthesis by SMCs and decreased proteolysis by A Disintegrin-like and Metalloproteinase with Thrombospondin motifs (ADAMTS) proteoglycanases in the ECM. Mouse models of TAAD indicated that these proteases exert a crucial, albeit complex and not fully elucidated, role in this disease. This has led to a mounting interest in utilising ADAMTS proteoglycanases as biomarkers of TAAD. In this review, we discuss the role of ADAMTSs in thoracic aortic disease and their potential use in facilitating the clinical diagnosis of TAAD and disease progression.*

Challenging level of rigid-body approach involving numerical elements (CHLORAINE) applied to repeated elastin peptides. Depenveiller C, Wong H, Crowet JM, Debelle L, Baud S, Dauchez M, Belloy N. (2023) Journal of Structural Biology 215, 107986. 10.1016/j.jsb.2023.107986
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Abstract: *Elastic proteins and derived biomaterials contain numerous tandemly repeated peptides along their sequences, ranging from a few copies to hundreds. These repetitions are responsible for their biochemical, biological and biomechanical properties. These sequences are considered to be intrinsically disordered, and the variations in their behavior are actually mainly due to their high flexibility and lack of stable secondary structures originating from their unique amino acid sequences. Consequently, the simulation of elastic proteins and large elastomeric biomaterials using classical molecular dynamics is an important challenge. Here, we propose a novel approach that allows the application of the DURABIN protocol to repeated elastin-like peptides (r-ELPs) in a simple way. Four large r-ELPs were studied to evaluate our method, which was developed for simulating extracellular matrix proteins at the mesoscopic scale. After structure clustering applied on molecular dynamic trajectories of constitutive peptides (5-mers and 6-mers), the main conformations were used as starting points to define the corresponding primitives, further used as rigid body fragments in our program. Contributions derived from electrostatic and molecular hydrophobicity potentials were tested to evaluate their influence on the interactions during simple mesoscopic simulations. The CHLORAINE approach, despite the thinner granularity due to the size of the patterns used, was included in the DURABIN protocol and emerges as a promising way to simulate elastic macromolecular systems.*

Monitoring the macrophage response towards biomaterial implants using label-free imaging. Lu CE, Levey RE, Ghersi G, Schueller N, Liebscher S, Layland SL, Schenke-Layland K, Duffy GP, Marzi J. Mater Today Bio. 2023 Jun 13, doi: 10.1016/j.mtbio.2023.100696
Corresponding author: julia.marzi@uni-tuebingen.de
Abstract: *Understanding the immune system's foreign body response (FBR) is essential when developing and validating a biomaterial. Macrophage activation and proliferation are critical events in FBR that can determine the material's biocompatibility and fate in vivo. In this study, two different macro-encapsulation pouches intended for pancreatic islet*

transplantation were implanted into streptozotocin-induced diabetes rat models for 15 days. Post-explantation, the fibrotic capsules were analyzed by standard immunohistochemistry as well as non-invasive Raman microspectroscopy to determine the degree of FBR induced by both materials. The potential of Raman microspectroscopy to discern different processes of FBR was investigated and it was shown that Raman microspectroscopy is capable of targeting ECM components of the fibrotic capsule as well as pro and anti-inflammatory macrophage activation states, in a molecular-sensitive and marker-independent manner. In combination with multivariate analysis, spectral shifts reflecting conformational differences in Col I were identified and allowed to discriminate fibrotic and native interstitial connective tissue fibers. Moreover, spectral signatures retrieved from nuclei demonstrated changes in methylation states of nucleic acids in M1 and M2 phenotypes, relevant as indicator for fibrosis progression. This study could successfully implement Raman microspectroscopy as complementary tool to study in vivo immune-compatibility providing insightful information of FBR of biomaterials and medical devices, post-implantation.

Inverse Regulation of Cartilage Neogenesis at Physiologically Relevant Calcium Conditions by Human Articular Chondrocytes and Mesenchymal Stromal Cells. Hammersen T, Buchert J, Zietzschmann S, Diederichs S, Richter W. Cells. 2023 Jun 18, doi: 10.3390/cells12121659

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Abstract: Elaborate bioreactor cultivation or expensive growth factor supplementation can enhance extracellular matrix production in engineered neocartilage to provide sufficient mechanical resistance. We here investigated whether raising extracellular calcium levels in chondrogenic cultures to physiologically relevant levels would provide a simple and inexpensive alternative to enhance cartilage neogenesis from human articular chondrocytes (AC) or bone marrow-derived mesenchymal stromal cells (BMSC). Interestingly, AC and BMSC-derived chondrocytes showed an opposite response to a calcium increase from 1.8 mM to 8 mM by which glycosaminoglycan (GAG) and collagen type II production were elevated during BMSC chondrogenesis but depressed in AC, leading to two-fold higher GAG/DNA values in BMSC-based neocartilage compared to the AC group. According to control treatments with Mg^{2+} or sucrose, these effects were specific for $CaCl_2$ rather than divalent cations or osmolarity. Importantly, undesired pro-hypertrophic traits were not stimulated by calcium treatment. Specific induction of PTHrP mRNA and protein by 8.0mM calcium only in AC, along with negative effects of recombinant $PTHrP_{1-34}$ on cartilage matrix production, suggested that the PTHrP pathway contributed to the detrimental effects in AC-based neocartilage. Altogether, raising extracellular calcium levels was discovered as a novel, simple and inexpensive stimulator for BMSC-based cartilage neogenesis without the need for special bioreactors, whereas such conditions should be avoided for AC.

The role of glycosaminoglycan modification in Hedgehog regulated tissue morphogenesis. Gude F, Froese J, Steffes G, Grobe K. Biochem Soc Trans. 2023 Jun 28, doi: 10.1042/bst20220719

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Abstract: *Patterns of gene expression, cell growth and cell-type specification during development are often regulated by morphogens. Morphogens are signalling molecules produced by groups of source cells located tens to hundreds of micrometers distant from the responding tissue and are thought to regulate the fate of receiving cells in a direct, concentration-dependent manner. The mechanisms that underlie scalable yet robust morphogen spread to form the activity gradient, however, are not well understood and are currently intensely debated. Here, based on two recent publications, we review two in vivo derived concepts of regulated gradient formation of the morphogen Hedgehog (Hh). In the first concept, Hh disperses on the apical side of developing epithelial surfaces using the same mechanistic adaptations of molecular transport that DNA-binding proteins in the nucleus use. In the second concept, Hh is actively conveyed to target cells via long filopodial extensions, called cytonemes. Both concepts require the expression of a family of sugar-modified proteins in the gradient field called heparan sulphate proteoglycans as a prerequisite for Hh dispersal, yet propose different - direct versus indirect - roles of these essential extracellular modulators.*

Exploring the relationship between epigenetic DNA methylation and cardiac fibrosis through Raman microspectroscopy. Becker L, Montes-Mojarro IA, Layland SL, Nsair A, Fend F, Marzi J, Schenke-Layland K. Am J Physiol Cell Physiol. 2023 Jul 1, doi: 10.1152/ajpcell.00209.2023

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Abstract: *Cardiomyopathies are associated with fibrotic remodeling of the heart, which is characterized by the excessive accumulation of collagen type I (COL I) due to chronic inflammation and suspected epigenetic influences. Despite the severity*

and high mortality rate of cardiac fibrosis, current treatment options are often inadequate, underscoring the importance of gaining a deeper understanding of the disease's underlying molecular and cellular mechanisms. In this study, the extracellular matrix (ECM) and nuclei in fibrotic areas of different cardiomyopathies were molecularly characterized by Raman microspectroscopy and imaging and compared with the control myocardium. Patient samples were obtained from heart tissue affected by ischemia, hypertrophy, and dilated cardiomyopathy and analyzed for fibrosis through conventional histology and marker-independent Raman microspectroscopy (RMS). Prominent differences between control myocardium and cardiomyopathies were revealed by spectral deconvolution of COL I Raman spectra. Statistically significant differences were identified in the amide I region of spectral subpeak at 1,608 cm^{-1}, which is a representative endogenous marker for alterations in the structural conformation of COL I fibers. Moreover, epigenetic 5mC DNA modification was identified within cell nuclei by multivariate analysis. A statistically significant increase in signal intensities of spectral features indicative of DNA methylation was detected in cardiomyopathies in accordance with immunofluorescence 5mC staining. Overall, RMS is a versatile technology in the discrimination of cardiomyopathies based on molecular evaluation of COL I and nuclei while providing insights into the pathogenesis of the diseases.

TAPT1-at the crossroads of extracellular matrix and signaling in Osteogenesis imperfecta. Etich J, Semler O, Stevenson NL, Stephan A, Besio R, Garibaldi N, Reintjes N, Dafinger C, Liebau MC, Baumann U, Mörgelin M, Forlino A, Stephens DJ, Netzer C, Zaucke F, Rehberg M. EMBO Mol Med. 2023 Jul 10, doi: 10.15252/emmm.202317528
Corresponding author: mirko.rehberg@uk-koeln.de
Abstract: *Osteogenesis imperfecta (OI) is a hereditary skeletal disorder primarily affecting collagen type I structure and function, causing bone fragility and occasionally versatile extraskeletal symptoms. This study expands the spectrum of OI-causing TAPT1 mutations and links extracellular matrix changes to signaling regulation.*

High fat diet has a protective sex-dependent effect on aortic aneurysm severity in a Marfan syndrome mouse model. Lau C, Muthu ML, Siddiqui IF, Li L, Reinhardt DP. Can J Cardiol. 2023 Jul 21 doi: 10.1016/j.cjca.2023.07.020
Corresponding author: dieter.reinhardt@mcgill.ca
Abstract: *Background: Marfan syndrome (MFS) is a genetic disorder caused by mutations in fibrillin-1 and is characterized by thoracic aortic aneurysms and other complications. Previous studies revealed sexual dimorphisms in formation of aortic aneurysm in patients with MFS. The current study aimed to investigate the combined role of a high-fat diet (HFD) and biological sex in aortic disease using the mgR/mgR MFS mouse model.*
Methods: Male and female mgR/mgR mice, as well as wild-type (WT) littermate mice, were fed a control diet (CD [10% fat]) or HFD (60% fat) from 4 to 12 weeks of age. Key aortic disease parameters analyzed included the diameter of the aortic wall; elastic fibre fragmentation; proteoglycan content; mRNA levels of Mmp12, Col1a1, Col3a1, and Fbn1; and fibrillin-1 deposition in the aortic wall.
Results:HFD-fed female mgR/mgR mice had significantly reduced aortic diameters (35%), elastic fibre fragmentation (56%), pathologically enhanced proteoglycans (45%), and expression of Mmp12 (64%), Col1a1 (41%), and Col3a1 (43%) compared with male mgR/mgR mice on HFD. Fibrillin-1 deposition and Fbn1 mRNA levels were unaffected. The data reveal a protective effect of HFD in female mice. In contrast, CD did not exert any protective effects.
Conclusions:This study demonstrates a specific sexual dimorphism in MFS mice, with HFD exerting an explicit protective effect on severity of aortic disease in female mice. These preclinical data may be useful for developing nutritional recommendations for individuals with MFS in the longer term.

Secretomics reveals gelatinase substrates at the blood-brain barrier that are implicated in astroglial barrier function. Burmeister M, Fraunenstein A, Kahms M, Arends L, Gerwien H, Deshpande T, Kuhlmann T, Gross CC, Naik VN, Wiendl H, Klingauf J, Meissner F, Sorokin L. Sci Adv. 2023 Jul 21, doi: 10.1126/sciadv.adg0686
Corresponding author: sorokin@uni-muenster.de
Abstract: *The gelatinases, matrix metalloproteinase 2 (MMP-2) and MMP-9, are key for leukocyte penetration of the brain parenchymal border in neuroinflammation and the functional integrity of this barrier; however, it is unclear which MMP substrates are involved. Using a tailored, sensitive, label-free mass spectrometry–based secretome approach, not previously applied to nonimmune cells, we identified 119 MMP-9 and 21 MMP-2 potential substrates at the cell surface of primary*

astrocytes, including known substrates (β-dystroglycan) and a broad spectrum of previously unknown MMP-dependent events involved in cell-cell and cell-matrix interactions. Using neuroinflammation as a model of assessing compromised astroglial barrier function, a selection of the potential MMP substrates were confirmed in vivo and verified in human samples, including vascular cell adhesion molecule–1 and neuronal cell adhesion molecule. We provide a unique resource of potential MMP-2/MMP-9 substrates specific for the astroglia barrier. Our data support a role for the gelatinases in the formation and maintenance of this barrier but also in astrocyte-neuron interactions.

FADD- and RIPK3-Mediated Cell Death Ensures Clearance of Ly6C^{high} Wound Macrophages from Damaged Tissue. Injarabian L, Willenborg S, Welcker D, Sanin DE, Pasparakis M, Kashkar H, Eming SA. J Invest Dermatol. 2023 Jul 27, doi: 10.1016/j.jid.2023.06.203

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Abstract: *Cells of the monocyte/macrophage lineage are an integral component of the body's innate ability to restore tissue function after injury. In parallel to mounting an inflammatory response, clearance of monocytes/macrophages from the wound site is critical to re-establish tissue functionality and integrity during the course of healing. The role of regulated cell death in macrophage clearance from damaged tissue and its implications for the outcome of the healing response is little understood. In this study, we explored the role of macrophage-specific FADD-mediated cell death on Ripk3$^{-/-}$ background in a mechanical skin injury model in mice. We found that combined inhibition of RIPK3-mediated necroptosis and FADD-caspase-8-mediated apoptosis in macrophages profoundly delayed wound healing. Importantly, RIPK3 deficiency alone did not considerably alter the wound healing process and macrophage population dynamics, arguing that inhibition of FADD-caspase-8-dependent death of macrophages is primarily responsible for delayed wound closure. Notably, TNF blockade reversed the accumulation of Ly6C^{high}macrophages induced by combined deficiency of FADD and RIPK3, indicating a critical dual role of TNF-mediated prosurvival and cell death signaling, particularly in this highly proinflammatory macrophage subset. Our findings reveal a previously uncharacterized cross-talk of inflammatory and cell death signaling in macrophages in regulating repair processes in the skin.*

Endothelial basement membrane laminins - new players in mouse and human myoendothelial junctions and shear stress communication. Luik AL, Hannocks MJ, Loismann S, Kapupara K, Cerina M, van der Stoel M, Tsytsyura Y, Glyvuk N, Nordenvall C, Klingauf J, Huveneers S, Meuth S, Jakobsson L, Sorokin L. Matrix Biol. 2023 Aug, doi: 10.1016/j.matbio.2023.06.001

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Abstract: *Basement membranes (BMs) are critical but frequently ignored components of the vascular system. Using high-resolution confocal imaging of whole-mount-stained mesenteric arteries, we identify integrins, vinculin, focal adhesion kinase (FAK) and several BM proteins including laminins as novel components of myoendothelial junctions (MEJs), anatomical microdomains that are emerging as regulators of cross-talk between endothelium and smooth muscle cells (SMCs). Electron microscopy revealed multiple layers of the endothelial BM that surround endothelial projections into the smooth muscle layer as structural characteristics of MEJs. The shear-responsive calcium channel TRPV4 is broadly distributed in endothelial cells and occurs in a proportion of MEJs where it localizes to the tips of the endothelial projections that are in contact with the underlying SMCs. In mice lacking the major endothelial laminin isoform, laminin 411 (Lama4−/−), which we have previously shown over-dilate in response to shear and exhibit a compensatory laminin 511 upregulation, localization of TRPV4 at the endothelial-SMC interface in MEJs was increased. Endothelial laminins do not affect TRPV4 expression, rather in vitro electrophysiology studies using human umbilical cord arterial endothelial cells revealed enhanced TRPV4 signalling upon culturing on an RGD-motif containing domain of laminin 511. Hence, integrin-mediated interactions with laminin 511 in MEJ structures unique to resistance arteries modulate TRPV4 localization at the endothelial-smooth muscle interface in MEJs and signalling over this shear-response molecule.*

Mechanotransduction through hemidesmosomes during aging and longevity. Ewald CY, Nyström A. J Cell Sci. 2023 Aug 1**,** doi: 10.1242/jcs.260987

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Abstract: *Hemidesmosomes are structural protein complexes localized at the interface of tissues with high mechanical demand and shear forces. Beyond tissue anchoring, hemidesmosomes have emerged as force-modulating structures important for translating mechanical cues into biochemical and transcriptional adaptation (i.e. mechanotransduction) across*

tissues. Here, we discuss the recent insights into the roles of hemidesmosomes in age-related tissue regeneration and aging in C. elegans, mice and humans. We highlight the emerging concept of preserved dynamic mechanoregulation of hemidesmosomes in tissue maintenance and healthy aging.

Autonomic nervous regulation of cellular processes during subchondral bone remodeling in osteoarthritis. Rösch G, Zaucke F, Jenei-Lanzl Z. Am J Physiol Cell Physiol. 2023 Aug 1 doi:10.1152/ajpcell.00039.2023
Corresponding author: zsuzsa.jenei-lanzl@kgu.de
Abstract: *Osteoarthritis (OA) is the most prevalent degenerative joint disease. Besides loss of articular cartilage and synovial inflammation, OA progression is characterized by pathological changes in the subchondral bone. In early OA, subchondral bone remodeling typically shifts to an increased bone resorption. However, as the disease progresses an increased bone formation takes place, leading to higher bone density with subsequent bone sclerosis. These changes can be influenced by different local or systemic factors. Recent evidence suggests that the autonomic nervous system (ANS) plays a role in regulating subchondral bone remodeling in OA. In this review, we 1) introduce bone structure and cellular mechanisms of bone remodeling in general, 2) explain the subchondral bone changes during OA pathogenesis, 3) then describe the contribution of the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS), the two major autonomic branches, to physiological subchondral bone remodeling, 4) followed by the influence of the SNS and PNS on subchondral bone remodeling in OA, and 5) finally, discuss the potential of therapeutic approaches targeting different components of the ANS.*

Sox-2 positive cells identified in lymph nodes from endometriosis patients may play a role in the disease pathogenesis. Velho RV, Danielyan I, Mechsner S, Götte M. Eur J Obstet Gynecol Reprod Biol. 2023 Sep, doi: 10.1016/j.ejogrb.2023.07.017
Corresponding author: mgotte@uni-muenster.de
Abstract: *Objective: This study aimed to characterize Sox-2 in sentinel lymph nodes and randomly obtained lymph nodes from endometriosis (EM) patients for the first time.*
Study Design: This prospective study analyzed tissue samples from surgical specimens collected from May until December 2007 in the Endometriosis Center Charité, Berlin. Lymph node samples from 38 women aged between 22 and 49 years who underwent laparoscopy due to symptomatic EM were analyzed. The material was obtained either randomly or, in the case of deep infiltrating endometriosis, detected using 4 cc Patent Blue®, labeled intraoperatively, which made the sentinel lymph nodes available for histological examination. Together with hematoxylin and eosin staining, the sections were evaluated by immunohistochemistry with antibodies against estrogen and progesterone receptors and Sox-2. Using double-immunofluorescence microscopy, the colocalization of Sox-2 and estrogen receptors were evaluated.
Results: Sox-2-positive cells were identified in the lymph nodes' cortical and medullary zones, with a higher expression in the medullary layer. Occasionally, Sox-2 positive stained cell groups, called cell nests, could also be detected. The number of Sox-2 positive cells in the sentinel lymph nodes was almost three times higher than in the random lymph nodes (p = 0.031). A significant five-fold increase (p = 0.0013) in Sox-2 expression was seen in the estrogen and progesterone receptor (ER/PR) positive patient group compared to the progesterone receptor positive group or hormone receptor negative patients. Identical hormone-related Sox-2 expression was also detected separately for the sentinel lymph node group (p = 0.0174). Sox-2 showed pronounced colocalisation with estrogen receptors.
Conclusion: The lymphatic involvement in EM is evidence of a systemic disease manifestation and provides evidence of an immune system failure. In recent years, many theories have been studied, but there is no single theory that could explain all aspects of EM. The future concept of EM is likely to incorporate the elements from all the pathogenetic theories already described. Through this study, stem cells and lymphatic metastasis theories were incorporated.

Meeting announcements

October 22-25, 2023 ASMB Biennial Meeting

Tissue, Matrix, and Pathobiology is an exciting new multidisciplinary and collaborative joint meeting of the American Society for Matrix Biology (ASMB), The Histochemical Society (HCS), and the American Society for Investigative Pathology (ASIP). The Program Committee has put together a fantastic four-day lineup of speakers, career sessions, workshops, and events - and can't wait to welcome you in Salt Lake City!

Who Should Attend: students, trainees, investigators, and educators who are working on cutting-edge basic, clinical, and translational research related to biology of cells and tissues, as well as molecular and cellular alterations that drive disease processes.

Scientific Programming

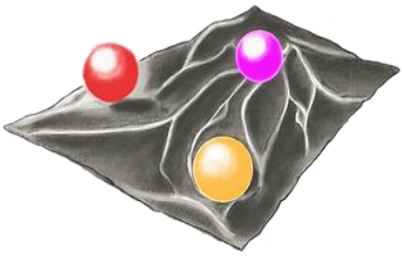
The scientific program includes award lectures, plenary sessions, symposia, and workshops featuring speakers who are leaders in their respective fields of study. Thematic highlights include:

- Extracellular Matrix Biology
- Liver Pathobiology
- Cardiovascular Diseases
- ECM Receptors
- Pathobiology of Fibrosis
- Cancer Pathobiology
- Matrix Disorders
- Neuropathology
- Organoid Models of Disease
- Mechanobiology
- Inflammatory and Infectious diseases
- Cell-Matrix Interactions

The meeting will also feature education and career development sessions that are focused on the needs of the current generation of trainees and young investigators. A generous scholar award program will provide travel grants for trainees and junior faculty who are members of the participating Societies. View the full program. https://asip2023.asip.org/program/joint-scientific-programs/

Danish Society for Matrix Biology Annual Meeting 30th and 31st October 2023

Nielsine Nielsens Auditorium, University of Copenhagen, Denmark



In conjunction with the PhD Course "Matrix Biology: Physiology and Function of the Extracellular Matrix", 1st-3rd November 2023

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* Denotes speaker in competition for best oral presentation awards.
Monday 30th October 2023

13.00-13.05 Welcome address by the DSMB Chairwoman

Session 1. Plenary session and AGM
Session chair: Christine Chuang
13.05-13.55 Anthony Day (Wellcome Centre for Cell-Matrix Research, Manchester, UK) "TSG-6: a Multifunctional Tissue-Protective Protein with Therapeutic Potential" (40 min + 10 min discussion)
13.55-14.20 DSMB Annual General Meeting (25 min)

COFFEE BREAK 14.20-14.45 Refreshments and posters

Session 2. Matrix in Cancer and Fibrosis
Session chair: Daniel Madsen
14.45-15.15 Neel Nissen (Nordic Bioscience, DK)
"Using the Collagen Landscape in Cancer as Non-Invasive Biomarkers" (20+10 min)
15.15-15.30 Kevin Baker* (Centre for Cancer Immune Therapy, Herlev)
"Immunomodulation by Cell-Derived Matrix on Macrophages" (10+5 min)
15.30-15.45 Dimitra Manou* (Lund University, Sweden)
"Post-Irradiation Extracellular Matrix Landscape in Glioblastoma" (10 + 5 min)
15.45-16.00 Emily Martin* (Nordic Bioscience)
"A Biomarker of Collagen Type-I Degradation is Associated with Cardiac Fibrosis and Inflammation in Patients with Liver Cirrhosis" (10 + 5 min)

POSTER SESSION 16.00 - 17.30 Poster session with drinks

CONFERENCE DINNER (OPTIONAL, SELF-PAID) From 18.30 Tuesday 31 October 2023 (685 DKK per person)

Session 3. Matrix Synthesis and Remodeling
Session chair: Rebecca Miller
9.00-9.30 Joan Chang (Wellcome Centre for Cell-Matrix Biology, UK)
"(Circadian) Regulation of Collagen-I Homeostasis? New Paradigms and Considerations" (20 + 10 min)
9.30-9.45 Jerry Turnbull (Keele University, UK and University of Copenhagen, DK)
"Dynamic Regulation of Stem Cells with Exogenous Heparan Sulfate Mimetics" (10 + 5 min)

9.45-10.15 Seyda Ünsal* (University of Southern Denmark)
"Role of Vascular MFAP4 in Age-Induced Extracellular Matrix Remodeling and Angiotensin II-Induced Hypertension" (10 + 5 min)
10.15-10.30 Rudi Hansen* (Department of Physical and Occupational Therapy and Institute of Sports Medicine Copenhagen)
"Do Patients with Patellofemoral Pain have Increased Subchondral Metabolic Activity?" (10 + 5 min)

COFFEE BREAK 10.30-11.00 Refreshments

Session 4. Early Career Researchers Open Session
Chair: Monika Bayer
11.00-11.30 Michal Dudek (University of Manchester, UK)
"The Circadian Clock as a Regulator of Cartilage Homeostasis" (20 + 10 min)
11.30-11.45 Grith Højfeldt* (Institute of Sports Medicine Copenhagen)
"Achilles Tendon Tissue Turnover Before and Immediately After an Acute Rupture" (10 + 5 min)
11.45-12.15 Luke Gamon (Department of Biomedical Sciences, University of Copenhagen)
"Spatial Proteomics of the Human Atherosclerotic Microenvironment" (10 + 5 min)
12.15-12.30 Gloria Kyrila* (Department of Cellular and Molecular Medicine, University of Copenhagen)
"Title TBC" (10 + 5 min)
12.30-12.45 Awards and closing remarks from the chairwoman (15 min)

LUNCH TO GO 12.45 Packed lunches to go

Details on registration PhD Course: This year's DSMB meeting is taking place in conjunction with the Matrix Biology PhD course at the University of Copenhagen taking place 1-3 November 2023. To register for the course, please visit this site. N.B. The registration limit for the PhD course has been reached, and new sign-ups will now be placed on a waiting list.

Registration fee: 100 DKK (and includes DSMB membership for 2024).

Deadlines: Deadline for registration is 20/10/23. Deadline for optional conference dinner registration is 13/10/23. Deadline for abstracts for selected oral presentations has now passed. Abstracts submitted after 28/09/23 may only be permissible for poster presentations, at the discretion of the DSMB.

To register: First, pay either by Mobile Pay to 46151 (Danish Society for Matrix Biology) or transfer the amount to DSMB bank account: Reg: 1551 Account: 1227130 (Danske Bank). (E.g., For registration + conference dinner will be 785 DKK.) Please indicate "DSMB and the name (+dinner)" on the payment. Take a screenshot of your payment and send it to dsmb.dk@gmail.com and fill out the registration form. If you want to pay by international transfer, please contact Chloé Yeung at dsmb.dk@gmail.com.

Abstract submission: Please upload a word document with the title, author list, affiliations with the sections Introduction, Hypothesis, Methods, Results and Conclusion (max. 250 words) to this form . Oral presenters will be notified by 29/09/23. Abstracts submitted after 28/09/23 may only be permissible for poster presentations, which will be at the discretion of the DSMB.

Presentation prizes: There will be prizes for the two best oral, sponsored by Tebubio, and two best poster presentations. Only early career researchers (students, PhDs and postdocs) are eligible. Presenters marked with an asterisk are in the best oral presentation competition.

Interested in joining the board?

The DSMB are looking for matrix enthusiasts from Denmark, Sweden and Norway to join the board to run the society and serve our members better. If you're interested, please drop us an email at dsmb.dk@gmail.com!

Upcoming MBSANZ Conference 6-7th November 2023 in Brisbane (Australia)

MBSANZ
MATRIX BIOLOGY SOCIETY OF AUSTRALIA AND NEW ZEALAND

https://www.mbsanz.org/events

Save the Date for the next Basement Membrane Workshop!
April 11 - 13, 2024 University of Manchester, UK

Workshop Organizing Committee

Rachel Lennon	*University of Manchester (UK)*
Jeff Miner	*Washington University in St. Louis (USA)*
Ambra Pozzi	*Vanderbilt University Medical Center (USA)*
Sylvie Ricard-Blum	*University Claude Bernard Lyon (France)*
David Sherwood	*Duke University (USA)*
Roy Zent	*Vanderbilt University Medical Center (USA)*

More information coming soon. Registration will open in early 2024.

Contact Info: American Society for Matrix Biology, 1801 Rockville Pike, Suite 350 Rockville, MD 20852, (USA) info@asmb.net

ECM Pharmacology Congress
17-19 June 2024, Copenhagen (Denmark)

https://ecm-congress.org/

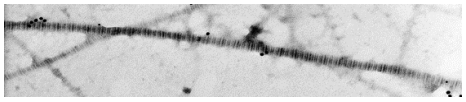
The program will include internationally renowned ECM researchers, practitioners, and industry experts from different disease areas: liver, lung, kidney, cardiovascular, metabolic, cancer (tumor fibrosis), skin, and immunology, in which the central common denominator is changes to the ECM.

the extracellular matrix pharmacology congress

Organizing Committee Chair: Prof. Morten Karsdal

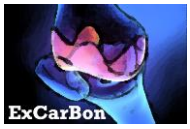
Key dates:
Registration and abstract submission opens: November 2023
Abstract submission deadline: 4 March 2024
Abstract notification: 2 April 2024
Early registration deadline: 15 April 2024
Late-breaking abstract submission deadline: 13 May 2024

Save the date 2024
Annual Meeting of the German Society for Matrix Biology, Regensburg, Bavaria (October 11-13, 2024)



Deutsche Gesellschaft für Matrix Biologie

Sponsored by DFG FOR 2407 and



ExCarBon

Student pre-meeting
Morning until noon at the **11.09.2024**

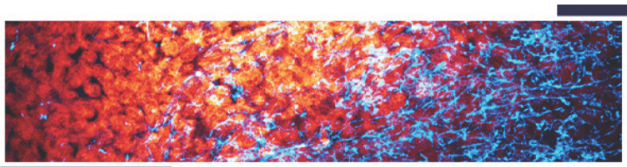
Main meeting
Early afternoon **11.09.24** (Wednesday) until early afternoon **13.09.24** (Friday)

Congress Chairs
Susanne Grässel (Dept. Orthopedic Surgery, University Regensburg)
Attila Aszodi (Dept. Trauma Surgery, LMU Munich)

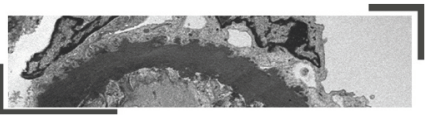
Scientific Committee

Attila Aszodi	Munich
Bent Brachvogel	Cologne
Solvig Diederichs	Heidelberg
Susanne Grässel	Regensburg
Frank Zaucke	Frankfurt

MATRIX BIOLOGY EUROPE 2024



YOUTH@MBE
SATELLITE MEETING



The **Matrix Biology Europe (MBE)** conference is the main European scientific event for the extracellular matrix research community that takes place every other year in a different European city. After Florence (Italy) in 2022, it is our great pleasure to organize the 2024 edition in the beautiful and great city of **Lyon**, France, at the Ecole Normale Supérieure de Lyon, on **September 24-27th.**

MBE2024 intends to be the international scene of major findings, new concepts related to the field of extracellular matrix research and to provide a framework for future research. **MBE2024** will bring together scientists from academia, medicine, and industry to discuss all aspects of Extracellular Matrix Research, from genes to organisms.

MBE2024 aims at providing a collegial atmosphere that fosters exchanges of new ideas between senior investigators, upcoming young scientists and students. In addition to the traditional Ruppert Timpl award and Dick Heinegärd Young Investigator award, **MBE2024** will host a novel half-day satellite, **Youth@MBE2024**, organized by and for graduate students and postdoctoral fellows. All students and post-docs are strongly encouraged to attend the meeting which will highly participate in the creation of their network.

We thus envision this meeting as a unique forum for our community and a unique opportunity to welcome newcomers in the field and to present and exchange new data and cutting-edge ideas about Matrix Biology research. **Program, Registration, Important Deadlines, Prizes, Venues: all info at Matrix Biology Europe (MBE).**

Why Lyon? Conveniently located at the crossroads of Europe, the central location of the charming and beautiful city of Lyon offers easy access to many key European cities. Ten per cent of the city and 162 buildings are listed as UNESCO World heritage including the Presqu'île, Vieux-Lyon, Fourvière Hill and the slopes of the Croix-Rousse. Lyon is also known as the French Capital of gastronomy, home to Michelin star Chef Paul Bocuse and numerous internationally renowned Chefs. But Lyon is also a vibrant and dynamic labor market and hosts major cultural events all year round.

So come and join us in Lyon for this unique scientific (and gastronomic) adventure!

Florence Ruggiero
Conference Chair

https://mbe2024.sciencesconf.org/

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. Junior members are eligible for discounted registration fees at matrix biology 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), who is starting on their independent career, 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, for recent job postings and newsletters.

Composition of ISMB council subcommittees

Meetings and travel grants
Ruud Bank (The Netherlands, chair)
Alexandra Naba, Suneel Apte (USA)
Tom Cox, Hiromi Yanigasawa (Pan-Pacific)

Outreach committee
Valerio Izzi (Finland)
Zoi Piperigkou (Greece)
Konstantina Kyriakopoulou (Germany)
Chloé Yeung (Denmark)

Follow the ISMB on Twitter
ISMB@IntSocMatBio https://twitter.com/intsocmatbio

Communication group coordinated by Valerio Izzi (Finland, Valerio.Izzi@oulu.fi)
Raphael Reuten (Germany, raphael.reuten@pharmakol.uni-freiburg.de)
The ISMB correspondents of National Societies for Matrix Biology for Twitter

Twitter ISMB correspondents of National Societies for Matrix Biology

The ISMB together with National Societies for Matrix Biology continues to strengthen the visibility of Matrix Biology Research on social media. **Please include @IntSocMatBio on your tweets or contact the representatives of the Societies** (see below). We would be happy to share your announcements with the #MatrixBiology world!

The International Society for Matrix Biology (ISMB) Twitter @IntSocMatBio
Valerio Izzi, University of Oulu (Finland) valerio.Izzi@oulu.fi

The American Society for Matrix Biology (ASMB) Twitter @amsocmatbio
Alexandra Naba, University of Illinois, Chicago (USA) anaba@uic.edu

The British Society for Matrix Biology (BSMB) Twitter @BSMB1
Michal Dudek, University of Manchester (UK) michal.dudek@manchester.ac.uk

The Danish Society for Matrix Biology (DSMB) Twitter @DSMB_dk
Christine Chuang, University of Copenhagen (Denmark) cchuang@sund.ku.dk

The Finnish Connective Tissue Society (FSMB)
Valerio Izzi, University of Oulu (Finland) Valerio.izzi@oulu.fi
Piia Takabe, University of Eastern Finland, Kuopio (Finland) piia.takabe@uef.fi

The French Society for Matrix Biology (SFBMec) Twitter @SFBMEc
Patricia Albanese, University of Créteil (France) albanese@u-pec.fr

The German Society for Matrix Biology (DGMB) Twitter @Matrixbiologie
Julia Marzi, Eberhard Karls University Tübingen (Germany) julia.marzi@uni-tuebingen.de

Matrix Biology Ireland (MBI) Twitter @MBIreland
Alexandre Trotier, National University of Ireland, Galway (Ireland) A.TROTIER1@nuigalway.ie

Matrix Biology Society of Australia and New Zealand (MBSANZ) Twitter @MatrixBioANZ
Luise Kung, Murdoch Childrens Research Institute, Melbourne (Australia) louise.kung@mcri.edu.au

The Swiss Society for Matrix Biology (SSMB) Twitter @SwissMatrixBio
Collin Ewald, ETH Zurich (Switzerland) collin-ewald@ethz.ch