The Matrix Letter

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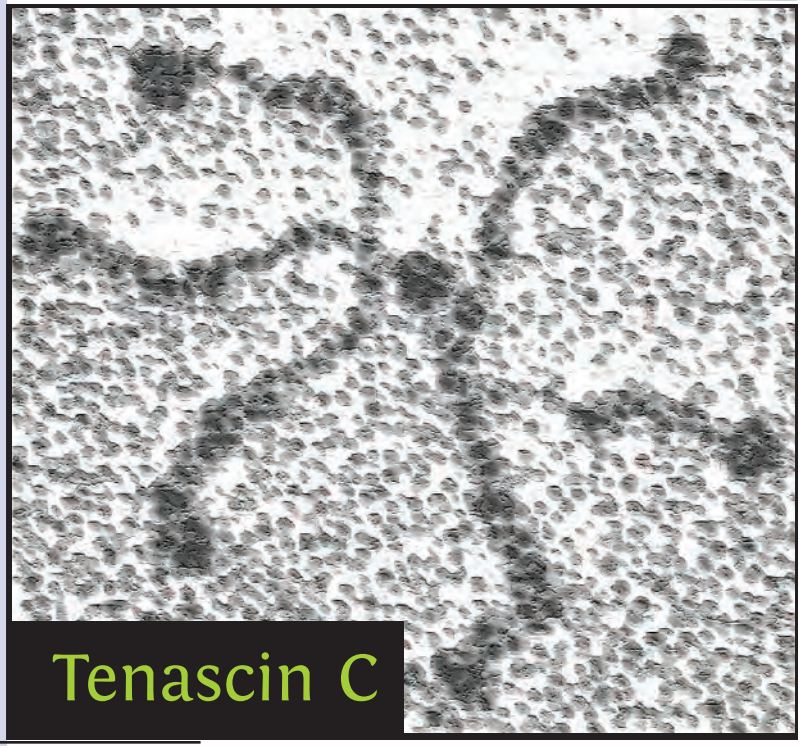


Tenascin C

Contents

Dear ASMB Colleagues,

On behalf of the Planning Committee and ASMB Executive Committee, I would like to invite you to attend the 2016 ASMB Biennial Meeting to be held at the Hilton St. Petersburg Bayfront in **St. Petersburg, Florida,** on **November 13-16, 2016.**

The theme chosen for the the 2016 meeting is:
"**The ECM Microenvironment: A Regulatory force in Aging and Disease.**"

The Programming Committee has crafted a program that will highlight the importance of ECM in the biology of disease and therapeutic applications. Judith Campisi from the Buck Institute for Research on Aging will be our keynote speaker. Plenary topics include sessions on Linking Metabolic Disease with the ECM Microenvironment, Aging and Fibrosis and ECM dysfunction, ECM in Regenerative Medicine and the Stem Cell Niche, Novel Mechanisms of ECM Regulation, and Therapeutics to regulate ECM in Diseases. In addition, we will honor our Junior, Senior, and Iozzo award winners in a dedicated session. We will hold 15 concurrent sessions for oral and poster presentations, and importantly, we have sufficient space to permit posters to be displayed throughout the entire meeting.

In keeping with ASMB tradition, Sunday afternoon has been reserved for sessions on topics organized by Special Interest Groups (SIGs). Trainees are particularly encouraged to contact me regarding organizing a SIG, and there is a strong movement towards 'Trainee governed' SIG's in the future.

St. Petersburg, Florida is a vibrant small city on Florida's Gulf Coast and Tampa Bay. The Hilton is located near the waterfront marina and restaurants and shopping are conveniently located within walking distance. The hotel also offers a free shuttle service to local venues. Temperatures in November are typically in the mid to upper 70's for highs and low 60's for lows. The hotel is about 20-30 minutes from the Tampa International Airport (TPA) and ASMB will be able to offer discounted shuttle rates.

We are excited to announce that our Tuesday evening banquet will be held at the **Salvador Dali Museum** (pictured above), located a short walk from the Hilton, overlooking Tampa Bay. The Dali Museum architecture reflects the unique perspectives of its artist. Docents will be available to assist us in interpreting Dali's unique vision. As scientists, our challenge is to see things differently so that we can make new connections in our attempts to understand the cellular and molecular world: hopefully, you will find some inspiration here.

There is also a wishing tree at the Museum, which we can decorate with our wishes for fundable grant scores!

I hope you will "save the date" for the 2016 meeting and encourage your trainees and colleagues to attend. Please contact me or Kendra LaDuca if you know of people or groups/societies who might be interested in attending so that we can publicize the meeting. Ideas for vendor, grant, and foundational support for the meeting are always welcome.

Additional information regarding registration, hotel reservations, and abstract submission will be available in early 2016.

Best wishes,
Joanne

Joanne Murphy-Ullrich
ASMB President Elect

Key Information:

Date: *November 13-16 2016*
Hotel: *Hilton St. Petersburg Bayfront*
Airports: *Tampa International (TPA)*
St. Petersburg (PIE) *(Allegiant, Sun Country)*
(Orlando is an option for those renting a car)

Cover: *The sixfold geomentry of the curved geodesic features of the Dali Museum (top, prior page) where the meeting will be held is reminiscient of Tenascins (see the memoriam to Dr. Chiquet-Ehrismann on page 7).*

ASMB 2016
Meeting Overview

PLENARY TOPICS

- Linking Metabolic Disease with the ECM Microenvironment
- ECM dysfunction in Aging and Fibrosis
- ECM in Regenerative Medicine and the Stem Cell Niche
- Novel Mechanisms of ECM Regulation
- Therapeutics to Regulate ECM in Diseases

CONCURRENT TOPICS

1. ECM Proteomics, Structure, Assembly, and Cross-linking
2. Signaling from the ECM: Cell Matrix Interactions and ECM Growth Factor Regulation
3. Tumor Microenvironment
4. Basement Membranes
5. Proteoglycans and Glycosylation
6. ECM: Immunity, Inflammation, and Infection
7. ECM in Fibrosis: Liver, Lung, Kidney
8. ECM in Cardiovascular Disease
9. Integrins and Novel Receptor Systems
10. Proteases and Their Inhibitors
11. Mechanobiology
12. ECM in Musculoskeletal Diseases
13. ECM in Wound Healing and Skin Diseases
14. ECM in Morphogenesis
15. ECM in Exosomes: Intercellular Communication

New this year – Poster Only Sessions.

Topics Include:

- ECM and Metabolic Disease
- ECM in Tissue Engineering
- ECM in the Stem Cell Niche
- Novel Mechanisms of ECM Protein Regulation
- Matricellular Proteins
- Therapeutics for ECM-related Diseases

Matrix Interactions

ASMB News and Announcements in Brief

ASMB Elections upcoming!

The ASMB elections are currently underway. All members should have received an email linked to their ballot. If you did not receive the link, please contact ASMB at ASMB@faseb.org.

See Page 8 of this issue to focus on the candidates.

Extracellular Matrisomics Tool

Naba et al., published a very useful review in ***Matrix Biology*** *this summer in which a novel 'Matrisome' website was introduced. This could be a very useful tool for ASMB members investigating the 'omics end of their ECM study.*

The website is found at: http://matrisomeproject.mit.edu.

Additional Selected 2015 Papers: The ECM in Cell Death & Differentiation

A few papers from this year you may have missed, but which might impact a number of ASMB disciplines.

Wang & Astrof, Development (125286 epub a.o.p.) ***Fibronectin's roles autonomous to Neural Crest Cells.***

Fiore et al, J Cell Biol 211:173; revealing a ***Conformational coupling between Integrins and Thy1 That Regulates Fyn-dependent Mechanotransduction.***

Pilling et al., Proc Nat Acad Sci USA 112:11929; showing that ***Lumican Stimulates Fibrocyte Differentiation***

Cieply et al. Matrix Biology 48:42-54, demonstrating that ***CD44S-hyaluronan interaction acts as an anti-anoikis mechanism in EMT.***

Ozmadenci et al., Nat Commun 6:7398; reporting that ***Netrin Regulates Stem Cell Pluripotency.***

Arulmoli et al;, Scientific Rep 5:8489, discussing the ***ECM dependence of static stretch in Neural Stem Cell Differentiation***

Upcoming Events

March 6-10, 2016
Gordon Research Conference: Craniofacial Morphogenesis & Tissue Regeneration
Ventura, California USA
www.grc.org/programs.aspx?id=13289

March 30-31, 2016
Location, Location, Location: The Matrix and the Microenvironment
Chester, UK
http://www.bsmb.ac.uk/meetings-index/chester/

June 11-14, 2016
Matrix Biology Europe
Athens, Greece,
http://www.mbe2016.upatras.gr/

November 13-16, 2016
American Society for Matrix Biology
St. Petersburg, Florida USA
http://www.mbe2016.upatras.gr/

December 11-16, 2016
TERMIS Americas
San Diego, California USA
http://www.mbe2016.upatras.gr/

Matrix Biology

The Elsevier journal is run by long time ASMB member and Editor in Chief, Dr. Renato Iozzo. Papers focus on the extracellular matrix or its biological influence on cells, using a variety of different approaches. ASMB members are entitled to discounts on Matrix Biology subscriptions.

The current impact factor is now above 5, more than a 25% rise from historical values. In addition to Regular Research papers, the journal is also home to Brief Reports (similar to a Regular Report, but more focused in scope or topic). Mini Reviews are also published, frequently as a cluster, as well as Full Length Reviews.

http://www.journals.elsevier.com/matrix-biology/

Post Doctoral Positions

For students as well as for PI's who are looking to help their students find their next position:
http://asmb.net/careeropps.php.

An Interview with Michelle Tallquist

The Matrix Letter recently caught up with Dr. Michelle Tallquist, who happens to be the newest counsellor elected to the ASMB Executive Council. Dr. Tallquist's lab had its origins in Texas at UTSW, but she has (relatively) recently relocated her lab.

ML: Michelle, your lab recently moved to Hawaii. What is it like to work in Hawaii and how does it compare with Texas?

MT: With the day to day lab activities there are no major differences. One of my concerns with moving was the time required to receive reagents, but most items arrive in 24-48 hours. One unique feature about Hawaii is the diverse population. Although I don't do much work with human samples now, the possibility for future studies of this nature is more realistic. Overall, the scientific community here is small, but very active and expanding.

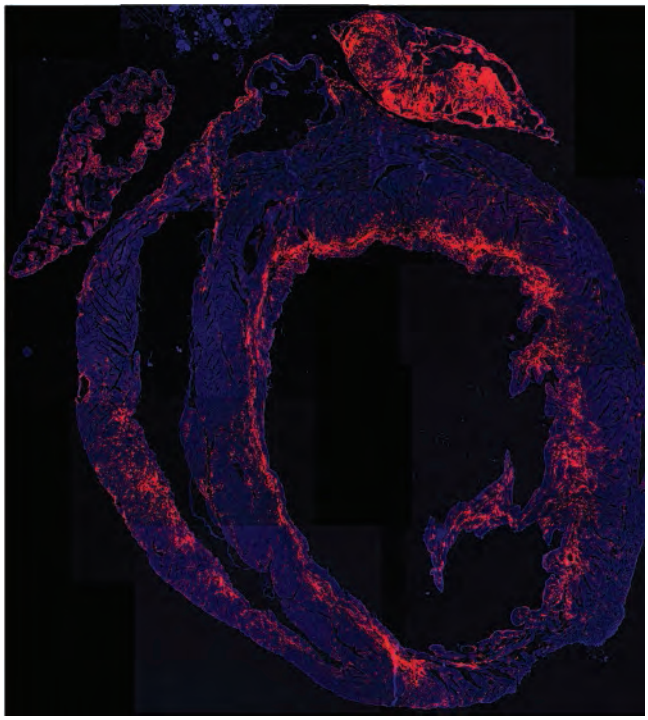
ML: Your lab work focuses on fibroblasts in the heart. How did you come to work in this area? What was it about it captured your interest?

MT: We began working on cardiac fibroblasts while working on a project examining the role of PDGFRβ in the formation of coronary vascular smooth muscle cells. Both the VSMC and cardiac fibroblasts arise from the heart's epicardium through the process of epithelial to mesenchymal transition (EMT). We found that the other PDGF receptor, PDGFRα, was essential for the formation of the cardiac fibroblast lineage. One of the challenges in the study was to precisely define the cardiac fibroblast. At that time, we found that there were few reagents and tools to define and track these cells in vivo. We were forced to develop a number of mouse lines which permit us to trace and manipulate fibroblasts. Now, we can track these cells in most of the organs that exhibit fibrosis, including heart, liver, lung, and kidney.

ML: What keeps you motivated?

MT: My motivation is driven by learning new information and by challenging the 'norm,' which is often based on presumption and not direct scientific evidence. A good example would be our recent work on quantifying the relative number of cardiac fibroblasts to other cell populations resident within the heart. We have found that, contrary to popular belief, endothelial cells actually outnumber cardiac fibroblasts. Based simply on the abundance and the accessibility of endothelial cells, it stands to reason that these cells might be good targets for manipulating signals that result in matrix deposition and fibrosis.

ML: You've had a successful career and you have a family. Do you find it difficult to balance these two? Would you offer any advice for women who are earlier on in their careers, and may be considering different options?



Isoproterenol-induced myocardial injury.
Red shows resident fibroblast accumulation after two weeks of isoproterenol treatment in an adult mouse heart. Figure is a composite of multiple images on a Zeiss Axiovert 200.

(Red is ROSA26tdtomato reporter activity after Tcf21iCre tamoxifen induced recombination which results in a lineage trace of resident cardiac fibroblasts)

MT: That really is a complex question. I do believe that balance is the key to life. The challenge is finding the balance that works for you. Different people will certainly find different balance points.

I have always approached my career by not limiting my options. I have strived to reach goals that I feel might be a little beyond my reach. If you don't try, you never know what you can achieve.

That being said, I also attempt to find a positive side to 'less than ideal' situations. Don't have regrets; learn and just keep moving forward.

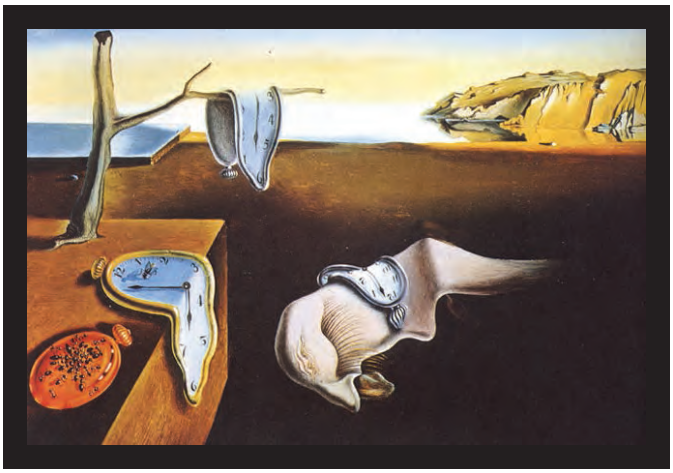
With regards to family, I not only want to be a good role model for my twins, but I also want to take an active part in their lives. Luckily I have a spouse who supports my career goals. I do have had to accept that I can't do everything that I used to before children. I have to say "no" more often. I have also become more efficient. And…let's face it - I survive on less sleep. The beauty of being a researcher and a mother is that you get to have two families. They may frustrate you some times, but more often will fill you with pride and joy.

ML: Now, you are also a member of ASMB – our most newly elected to date, though we have another round of elections looming. I know that being active in ASMB takes your time. Why do you think its important to stay involved?

MT: Matrix biology has become the field that links disparate topics together and provides context. It is a diverse array of researchers who are very supportive and collaborative. By bringing this wide range of investigators together we can have a deeper and more comprehensive understanding of biology that will enhance our ability to treat human disease.

Editorial

The Extracellular Matix and The Persistence of Memory



Given the choice of the Dali Museum for the banquet of the 2016 ASMB meeting (article on the page 2), and Dali's most popularly known work 'The Persistence of Memory, (complete with melting clocks shown above), this seems a perfect time to reflect upon some recent, and not-so-recent, evidence for Extracellular matrix (ECM) in 'memory' functions.

At the recent Society for Neuroscience meeting, some very interesting results were shown that bolster the status of the ECM to one that is 'on par with the discovery that DNA encodes genetic data.' The discovery was that alteration of a structure in the brain called the Perineural net (PNN), which is composed of Aggrecan, Versican, Neurocan, Brevican, Tenascin-R as well as link protein and HA, may be a key structure to holding long term memory. This has been a complex riddle, given that actual proteins within neurons themselves turn over rapidly, with a timeframe of days or hours. In the case of the PNN, it appears that the fine structure is not the critical effector, but rather, it is the superstructure of the PNN and the location of gaps in them that may be important. Alterations to the PNN appear capable of intuitive programs (such as fear) as well as learned behaviors. This would be the initial evidence showing that the ECM regulates memory on the level of the organism.

Depending on how you think about it, however, it is not the first evidence that the ECM fosters 'memory.'

Since its discovery, the ECM has been subject to ongoing interpretation and subsequent revisions of its perceived functions. Certainly, we have long passed through the initial phases where it was recognized as 'tissue' and particularly only a connective tissue, which could lend strength to both acellular and cellular physiologic material. From there, it was a small step for us to see other roles as a scaffold and biologic glue. But not all ECM has significant strength or was noted to provide a good scaffold. Some types of ECM will only poorly support physical attachment. There is a dawning recognition that the ECM actually provided a context for the existence of the cell itself; that the ECM has a signaling function that is dependent upon not only its composition, but also its local complexity, geometry and mechanical properties. The identification of the composition and the recognition that certain ECMs were confined to unique physiological locations provided a further advance in our understanding that the ECM helped program cells. The concept that cells were not (typically) irreversibly programmed into their roles became clear, particularly with the ascension of the field of stem cell biology. Indeed, even stemness may be regulated by interactions with ECM.

We frequently use the word 'homeostasis' in our writing as a foil to pathology, to illustrate the most 'boring' or uninteresting of states, where all cells function in a state that is absent of disease. For the most part, we don't stop to consider precisely what homeostasis represents. We accept it as a cell locked into its natural, neutral state. Probably, maintaining its last genetic input – the status quo – via epigenetic alterations to key regions of our genome. In fact, it may be a more highly active process involving constant crosstalk between DNA, ECM, and in some cases, cytokines.

The past two decades or so have stongly implicated the ECM in the process of what we might call supra_cellular memory. Indeed, the nature of the matrix is such that it is ideally suited for integrating tissue memory . Within each cell, we understand that the ongoing 'memory' of who that cell was, and is, is dictated in large part by epigenetic alterations to its DNA. Yet, one question that arises is whether this is sufficient to facilitate 'ongoing' memory within a cell.

Such a system would seem to be far from perfect. The entropic/stochastic nature of biology cell division provides one with little confidence that the genetics alone would be sufficient. Rather, it seems more likely that additional signals might be required to prevent cellular drift from their assigned duties. Before pursuing this further, we need to agree that we collectively tend to call this 'programming.' But it seems like this the word memory has a much broader context that would include scales beyond individual cells. It might be useful to consider two well established tropes from cell biology.

The first is the suppression of transformation by elements of the basal lamina noted in breast cancer. This has been shown by a growing host of investigators, but was pioneered by Mina Bissell. In this case, ECM secreted by the epithelial cells, and particular laminins, serve to remind that cells of their status. The simple disruption of the interaction with the ECM interferes with this 'reminder' function, and disrupts it has on the cells. 'Reminder' would seem to be the functional word, since tumor cells that metastasize often undergo a transition from mesenchymal to epithelial phenotype upon landing in an epithelial target tissue.

Some years ago, Donald McDonald also provided striking evidence for ECM providing a tissue-scale of memory. His focus was on angiogenesis, and in particular on anti-angiogenic therapies. Notably, his lab showed that tumor revascularization occurred following withdrawal of anti-angiogenic therapy, and that the regrowth of new vascular sprouts followed exactly the track of the the prior existing vessels. In this case, a pre-existing track of ECM that was produced by the intial vasculature was responsible for templating the regrowth. This suggests another method of tissue-architectural level of memory, and provides an example of a tissue specific memory beyond that of the individual cell that secretes the ECM.

If this is the case, it would seem that tissues could 'forget' as well, and that this must require reorganization of the ECM, perhaps by protease or glycosidase activities, and certainly by replacement with a new ECM. In this case, cells would certainly forget their prior roles with time, regardless of original epigentic programming, and new cells would be reminded of new roles. Do Dali's clocks look fibrotic to you?

Ruth Chiquet-Ehrismann:
Tenascin Pioneer

ASMB mourns the passing of Ruth Chiquet-Ehrismann. Ruth made seminal contributions to understanding tenascins and their structure and functions and she is considered the founder of the tenascin field. In addition to her outstanding and careful science, Ruth was also an exemplary mentor and colleague. She was a role model for women in science with families. Her scientific legacy is evident in the many trainees and colleagues she has nurtured to successful, independent careers. Her warmth, smile, laugh, and collegiality will be missed terribly. ASMB extends its condolences to her husband, Matthias Chiquet, and to her children and grandchildren. Three of Ruth's colleagues (Richard Tucker, University of California-Davis; Kim Midwood, University of Oxford; Gertraud Orend, INSERM Strasbourg) have written an extended tribute to Ruth which more fully captures this most remarkable woman and scientist:

We are saddened to convey the passing of Ruth Chiquet-Ehrismann at her home near Basel, Switzerland, on September 4, 2015. She is survived by her husband Matthias Chiquet, her children Daniel, Patrice and Fabian, and three grandchildren.

Ruth earned her Ph.D. from the ETH in Zurich where she studied myoblast interactions with fibronectin with David Turner. After completing her postdoctoral studies at the Johns Hopkins University she joined the Friedrich Miescher Institute in Basel in 1985, where she worked as a group leader until her death.

Ruth worked for all of her career in the field of Matrix Biology. Her 1986 paper in Cell characterized the matrix glycoprotein tenascin-C; she gave this molecule its name and provided the first real, key insights into its biology and associated pathologies. Often described as the "mother of the tenascins", she spent three decades exploring these multifaceted matrix proteins. Her comprehensive analysis of tenascin-C revealed its very particular pattern of expression and the underlying regulatory mechanisms behind this, its role in adhesion modulation, and its ability to bind to matrix molecules and cell surface receptors.

She also pioneered the study of the importance of this molecule in tumor biology. By the early 1990s she had incorporated into her studies the entire tenascin family, and along the way she discovered a new family of neuronal signaling proteins, the teneurins.

Authoring more than 150 papers, Ruth's work was always fundamentally original and highly creative. Publication of her papers was highly anticipated in the field, creating the sense that something exciting was about to be revealed within. Every paper, whilst diverse in scope and methodology, was unfailingly characterized by her trademark scientific rigor that left the reader both with the satisfaction of an important question well answered, and with the distinct feeling that you wished you had thought of this idea or approach yourself. With her tenacious belief that matrix is more than just pretty fibrils.

Ruth was a highly supportive mentor to more than 70 students, postdocs, visiting scientists and technical personnel. Her mentees benefitted from the scientific freedom that she granted, her generosity and her strong career support. Ruth helped several of her mentees to develop their own scientific careers by staying in contact and collaborating.

Ruth played a huge role in establishing widespread awareness of the complexity of matrix biology and the exciting roles of matrix in regulating tissue homeostasis and diseases.

Ruth was also a role model to many of her female mentees who faced the challenges of motherhood while doing science. Ruth was remarkable in many ways, not least in the fact that she not only founded the tenascin field but that she continually revolutionized it. She was respected by all who knew her, or her work, and she was an inspiration to everyone in the field, regard less of their age or position. She will be remembered as much for her pioneering work, as for her generosity as a colleague, her constructive advice, her kindness, her smile and her laugh. Her absence from future matrix meetings will be a great loss. She will be missed enormously.

ML Notes: *We often forget the mixed use of common sense and clever approaches that were standard in the pre-omics era of science. These selected papers from the first three years of tenascin research by Dr. Chiquet-Ehrissmann are well worth reading for their thoroughness and the way they might inspire our current work.*

1. Tenascin: an extracellular matrix protein involved in tissue interactions during fetal development and oncogenesis. Chiquet-Ehrismann et al., Cell 47:131-9. 1986.

2. Tenascin Interferes with fibronectin action. Chiquet Ehrismann et al., Cell 53:383-90, 1988.

3. The distribution of tenascin coincides with pathways of neural crest cell migration. Mackie et al. Development, 102, 237-50 1988.

4. Tenascin: cDNA cloning and induction by TGF-beta. Pearson et al., EMBO J 7:2977-82.

5. Tenascin, an extracellular matrix protein, exerts immunomodulatory activities. Ruegg et al, Proc Natl Acad Sci USA, 86:7437-41, 1989.

6. Two contrary functions of tenascin; dissection of the active sites by recombinant tenascin fragments. Spring et al., Cell 59:325-34,1989.

ASMB Council Election Candidates

Election season is upon us, and all members should have been emailed a link to their ballot. If you haven't yet received one, email us at ASMB@FASEB.org. We will be electing two councillors this year.

Andrew Leask

Dr. Leask was born in Vancouver and obtained his BSc from the University of British Columbia. He then finished his PhD with Elaine Fuchs at the University of Chicago, where he identified the mechanism underlying keratinocyte-specific transcription. After a postdoctoral fellowship at Stanford, Andrew was recruited by George Martin to FibroGen, a San Francisco Bary Area startup focused on fibrosis. There, he studied how CTGF/CCN2 was regulated in normal and fibrotic cells. These studies led to a partnership with Sankyo and helped validate CTGF/CCN2 as a bona fide anti-fibrotic target. Fibrosis, one of largest groups of disease for which there is no universally agreed-upon therapy, is a major cause of loss of organ function and death. Fibrosis, the excessive deposition of scar tissue, is hallmark of connective tissue diseases such as scleroderma and osteoarthritis. Moreover, fibrosis is a major cause of complications resulting from arthroplasty. All of these processes have a significant result on mobility and pain, but can also result in significant morbidity. Dr Leask's objective is to identify the cell types specifically responsible for fibrosis and to develop methods of altering the local microenvironment to block and reverse the fibrotic process. Specifically, we are focusing on how resident mesenchymal cells and mesenchymal precursors show highly reactive adhesive signaling and how this process modifies responses to growth factors and the surrounding extracellular matrix to generate fibrogenic responses and scar tissue. In particular, Dr Leask studies scleroderma and melanoma (a highly metastatic cancer with a strong fibroproliferative component). Dr Leask has over 170 publications and an h factor 0f 46.He is a member of the boards of the Canadian Connective Tissue and International CCN Societies. He was Scientific Editor In Chief and is currently Section Editor of Journal of Cell Communication and Signaling. He is on the editorial board of three other journals.

https://www.schulich.uwo.ca/dentistry/leask/

Douglas Gould

Dr. Gould is a member of the Departments of Ophthalmology and Anatomy and the Institute for Human Genetics in the University of California, San Francisco (UCSF) School of Medicine. Dr. Gould began his training with a BSc in Genetics and PhD in Medical Genetics from the University of Alberta in Edmonton, Canada. He then completed his postdoc training at the Jackson Laboratory in Bar Harbor, Maine. There, Dr. Gould discovered pathogenic mutations in the gene encoding type IV collagen alpha 1. Since 2006 he has led an independent research group that is investigating the pathogenic mechanisms by which defects in COL4A1 and COL4A2 lead to disease. During this time his laboratory has been continuously supported by multiple NIH grants as well as funds from the American Heart Association, Muscular Dystrophy Association and others. Dr. Gould has received a Career Development Award from Research to Prevent Blindness and is currently an elected member of the Genetics section of the Annual Meeting Program Committee for the Association for Research in Vision and Ophthalmology.

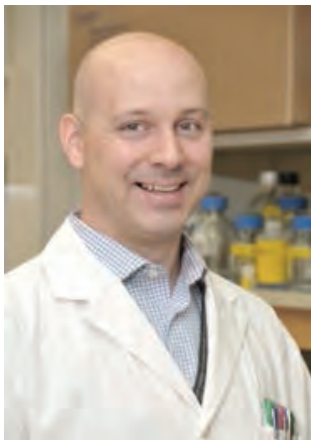
http://vision.ucsf.edu/gould/Gouldlab/Welcome.html

Karen Posey

Dr. Posey has been an Assistant Professor at the University of Texas Medical School Pediatric Research Center since 2006. She studied Botany at Louisiana Tech University and received a Master's degree in Plant Physiology from Texas A & M University. Dr. Posey's PhD studies were in Biochemistry at the University of Houston. Her postdoctoral training was at the Texas A & M University Institute of Biotechnology and her studies focused on protein biochemistry. She began studying cartilage-related disorders in 2003. Dr. Posey is very active in the The Rolanette and Berdon Lawrence Bone Disease Program of Texas, which provides a platform for scholarly exchange and collaboration among investigators studying cartilage, bone and the extracellular matrix. The major focus of her work is understanding the pathology caused by mutations in cartilage oligomeric matrix protein (COMP) that produce pseudoachondroplasia, a severe disproportionate short stature condition. Her current investigations are centered on the development of therapies for cartilage-related disorders, characterizing protein interactions and pathological pathways that contribute to skeletal disease. She has funding from NIH and Bone Disease Program of Texas. Some of her recent research was highlighted in the NIH National Institute of Arthritis and Musculoskeletal Skin Disease spotlight newsletter (see link below).

https://med.uth.edu/pediatrics/faculty/karen-posey-ph-d/
https://gsbs.uth.edu/faculty/faculty-directory/faculty-profiles.htm?id=1347005
http://www.niams.nih.gov/News_and_Events/Spotlight_on_Research/2014/PSACH.asp

Sean Gill



Dr. Gill is an Assistant Professor in the Departments of Medicine and Physiology & Pharmacology at Western University, as well as a Scientist with the Lawson Health Research Institute in the Critical Illness Research Program. He received his PhD in Physiology from Western in 2006 and completed postdoctoral training with Dr. William Parks at the University of Washington in Seattle, Washington. He joined the faculty at the University of Washington in the Division of Pulmonary and Critical Care Medicine as an Acting Instructor in 2010 before returning to Western in 2012. Dr. Gill served as the Chair of the Gordon Research Seminar on Matrix Metalloproteinases in 2013, was on the organizing committee for the 20th Annual Canadian Connective Tissue Conference in 2014, is an honorary editorial board member for the Journal of Metalloproteinases in Medicine, and is on the ASMB 2016 Program Planning Committee. He was also selected as the 2014 ASMB Junior Investigator. Throughout his research career, Dr. Gill has focused on the role of endogenous metalloproteinase activity (through use of mice lacking tissue inhibitor of metalloproteinases [TIMP] 3) in the regulation of extracellular matrix composition in the healthy and injured lung. Research in his laboratory, which is supported by the Canadian Lung Association and the Heart and Stroke Foundation of Canada, is currently focused on the resolution of inflammation and repair following lung injury. Specifically, projects are focused on the role of TIMP3 in regulating three different aspects of recovery following lung injury: 1) initiation and resolution of fibrosis; 2) macrophage polarization and apoptosis; and, 3) microvascular endothelial cell dysfunction.

http://www.schulich.uwo.ca/physpharm/people/bios/faculty/gill_sean.html
https://www.schulich.uwo.ca/deptmedicine/respirology/dr-sean-gill

Thomas Barker

Dr. Barker, Associate Professor and Petit Faculty Fellow, Georgia Institute of Technology, received his Ph.D. in Biomedical Engineering at the University of Alabama at Birmingham. He completed Postdoctoral Fellowships with Dr. Helene Sage at the Hope Heart Institute and UW and Dr. Jeffrey Hubbell at École Polytechnique Fédérale de Lausanne. Highlights from his training include his Ph.D. dissertation discovery that the cell-surface protein Thy-1 is a novel regulator of fibroblast-ECM interaction leading to altered cell adhesion, contractility, and migration. While in Helene Sage's lab he discovered Integrin-Linked Kinase (ILK) as a direct downstream intracellular signal in fibroblast responses to the matricellular protein SPARC. He established, in collaboration with Viola Vogel, the role of SPARC in directing fibroblast contractility-mediated fibronectin assembly in tissue repair. While in the Hubbell group, he demonstrated the utility of controlling domain stability of the integrin-binding domain of fibronectin through rational protein mutation in guiding mesenchymal stem cell integrin specific interactions and downstream lineage specification.

His research program is focused on cell-extracellular matrix (ECM) interactions, including uncovering fundamental mechanisms driving ECM-directed cell differentiation, migration and tissue/matrix remodeling and endogenous fibroblast subpopulations associated with fibrosis and scar. Dr. Barker's lab has focused on rational design of fibrin-based biomaterials through engagement and modification of fibrin's endogenous polymerization mechanism. He has continued his work demonstrating the role of integrin-specific engagement through control of fibronectin conformation in cell behaviors, particularly in fibrosis-associated epithelial-to-mesenchymal transition (EMT) and myofibroblast differentiation, through regulation of cell mechanotransduction and cell-substrate rigidity sensing.

He is published in top cell biology, pathology, and biomaterials journals including Nature Materials, PNAS, Blood, Nature Communications, Journal of Cell Biology, Biomaterials, Journal of Pathology, Journal of Biological Chemistry and others. He serves on the Board of Councillors for the International Fibrinogen Research Society, the International Board for Biomaterials, and the Editorial Board for Matrix Biology.

He is a guest editor for Special Issues in Acta Biomaterialia and Matrix Biology and was the first invited moderator for the in-journal debate forum in Biomaterials. Dr. Barker has received multiple R01s from the NIH as well as funding from DOD, Coulter Foundation, Health Effects Institute, and others. His awards include the 2000 National NASA Space Fellow, 2004 Ruth L. Kirshtein Postdoctoral Fellowship, 2008 Health Effects New Investigator Award, 2012 American Society for Matrix Biology (ASMB) Junior Investigator Award, and the 2015 NIH Director's Transformative Research Award.

Thank you

As mentioned at the beginning of this article, we will be replacing two members of the ASMB executive. These are Dr. Kayla Bayless and myself (Dwayne Stupack). Four years have passed, and there is still a lot to do - it feels more like we just started. For me, it has been a tremendously rewarding experience.

As you have seen, we have outstanding people willing to come onboard to help run our Society. Good luck to all the candidates, and thank you from all of us for your willingness to serve the ASMB!

The Back Page

Postdoctoral Positions

A postdoctoral position is available in the Gould Lab in the Departments of Ophthalmology and Anatomy and Institute for Human Genetics at UCSF School of Medicine.

My group is interested in understanding the diverse roles of extracellular matrix molecules in development and disease. Depending on his/her qualifications, the successful applicant will study **developmental cell biology** and genetic models of **congenital disorders** or **angiogenesis** and **cerebral small vessel disease**. The overarching goal of our research is to understand the biological roles of type IV collagens. The type IV collagens are half a billion years old and are one of the earliest extracellular matrix molecules in all metazoans. Recent evidence suggests that collagen type IV alpha 1 (COL4A1) participates in many cell signaling pathways and developmental processes. As such, COL4A1 mutations cause highly penetrant multisystem disorders including cerebrovascular disease and stroke. We seek to understand novel aspects of biology and to identify key molecular mechanisms contributing to human disease.

The successful applicant is expected to apply for external fellowships, present data at national meetings, prepare manuscripts, mentor junior lab members and participate in a robust training program to prepare the candidate for a career as an independent PI. The applicant must be hard working, passionate about research and be able to work independently and as part of a team. Candidates with a published track record in developmental and molecular biology or in vivo angiogenesis models are preferred. Training in biostatistics or matrix biology are also desirable.

This is a full-time position with the possibility of renewal annually based on performance and availability of funds. Review of applicants will start immediately and continue until the position is filled. Only shortlisted candidates will be notified. The annual stipend is based on NIH levels and will be determined based upon the applicant's qualifications.

Interested individuals should submit a single pdf file with all of the following to TheGouldLab@gmail.com:
1) CV
2) statement of research experience (no longer than 1 page)
3) statement of career goals (no longer than 1 page)
4) contact information for two references
5) please state "I saw your advertisement in the Matrix Letter"

For more information please visit our website:
http://vision.ucsf.edu/gould/Gouldlab/Welcome.html

Doug Gould, PhD.
Departments of Ophthalmology and Anatomy
Institute for Human Genetics
UCSF School of Medicine
10 Koret Way, Room K235
San Francisco, CA, 94143

More opportunities at: *http://asmb.net/careeropps.php*

Contributing Content

The content of ***The Matrix Letter*** includes both ASMB news items and also research-directed content that fosters the mission of the ASMB:

...to promote basic, translational, and clinical research on the extracellular matrix (ECM), cell-ECM interactions, and ECM-based therapies and devices, and to support the growth and professional development of the ECM research community...

From the perspective of this communication, connecting ASMB researchers with each other, based on their research focus or their approaches is the ultimate goal. The Matrix Letter currently publishes the following categories of lab-initiated content;

Matrix Mini-reviews

The Matrix Mini-review feature will be a focused summary the contribution of a particular lab in the context of the current state of knowledge in that field. Usually written by students, postdoctoral fellows or young faculty, the minireview runs about a single written page, with a single scientific illustration and a lab photo, and less than 10 references.

Matrix Essays

The purpose of a Matrix Essay is to promote a new or breaking hypothesis in the field of Matrix biology, with the expressed purpose of garnering supporting evidence and collaborators from the greater ASMB membership. Matrix essays are about one running page and may include a single illustration and up to 10 references.

Letters to the Editor

A letter to the editor should be short and succinct, and will focus on alerting the ASMB membership to recent advances or concerns in our, and related, fields. A letter to the editor is limited to 200 words and three references.

Matrix Images

These are submissions of particularly aesthetic or educational images that you are willing to share with the membership, along with a caption explaining the image.

Reference Format

1) Lewis R, Ravindran S, Wirthlin L, Traeger G, Fernandes RJ, McAlinden A. Disruption of the developmentally-regulated Col2a1 alternative splicing switch in a transgenic knock-in mouse model. Matrix Biol. 2012;31:214-26.

We welcome your contributions.

The Matrix Letter is a communication of the ASMB, edited by Dwayne Stupack with associate editors Carolyn Dancevic, Bryan Crawford and Yoshihiro Ishikawa (Vol14 #2). Thanks also to Suneel Apte and Joanne Murphy-Ullrich.