American Society for Matrix Biology

President's Letter

Jean Schwarzbauer

Dear Fellow Matrix Biologists,

I'm well into my first year as president and focused on the major activity of our society, the biennial meeting. This meeting will be special as our first joint effort with another group, the Society for Glycobiology (SfG). In San Diego, November 11-14, 2012, you will see a program and other activities that blend together two groups with distinct, yet significantly overlapping scientific interests. While this is an experiment, the preliminary data look promising in that the program co-chairs Jeff Davidson and Hudson Freeze, Presidents-elect for ASMB and SfG, respectively, and their Program Committee have already made significant progress on putting together an exciting program.

The process of combining the interests of two groups into one conference got me thinking about the role of the ASMB. After all, with the interdisciplinary nature of science these days, many of us belong to more than one society. What is it that the ASMB offers to matrix biologists that we can't get from another society? Here are some thoughts.

The ASMB meeting is THE place to meet for the community of matrix biologists. Our biennial meeting is the one event that brings together scientists from all areas of matrix biology. Various tissues and diseases, stages of development and aging, bench to biotech or bedside, all are represented in plenary, concurrent and poster sessions. While there are many excellent Gordon conferences, FASEB meetings and the like that focus on specific aspects of ECM biology, only the ASMB meeting covers such a broad range of matrix topics, making it the place to be every other year for anyone who wants to keep up to date on what's new and exciting in the broad field of matrix biology.

Networking works best in person, and our meeting is a great place to do that. Formal talks and less formal poster discussions provide us with new ideas for experiments and projects. Presenting our work helps us to make new connections and establish collaborations. If you are a student, this meeting is a great place to look for a mentor for the next stage of your career. If you are a post-doc, you can make contacts that may help you land an independent position. If you are already independent, no matter what your age or stage, this is a great way to publicize to others what's going on in your lab, at your company, or in your clinic.

The 2012 meeting, in particular, will provide many opportunities for interactions not only with other scientists but also with other societies. It is being jointly organized with the SfG, a society of about the same size as ASMB. The Distinguished Investigator of the International Society for Matrix Biology (ISMB) will speak in a special awards session. Plus several special Sunday afternoon Guest Society sessions are in the planning stages and will be announced later this year.

Membership has its benefits. The most cost-effective way to attend the ASMB/SfG meeting in 2012 is as an ASMB member since membership provides you with a discounted registration fee. As a member, you are eligible to be nominated for the Junior or Senior Investigator award, which honors up-and-coming or already-there matrix biologists. Plus, all member abstract submissions are considered for travel awards to support oral and poster presentations at the meeting.
Another perk of membership is the ASMB Newsletter. Marian Young (NIH) has graciously agreed to become editor-in-chief of the Newsletter and is working with a dedicated committee that includes Ambra Pozzi, Audrey McAlinden, and Bob Mecham. The Newsletter is published five times over two years and is emailed to all

members. In it, you will read highlights of interesting research articles, info on what's happening in other societies, postings for job openings, news about the meeting, and more.

Committee service is a rewarding way to spend your spare time. Help out the ASMB and have input into our goals and mission by volunteering to serve on one of the society's committees (http://www.asmb.net/committees.php). Or put your name in for an elected office. In fact, we will be holding elections for two Council seats in early 2012.

Helping young matrix biologists develop their careers is a relatively new, but very important, activity of the ASMB. Our Professional Development committee chaired by Amy Bradshaw ran a very successful mentoring breakfast at the 2010 meeting, and we already have plans for mentoring sessions at the 2012 meeting. Amy's term on Council ends this year but we will continue what she has started. It will truly benefit young scientists if we are available to offer them career and professional help and advice. Therefore, one of my goals for the coming year is to develop ways to provide mentoring and career advice via the ASMB website so that we can offer our services to all interested young matrix biologists throughout the year.

I hope that by pointing out a few benefits of ASMB membership, I have given the members who are reading this some incentive to recruit new members. For any non-members who happened to get ahold of this Newsletter, maybe I have whetted your appetite sufficiently so that you look into joining.

The ASMB is our community and it has a lot to offer to matrix biologists at all career stages. We should be proud of it, promote it, and help it to thrive. I am always happy to hear from anyone interested in the ASMB so feel free to email me with ideas, comments, and questions.

Thanks and best wishes,

Jean Schwarzbauer
ASMB President

As Jean Schwarzbauer has underscored, your meeting is a key benefit of your ASMB membership. It takes a village to make a successful meeting, and on behalf of your Program Committee I'm pleased to report that the plans for your next annual outing in sunny, warm San Diego next November for ASMB2012 are well underway. The new collaboration with the Society for Glycobiology is certainly in the spirit of experimentation, but your Program Committee and its SfG counterpart, led by Hud Freeze, have virtually completed the assembly of a splendid assortment of matrix and glycobiology plenary speakers. You will find the program not only "fair and balanced", but exceptional in terms of quality and breadth of presentation material. Our 16 concurrent, abstract-driven sessions — covering a wide range of topics — will be moderated by exemplary speakers. We are certain that this combined meeting will give you plenty of opportunities to stay on the forefront of field, to present your own great achievements, to renew and forge connections with your colleagues, and to participate in the only national meeting that brings so many matrix and glycobiology specialties together.

Prior to the formal opening of the meeting on Sunday evening, we expect to have 3-4 additional, half- or full-day satellite symposia, which will be sponsored by groups with related interests. If you know a group that would benefit from a free-standing symposium, please contact me to discuss the opportunity.

Now that many of the nuts and bolts of the meeting organization have been tightened, your Program Committee and Maurizio Pacifici in particular will turn to the task of engaging sponsored support, which is absolutely essential to the success of the program. Meetings such as ours cannot operate on registration fees alone, and we want to be able to extend travel assistance to deserving participants. If you have suggestions for contacts, either provide myself or Jen Holland with the information or take the initiative of contacting a potential sponsor. We have prepared a Prospectus that we'd be delighted to distribute. If you have any ideas or concerns about the meeting, please let us know.

On behalf of your Program Committee,
Jeff Davidson
ASMB Program Chair
Jeff.davidson@vanderbilt.edu

Meeting Organizers	Program Committees
Hudson Freeze (SFG)	Elaine Davis (ASMB)
Robert Haltiwanger (SFG)	Linda Sandell (ASMB)
Jeffrey Davidson (ASMB)	Tom Barker (ASMB)
David Roberts (ASMB)	Ambra Pozzi (ASMB)
Jean Schwarzbauer (ASMB)	Jeff Esko (SFG)
	Hawkeye Pierce (SFG)
	Yu Yamaguchi (SFG)
	Linda Hsieh-Wilson (SFG)

Tell Us What Your Lab is Doing!

From the laboratory of Anne Delany, Ph.D
University of Connecticut Health Center

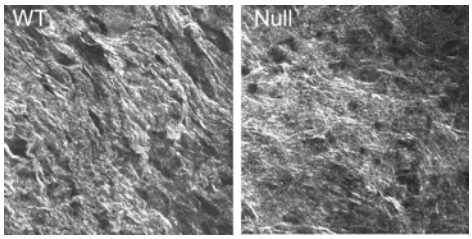
Delany lab (from left to right): Kirsten Saucier, Spencer Smith, Tiziana Franceschetti, Anne Delany, Neha Dole, Cathy Kessler.

Our lab is interested in understanding the function and regulation of osteonectin/SPARC in the skeleton. In the absence of osteonectin, mice develop profound, progressive, low turnover osteopenia, characterized by a decrease in the number and function of bone-forming osteoblasts. In addition, changes in bone matrix and mineral properties are observed in osteonectin-null mice. Osteonectin promotes osteoblast commitment and maturation, and as well as the survival of cells of the osteoblastic lineage. Moreover, osteonectin is essential for the bone-anabolic response to intermittent parathyroid hormone (PTH) administration, which is the only approved anabolic agent for the treatment of osteoporosis.

To determine whether polymorphisms in the osteonectin gene could be associated with bone mass, we examined 3 single nucleotide polymorphisms (SNPs) in the 3' untranslated region (UTR) of the osteonectin gene, in a well characterized cohort of men with idiopathic osteoporosis. We assembled 6 haplotypes consisting of these 3 SNPs, and found that the most common haplotype was associated with lowest bone mass in the patient population, whereas the second most common haplotype was found at a significantly higher frequency in the matched healthy controls and in patients with higher bone mass. Using a series of luciferase-osteonectin 3' UTR reporter constructs, we found that the osteonectin 3' UTR haplotypes differentially regulate gene expression. These data suggest that osteonectin levels may vary among individuals; it is possible that this variation could impact the anabolic response to PTH therapy. This project provided key data that seeded our present studies on the regulation of osteonectin and other skeletal genes by microRNAs (miRNAs).

miRNAs are a class of small, non-coding RNAs that can interact with target mRNAs and, for the most part, negatively regulate transcript expression. We found that the miR-29 family of miRNAs interacts with the osteonectin 3' UTR and down regulates osteonectin expression during osteoblastic differentiation. The expression of miR-29a and -29c are induced during osteoblast differentiation and by canonical Wnt signaling. In turn, canonical Wnt signaling rapidly down regulates osteonectin translation, providing a mechanism for fast repression of a protein that has a very stable mRNA. Since miR-29 and osteonectin are co-expressed in extra-skeletal tissues, we believe that the post-transcriptional mechanisms regulating osteonectin in osteoblasts are likely to be active in other cell systems.

In addition, we found that certain negative regulators of Wnt signaling are directly targeted and down regulated by miR-29. We demonstrated a novel regulatory circuit in which canonical Wnt signaling induces miR-29a transcription. The subsequent down regulation of key Wnt signaling antagonists, Dkk1, Kremen2, and sFRP2, by miR-29a potentiates Wnt signaling and promotes osteoblast differentiation. These findings provide insight into how miRNAs can fine tune the process of osteoblastogenesis.



Second Harmonic Generation (SHG) imaging of collagen matrix synthesized in vitro by wild type (left) and osteonectin-null (right) osteoblasts. Matrix synthesized by wild type osteoblasts is highly organized, whereas that synthesized by osteonectin-null cells is assembled in random networks.

Delany, A.M., McMahon D., Powell J.S., Greenberg D.A., Kurland E.S. Osteonectin/SPARC polymorphisms in Caucasian men with idiopathic osteoporosis. Osteoporosis International 19:969-973, 2008

Machado do Reis L., Kessler C.B., Adams D.J., Lorenzo J., Jorgetti V., Delany A.M. Accentuated osteoclastic response to parathyroid hormone undermines bone mass acquisition in osteonectin-null mice. Bone 43:264-273, 2008

Kapinas K., Kessler C.B., Delany A.M. miR-29 suppression of osteonectin in osteoblasts: regulation during differentiation and by canonical wnt signaling. J Cell Biochem 108:216-224, 2009

Kapinas K., Kessler C.B., Ricks T., Gronowicz G., Delany A.M. miR-29 regulates Wnt signaling in human osteoblasts through a positive feedback loop. J Biol Chem 285: 25221-25231, 2010

Interesting Science (contributed by ASMB members)

A novel splice variant in the N-propeptide of COL5A1 causes an EDS phenotype with severe kyphoscoliosis and eye involvement

Symoens S, Malfait, F, Vlummens P, Hermanns-Lê T, Syx D, De Paepe A.
PLoS One. 2011;6(5):e20121. Epub 2011 May 17

This is an interesting report describing the identification of a novel splicing mutation in an intronic region of the human *COL5A1* gene that encodes the N-propeptide of type V procollagen. The major strength of this paper is the combination of human clinical data with molecular analyses to attempt to understand the biological consequences of the mutation. The mutation was identified in the acceptor splice site region in intron 6 of COL5A1. Splicing studies showed that this mutation results in skipping of exon 7 or both exon 6 and exon 7, thereby resulting in alternatively-spliced truncated mRNA isoforms. Although truncated protein isoforms were not detected *in vivo*, recombinant N-propeptide proteins were identified following transfection of expression constructs into HEK-293 cells. This suggests that these alternative protein isoforms may be secreted into the extracellular matrix and disrupt collagen fibrillogenesis. Indeed, electron microscopic analysis of patient dermis tissue identified cauliflower-like collagen fibrils, which is the hallmark of disturbed collagen fibrillogenesis. Future studies to generate antibodies against these truncated protein isoforms should confirm if they are expressed in patient dermis. Also, it is not known at this stage whether the severe scoliosis and the abnormal eye phenotype are directly caused by a disturbed collagen V network. It may be that the N-propeptide of type V procollagen carries out other functions, the activity of which would be disrupted as a result of removal of exon 6/exon 7. The work presented in this paper may also be of interest to other groups studying collagen mutations and disease, such as Stickler-like syndromes caused by various mutations in the type II procollagen gene (COL2A1). A number of COL2A1 mutations identified to date are located within regions encoding the N-propeptide of type II procollagen and, interestingly, within the alternatively-spliced exon 2 domain. It may be worth re-exploring some of these mutations to determine whether or not they result in altered splicing and/or production of truncated or differentially-spliced protein isoforms.

Contributed by Audrey McAlinden

The requirement for fibroblasts in angiogenesis: fibroblast-derived matrix proteins are essential for endothelial cell lumen formation

Andrew C. Newman, Martin N. Nakatsu, Wayne Chou, Paul D. Gershon, Christopher C.W. Hughes
Mol. Biol. Cell, 22: 3791-800, 2011

Newman *et al.* recently published a report highlighting the critical role of fibroblast-secreted factors in regulating angiogenesis. Using an *in vitro* 3D model, the authors had previously observed enhanced endothelial cell (EC) sprouting and lumen formation when fibroblasts were co-cultured with endothelial cells. Thus the authors used a combined candidate gene approach, column chromatography (HPLC), and mass spectrometry (MS) to identify proteins secreted by fibroblasts that support angiogenesis by ECs. Two classes of fibroblast-derived factors were identified, one class promoted EC sprouting whereas the second class induced lumen formation. Factors that drive EC sprouting were ANG-1, angiogenin, HGF, TGF-ß and TNF however; this angiogenic cocktail resulted in sprouts without lumens. As lumen formation could be rescued by fibroblast-conditioned media, fibroblast secreted proteins were subjected to HPLC/MS fractionation to further identify factors that induced lumenogenesis. Five proteins were identified, collagen a1(I), procollagen C-proteinase enhancer (PCOLCE) 1, secreted proteins acidic and rich in cysteine (SPARC), IGF binding protein (IFGBP) 7, and ßig-h3. As both PCOLCE 1 and SPARC have been shown to be important for the processing and deposition of collagen I, increases in these proteins coincident with increased expression of collagen a1(I) were predicted to effect both the amount and the assembly of collagen in the ECM of the 3D cultures and thereby alter the physical properties of the EC milieu. In fact, a significant increase in gel stiffness upon addition of the lumenogenic factors was found. Thus, these authors concluded that fibroblast-derived factors contribute to greater ECM stiffness that in turn enhances lumen formation by angiogenic endothelial cells. The interplay between fibroblasts and endothelial cells in the tumor microenvironment is appreciated as a critical factor in tumor progression and an active area of research. These studies provide interesting insight into the role of fibroblasts and ECM in tumor angiogenesis.

Contributed by Amy Bradshaw

Latent TGF-β structure and activation

Shi, M., Zhu, J., Wang, R., Chen, X., Mi, L., Walz, T., and Springer, T.A.
Nature, 474, 343-349, 2011

This article is highly relevant to the extracellular matrix (ECM)/cell-matrix biology community from several perspectives. TGF-β is a major driving force in the production of ECM, since biosynthesis of many structural matrix proteins, matrix-degrading proteases and protease inhibitors is induced by this growth factor. Indeed, TGF-β and/or aberrant TGF-β signaling are believed to be major driving forces in fibrosis and connective tissue disorders. Conversely, ECM has a pivotal role in controlling TGF-β activity. TGF-β is synthesized as a proprotein which undergoes furin-mediated cleavage of the propeptide (latency associated peptides (LAP)), which nevertheless, remains attached non-covalently to the active growth factor. Together, LAP and the active growth factor dimer form a small latent complex (SLC), which is sequestered in the extracellular matrix by covalent interaction with latent TGF-β binding proteins-1, -3 or 4. This large latent complex in turn binds to fibrillin-1 and and fibronectin, positioning TGF-β appropriately for its activation by cells. The LAP contains an RGD sequence, which binds to α_v integrins. Previous analysis of integrin deficient mice (specifically *ITGB6* and *ITGB8* inactivation) had established the pivotal role of integrins in this process, thus placing TGF-β activation squarely at the cell-matrix interface. The ECM interactions are crucial for tethering of the TGF-β complexes and for creation or transmission of mechanical forces following integrin engagement. From yet another perspective, a biomechanics one, this study is of interest since it provides the molecular basis of a mechanical activity, i.e. of force transforming a molecular message into a cellular signal. Tensile forces applied to the RGD motifs by cell contraction via the α_v integrins are resisted at the opposite end of the SLC where it binds LTBP and is anchored to ECM. The pulling force that is generated "shells out" the TGF-β dimer from the grasp of the LAP and permits receptor recognition. It is an elegant regulatory mechanism that brings ECM to the forefront of cell regulation.

Why do I believe that this article is so impressive, and insightful? First, it provides the crystal structure of porcine SLC, which is elegantly described. This novel structure, both intuitively and together with the previous literature, explains how integrin engagement and cell contraction or motion might lead to activation. The studies identify a ring-shaped SLC complex, a novel fold for the LAP, and illustrate both how LAP might protect the growth factor from recognition by receptors and alter its conformation upon application of mechanical force. Second, it shows at lower resolution the structure of SLC complexed to integrins. Together, both constitute a significant advance.

The article is written with exemplary clarity and economy, yet displays admirable scholarship in its discussion of the implications for TGF-β biosynthesis, disease-causing mutation of TGF-β and regulation of the TGF-β superfamily. A similar novel fold appears to exist in all members of the superfamily. I recommend it as one of the must-read publications of 2011 for our community.

Contributed by Suneel Apte

In addition to the implications for integrins-ECM interactions in regulating TGF-β latency, the structural information provided by Shi et al also confirms previous biochemical findings regarding other matrix-related mechanisms of controlling TGF-β activation by the matricellular protein thrombospondin 1. The KRFK sequence in the TSR of TSP1 activates the latent complex by competitively disrupting the latency interaction between the LSKL sequence in the LAP and the RKPK sequence in the mature domain (Young and Murphy-Ullrich, 2004). The importance of the LSKL sequence was confirmed by Walton et al using mutational and biochemical approaches (Walton et al., 2010). Now Shi et al provide exciting structural information indicating that this $lysine_{26}$ in the LSKL ($lysine_{55}$ from signal peptide) sequence of the latency associated peptide (LAP) is a "fastener residue" in the latency lasso which comprises a structural constraint necessary for maintaining latency (Shi et al., 2011): integrin binding to LAP disrupts this lasso. This new structural information coupled with previous biochemical approaches suggests a common molecular basis for activation of the latent complex. The critical role of $lysine_{26}$ is consistent with the use of LSAL peptide as an inactive control for LSKL. Interestingly, other proteins with RFK motifs (neuropilin-1 has an RKFK sequence, F-spondin has KRFK in TSR repeat 6, and ADAMTS1 has KTFR) have also been shown to activate latent TGF-β and topical application of a peptide analogue of KRFK (KVK) has been used to stimulate TGF-β-dependent collagen expression in cosmetic applications (SYN-COLL™) (Attur et al., 2009; Bourd-Boittin et al., 2011; Glinka and Prud'homme, 2008). This suggests that the "RFK-LSKL" mechanism might be a common mechanism of latent TGF-β activation utilized by multiple proteins.

Attur, M.G., Palmer, G.D., Al-Mussawir, H.E., Dave, M., Teixeira, C.C., Rifkin, D.B., Appleton, C.T., Beier, F., Abramson, S.B., 2009. Faseb J 23, 79-89.
Bourd-Boittin, K., Bonnier, D., Leyme, A., Mari, B., Tuffery, P., Samson, M., Ezan, F., Baffet, G., Theret, N., 2011. Hepatology.
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Shi, M., Zhu, J., Wang, R., Chen, X., Mi, L., Walz, T., Springer, T.A., 2011. Nature 474, 343-349.
Walton, K.L., Makanji, Y., Chen, J., Wilce, M.C., Chan, K.L., Robertson, D.M., Harrison, C.A., 2010. J. Biol. Chem. 285, 17029-17037.
Young, G.D., Murphy-Ullrich, J.E., 2004. J. Biol. Chem. 279, 38032-38039.

Contributed by Joanne Murphy-Ullrich

Potent Inhibition of Heterotopic Ossification by Nuclear Retinoic Acid Receptor γ Agonists

Kengo Shimono, Wei-en Tung, Christine Macolino, Amber Hsu-Tsai Chi, Johanna J. Didizian, Christina Mundy, Roshantha A. Chandraratna, Yuji Mishina, Motomi Enomoto Iwamoto, Maurizio Pacifici, and Masahiro Iwamoto

Nat Med. 17(4): 454–460, 2011

Retinoic acid, a metabolite of Vitamin D, controls many different cell functions via binding and activation of nuclear receptors such as retinoid x receptor (RXR) and retinoic acid receptor (RARs, subtypes α, β and γ). Although retinoids are well known to developmental biologists due to their ability to orchestrate early development and organogenesis, they have been also the center of attention of general practitioners, due to their ability to ameliorate skin diseases and acute promielocytic leukemia.

Based on the observation that retinoid signaling is a strong inhibitor of chondrogenesis and that unliganded RAR transcriptional repressor activity is needed for chondrogenic differentiation, Shimono and colleagues thought to determine whether RAR activation might have beneficial effects in Heterotopic Ossification (HO), a severe pathology in which ectopic bone forms within muscles and connective tissues and near blood vessels or nerves. HO can be congenital (i.e. Fibrodysplasia Ossificans Progressiva) or the result of surgery or trauma. Indeed the authors provide evidence that treatment with a selective RAR-γ agonist blocks heterotopic ossification in mice. The RAR-γ-mediated beneficial effects seem to reside in its ability prevent mesenchymal stem cells to undergo a skeletogenic program. The authors elegantly show that RAR-γ agonist treated mesenchymal stem cells fail to respond to BMP-2 and thus activate skeletogenic genes such as Smads1/5/8. This result seems to agree with the finding that selective inhibitor of BMP type I receptor kinases in vivo induces partial inhibition of muscle-associated HO. Finally, the authors show the exciting data that RAR-γ agonist blocks HO in mice carrying a constitutively active form of ALK2 related to the mutated form found in individuals with fibrodysplasia ossificans progressive.

All together these findings clearly suggest that activation of RAR-γ can be viewed as a valid strategy to halt and ideally prevent trauma-mediated or congenital heterotopic ossification by counteracting BMP pro-skeletogenic action.

Contributed by Ambra Pozzi

Newsletter Committee Wants Your Help!

The newsletter committee is excited about our new features that include "Tell us what your lab is doing", "Interesting Science" along with new meeting reports and announcements. This year we will be publishing three newsletters and we seek your help to send us items you think would be interesting to the ASMB community. This could including pictures from past meetings, awards, and even commentary on a subject that you feel important to raise. Contributions should be sent to Marian Young the Editor in Chief of the newsletter (myoung@dir.nidcr.nih.gov) or Jennifer Holland, Executive Director of ASMB (jholland@faseb.org). We appreciate your help in this important endeavor.

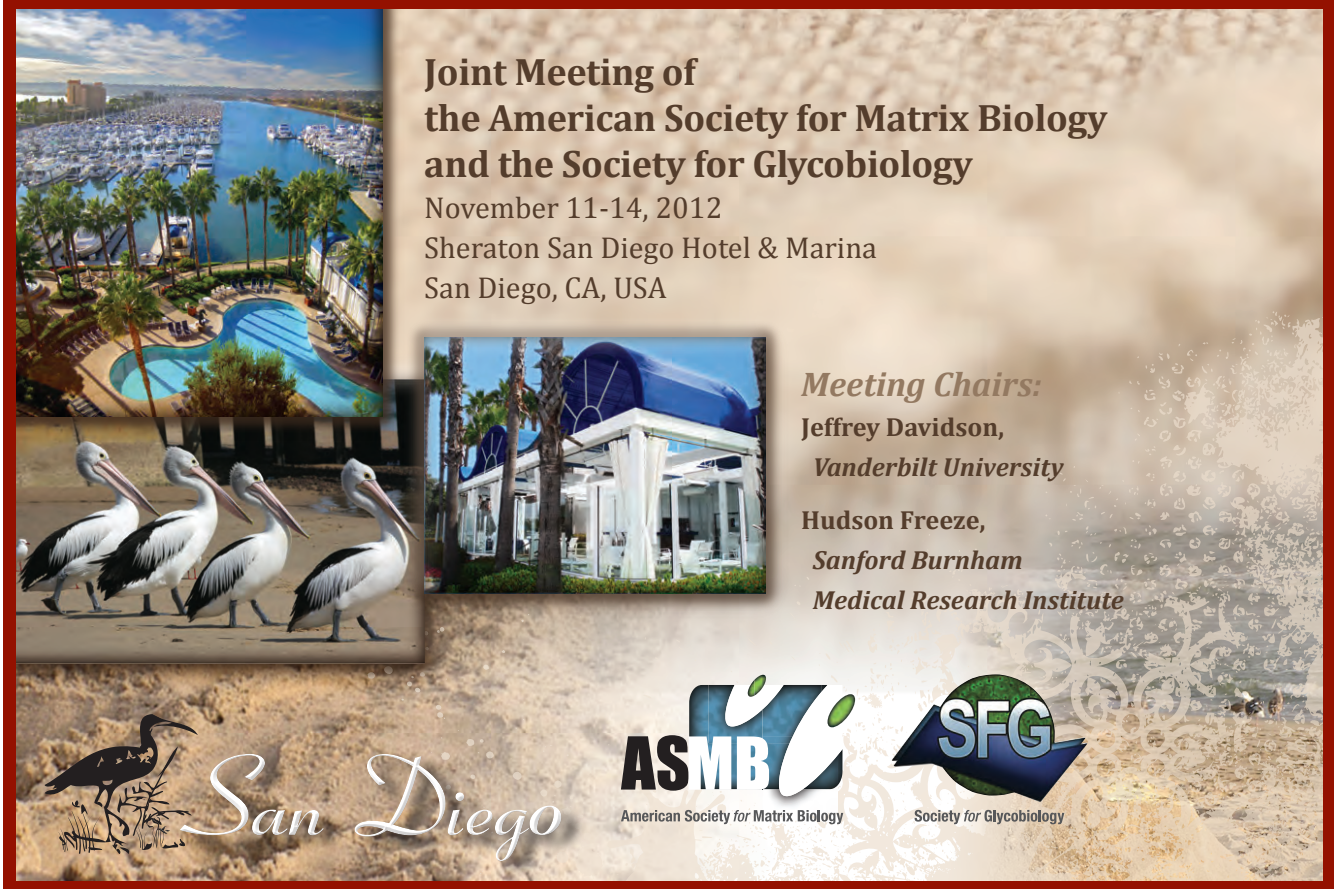
Marian Young (Editor-in-Chief)
Ambra Pozzi
Audrey McAlinden
Joanne Murphy-Ullrich
Bill Parks
Jean Schwarzbauer

We Seek Your Input

A message from Bill Parks

In an effort to provide additional value to being a member of ASMB, we are considering developing a publication alert (PubAl). The PubAl would be a monthly or every other month list of publications of interest to ASMB members. (If you happen to be a member of NAVBO, then you are familiar with what this service provides.)The PubAl would be assembled by a small working group of about 4 who would scan the table of contents of a range of high impact journals, such as Cell, Science, Genes and Developments, JCI, JCB, etc., and specialty journals, such as the various vascular/cardiology and bone-related journals. (We would skip articles in Matrix Biology as not only do we assume you are already perusing this journal, but all articles would be-by definition-of interest to ASMB members.) The PubAl would be distinct from the Newsletter and the Papers of Interests feature submitted by various members.

If you find the idea of an ASMB PubAl to be of interest and potentially valuable, we would greatly appreciate your feedback and ideas on to development activity. Please send comments to Bill Parks at parksw@uw.edu.



Now Online: Go to www.asmbsfg2012.org for the latest program updates, accommodation, registration and abstract information

Meeting Report

Translational Opportunities for the Heritable Disorders of Connective Tissue

A meeting on *Translational Opportunities for the Heritable Disorders of Connective Tissue* was held in Portland, OR, July 10–14, just prior to the National Marfan Foundation's 27th Annual Conference. This scientific meeting was hosted by the Portland Shriners Hospital for Children and was sponsored by the Coalition for Heritable Disorders of Connective Tissue and the National Marfan Foundation. "It was important to me to bring the research community together again as we mark the 25th anniversary of the discovery of fibrillin, the culprit protein in Marfan syndrome," said Lynn Sakai, PhD, member of the NMF Professional Advisory Board and chair of this research meeting. "Sharing information among researchers can advance the entire field of connective tissue disorders, including Marfan syndrome."

Lynn Sakai

Co-organizers of the meeting included Dr. Hans Peter Bächinger (Portland), Dr. Leena Bruckner-Tuderman (Freiburg, Germany), Dr. Peter Byers (Seattle), Dr. Hal Dietz (Baltimore), and Dr. William Horton (Portland). The meeting focused on diseases affecting skin (Epidermolysis Bullosa, Ehlers-Danlos), bone (Osteogenesis Imperfecta), cartilage (chondrodysplasia), and the vasculature (Marfan syndrome and related disorders).

The last meeting devoted to heritable disorders of connective tissue was held ten years ago. The National Institutes of Health (NIH) convened Workshops on Heritable Disorders of Connective Tissue in 1990, 1995 and 2000. The focus of the first two Workshops was finding the genes for the various heritable disorders and understanding whether or not mutations could be correlated with specific phenotypes. In 2000, the theme of the meeting was intentionally broader, focusing on multidisciplinary approaches and common themes in matrix biology. In the decade since the 2000 Workshop, tremendous progress has been made, leading notably to new therapies for Marfan syndrome and Epidermolysis Bullosa and to prospects for new therapies for a number of other heritable disorders of connective tissue.

The overall goals of the 2011 meeting were to critically assess current therapies, define current opportunities, and stimulate the translation of novel basic information into new therapies for the heritable disorders of connective tissue. In addition, the meeting stimulated new collaborations between investigators and promoted the research interests of young scientists. As in the past, a report of the meeting proceedings and recommendations will be prepared and published by the meeting organizers.

The hosts and the organizers are grateful to the National Institute for Arthritis, Musculoskeletal, and Skin Diseases (NIAMS) and the Beattie Charitable Trust at the Shriners Hospital for providing funding for this important scientific meeting.

Contributed by Eileen Masciale

Important Meeting Announcements

Osteoarthritis Research Society 2012 International Meeting

April 26-29, 2012 Barcelona, Spain

Abstracts due December 2, 2011

For detailed information, please consult the following web site: http://www.OARSI.org

Proteoglycans Gordon Research Conference

July 8-13, 2012 Proctor Academy, USA

The goal of the 2012 Gordon Research Conference on Proteoglycans will be to bring together leading national and international scientists to present their latest findings in proteoglycan research. Topics that will be discussed include mechanisms regulating the biosynthesis of the proteoglycans as well as their turnover. The role of proteoglycans in development, skeletal pathology, cancer, stem cells, regenerative medicine, inflammation and cardiovascular disease, and diseases of the nervous system will also be addressed. Recent progress within these areas will pay particular attention to proteoglycan structure, analysis and glycomics. Emphasis will be given to new insights into basic molecular mechanisms and to translational efforts designed to understand the role of proteoglycans in human disease as well as their use in prevention and novel therapeutics.

For details on registration and information on the programs, please consult the following web site: http://www.grc.org/programs.aspx?year=2012&program=proteoglyc

AADR Annual Meeting

March 21-24, 2013

The American Association for Dental Research (AADR), has more than 3,900 members in the United States with a mission (1) to advance research and increase knowledge for the improvement of oral health; (2) to support and represent the oral health research community; and (3) to facilitate the communication and application of research findings. This year the AADR will host its annual meeting in Tampa, Florida, March 21-24, 2012. A new feature this year will be "Meet a Mentor Luncheon" designed for student outreach. For information on registration, schedule of events and other meeting information please go to: http://www.iadr.org/i4a/pages/index.cfm?pageid=3941

Biomineralization Gordon Research Conference

August 12-17, 2012 Colby-Sawyer College, USA

The Gordon Research Conference on Biomineralization is exploring the basic principles by which organisms synthesize, control and make use of minerals, as well as potential applications of these. Spectacular advances have been made in the last years and are impacting various scientific fields, from biology to geology, from medicine to materials science and from evolutionary sciences to engineering. The central goal of this GRC is to create a stimulating environment for scientists from all these disciplines to discuss latest ideas and recent advances on how minerals interact with biomolecules, on how cellularly driven biomineralization is regulated by extracellular matrix molecules, on how the structures of mineralized tissues relate to their function and on how these principles might influence our thinking in materials science and engineering. The conference will cover all kinds of biominerals, including carbonates, phosphates, oxides and silica, in vertebrates, invertebrates and plants. It will also address human health issues related to abnormal mineralization or diseases connected to mineral growth and homeostasis in the skeleton or in teeth.

The great success which this GRC experienced in the last years is largely due to its lively afternoon poster sessions, complementing the invited lectures. This provides the best opportunity to present latest research and exchange ideas in a more informal setting and all conferees are encouraged to submit and present posters. As done in previous conferences, 8-10 poster contributions will be selected for short oral presentations. All this has made this GRC very attractive for young scientists and - as the consequence of a vote in 2010 - the GRC on bimineralization 2012 will be complemented for the first time by a Gordon Research Seminar preceding the meeting.

For details on registration and information on the programs, please consult the following web site: http://www.grc.org/programs.aspx?year=2012&program=biomin

Musculoskeletal Biology & Bioengineering Gordon Research Conference

August 5-10, 2012 Proctor Academy, USA

The Gordon Research Conference on Musculoskeletal Biology and Bioengineering (previously titled Bioengineering and Orthopaedic Sciences) has been the premier forum for presentation and discussion of new and unpublished information in the field, and has consistently led to new insights, new interactions, new collaborations, and new research directions. The study of the musculoskeletal system encompasses a number of interdisciplinary fields, particularly biology and bioengineering, and has ultimate applications in clinical areas including orthopaedic surgery, rheumatology, and radiology. The theme of the planned 2012 conference is "Musculoskeletal Science: Bedside to Bench to Bedside," and the meeting will consist of 9 separate sessions, consisting of invited speakers who are experts in their field and selected attendees at various levels of career development. The format of the planned conference remains essentially unchanged from previous years, with ample time for formal and informal discussions. Our unique focus in 2012 is to facilitate the physician-scientist interaction to bring new ideas and treatments to improve the lives of patients. This is exemplified by our session on Translational Studies on Enhancing Soft Tissue Healing, with both physicians and scientists lecturing together on cutting edge solutions and investigations into clinical problems. Some of the chief issues are specific to individual tissues, including bone, articular cartilage, intra-articular ligament, meniscus, and both rotator cuff and flexor tendons, as well as other multi-tissue skeletal structures. A number of key scientific and engineering topics are relevant to fabrication and manipulation of these tissues and organs. The conference will examine many of these topics, including biomaterial fabrication, stem cells, biomarkers, skeletal development, growth and regeneration, and translation of basic research to clinical practice. In addition, the conference also includes several high-profile research areas highlighting the close collaboration required between clinician-scientists and PhDs to improve patients' health outcomes.

For details on registration and information on the programs, please consult the following web site: http://www.grc.org/programs.aspx?year=2012&program=musculo

Stand with Science-Protecting the Funding of Science

If you'd like to learn more about a petition to a super-committee that presents the importance of funding in science and for our economic future please link to this video: http://www.standwithscience.org/

You can support the effort by signing their letter with instructions at that site for doing so.

Signal Transduction by Engineered ECM Gordon Research Conference

July 8-13, 2012 Univ. of New England, USA

The objective of the conference will be to share the newest knowledge from research on: the development and regulation of cellular microenvironments; the control of cell function by engineered microenvironments; dynamic tracking of cell fate in vivo; and application of such insights to the development of human clinical therapies for tissue repair and regeneration. The meeting brings together researchers in diverse fields of stem cell and developmental biology, chemistry, bioimaging and engineering. For more information, please consult the following web site: http://www.grc.org/programs.aspx?year=2012&program=signtrans

Check out the ASMB Website

www.asmb.net

Website Features

- Society information, including bylaws, history, Council members, etc.
- Complete ASMB awards information including criteria, applications and history
- Historical information about past ASMB meetings
- Career opportunities as posted on our new forum site with Scientist Solutions
- Other meetings listings
- Links to other resources such as partnering societies
- Newsletter archive
- Image Gallery

ASMB business

- Join/Renew your membership
- Manage and update your ASMB record
- Search our member database
- Link to Scientist Solutions forums
- Post related meetings
- Post job opportunities (under forums)
- Manage your *Matrix Biology* journal subscription

Need help navigating the new website?
Email asmb@asmb.net and we'll be happy to assist!

Don't Forget to Renew!

Your participation in our Society is the most important contribution you can make to helping increase awareness of research and opportunities in extracellular matrix biology.

With the help of your membership dues, we have added professional management of the society and provided students and postdoctoral fellows with travel awards to our national meeting. In the coming year, your dues will be at work to improve our website. We urge you to pay your dues so we can continue to add programs that benefit matrix biology.

The 2012 Annual Dues are $125 for regular membership and $75 for students/postdoctoral fellows. Dues can be paid any time via the ASMB website: http://www.asmb.net/

Alternatively, checks can be sent to the administrative office: ASMB, 9650 Rockville Pike, Bethesda, MD 20814.

Advantages of Membership:

- Discounts on *Matrix Biology* subscriptions (print and online)
- 33% Discount on volumes of the Biology of Extracellular Matrix series
- Discounts on Biennial Meeting registration
- Access to online forums and image galleries
- Receive society newsletters with article reviews and summaries
- Partner links to numerous other societies and valuable scientific resources
- Opportunities to submit abstracts for biennial meeting presentations
- Biennial meeting award eligibility
- Eligibility to run for Council positions and help direct the Society
- Access to list and view career opportunities within the community
- Make valuable professional connections with junior and senior researchers

Thank you to our sustaining members!

Rolf Brekken *UT Southwestern*
Elaine Fuchs *Rockefeller University*
Renato Iozzo, *Thomas Jefferson University*
Robert Mecham, *Washington University*
William Parks, *University of Washington*
Peter Yurchenco, *UMDNJ-RW Johnson Medical School*

JOB OPENINGS

Washington University in St. Louis
SCHOOL OF MEDICINE

Faculty Position in Cell Biology & Physiology Washington University School of Medicine

The Department of Cell Biology and Physiology at Washington University School of Medicine in Saint Louis invites applications for tenure-track faculty positions at the level of Assistant Professor. Outstanding individuals working in any area of cell biology and physiology are encouraged to apply. At present, research in the department spans a wide range of topics including membrane trafficking and transport, signaling, cell cycle, cancer, aging, apoptosis, cell motility, extracellular matrix and prion biology. Additional information about the department is available at www.cellbiology.wustl.edu.

Applicants should email their curriculum vitae and a brief description of their research interests to the attention of Dr. Helen Piwnica-Worms at **cellbio.search@wustl.edu**. Applicants should request three letters of recommendation to be sent to the same address.

Washington University is an Equal Opportunity Employer and is committed to the recruitment of candidates traditionally underrepresented on university faculties.

Assistant/Associate Professor Boston University Henry M. Goldman School of Dental Medicine Department of Periodontology & Oral Biology

The Department of Periodontology & Oral Biology seeks candidates for a full-time faculty position at the rank of Assistant or Associate Professor. A competitive salary package, commensurate with experience and academic qualifications is available. Candidates must possess a PhD or equivalent degree; applicants holding a DDS, DMD or MD degree are preferred. Applicants should have extramural research funding and an independent research program that integrates well with oral biology and oral medicine. Qualified candidates should submit a letter of interest, including a description of research interests and any relevant teaching and mentoring experience with a *Curriculum Vita* to:

Philip C. Trackman, Ph.D.
Professor and Director of Oral Biology Research
Boston University Henry M. Goldman School of Dental Medicine
700 Albany Street, W-201
Boston, Massachusetts 02118
Email: trackman@bu.edu
Fax: 617-638-4924

Review of applications will begin immediately and continue until the position is filled. Boston University is an equal access, equal opportunity and affirmative action employer that is fully committed to achieving a diverse faculty and staff. Persons are selected on the basis of ability without regard to race, color, religion, sex, age, national origin, physical or mental disability, sexual orientation, genetic information, military service or marital, parental or veteran status.

Postdoctoral Opportunities at the NIH and the FDA

The National Institute of General Medical Sciences (NIGMS) is seeking truly outstanding postdoctoral fellows for consideration to positions in the NIGMS Intramural Pharmacology Research Associate (PRAT) Program see: http://www.nigms.nih.gov/Training/PRAT.htm This highly competitive postdoctoral fellowship program provides up to three years of support for exceptional postdoctoral trainees to work in the intramural laboratories of the National Institutes of Health (NIH), or the Food and Drug Administration (FDA). The Program is intended for individuals with strong leadership potential who have a background in the basic or clinical sciences and seek advanced experience in an area of pharmacology, or those with a pharmacology background who wish to gain experience in new fields. Research opportunities in pharmacology are broadly defined and can include, for example, molecular pharmacology, biochemistry, signal transduction mechanisms, drug metabolism, immunopharmacology, chemistry and drug design, endocrinology, cell biology, structural biology, neuroscience, gene therapy, or clinical pharmacology. Applicants must have received a Ph.D. or a professional degree (M.D., D.D.S, D.O., D.V.M., or Pharm.D.) in a basic or clinical science within the last five years, and they must be citizens or permanent residents of the U.S. Applicants may apply prior to coming to NIH or FDA, or they may have started postdoctoral research at NIH or FDA within the 12-month period prior to the application receipt deadline.

If you have outstanding postdoctoral fellows with leadership potential, who you feel would be a good fit for this program, we ask that you please encourage them to apply.

Applications are available on line https://prat.nigms.nih.gov/ Receipt deadline is January 27, 2012.

For more information contact Dr. Marino marinop@nigms.nih.gov or Dr. Okita okitar@nigms.nih.gov

Contributed by: Dr. Marino & Dr. Okita
PRAT Program Co-Directors

Postdoctoral Research Position in Anti-angiogenesis and Cartilage Washington University, St. Louis

Experience in angiogenesis, integrin signal transduction, apoptosis, matrix protein chemistry or cancer would be helpful. Apply to Linda J. Sandell, PhD, Washington University in St. Louis, sandell@wustl.edu

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Funding Opportunity

National Marfan Foundation Request for Proposals for the Victor A. McKusick Fellowship Program. Two-year fellowships awards are available for Ph.D.'s for up to $50,000 per year and M.D.'s up to $75,000 per year to cultivate promising young research or physician scientists conducting basic, translational or clinical research in the field of Marfan syndrome and related disorders. Application deadline is: February 6, 2012. Please visit the www.marfan.org for application and guidelines.