American Society for Matrix Biology

President's Letter

Jean Schwarzbauer

Dear Fellow Matrix Biologists,

A primary responsibility of the ASMB President is to organize the biennial meeting. Preparations for our next meeting, joint with the Society for Glycobiology, are well underway. The meeting program is in order, fundraising is moving along, junior and senior awardees have been named, and we are actively publicizing the meeting. Hopefully, you are planning to submit your most exciting research for presentation in San Diego in November.

There is now a bit of a lull in society activities, which has gotten me thinking about the role of the ASMB President. Should the President be adding value to the society for future matrix biologists, and, if so, what would that value be? I looked for guidance in the ASMB mission statement: *to promote research and education on the extracellular matrix (ECM), its role in human disease, and its application to therapy.* Accurate but vague. The Society's by-laws note that the President's role is to preside over meetings, sign official documents, and "have all the powers and duties normally incident to the office of president and as prescribed by the Council". It sounds like a tall order but what does it really mean? What are the President's "powers and duties"? What should future presidents, vice-presidents, and even presidential candidates be thinking about when they sign on to do this job or run for election?

All of these questions led to a decision, my contribution to the future of the ASMB will be to define the president's powers and duties, as well as the duties and responsibilities of other officers, councilors, and committees. In short, my goal is to develop an ASMB Handbook. This project will be accomplished with much help from Jen Holland, our dedicated director, and with input and feedback from the Executive Committee, the Council, and you, the membership.

Why do we need a handbook when we have by-laws? In general, by-laws are the rules that govern the day-to-day operations of a society. By-laws describe the elected positions, the number and types of standing committees, when to hold meetings, whether to have insurance, how to nominate candidates for elections, etc. We refer to the by-laws whenever there are questions about the society's procedures, and they are a useful resource. Our by-laws are available for download at the ASMB website if you are interested. As "rules", the by-laws do not explain the responsibilities of our leaders, the details of organizing our biennial meeting, the goals of our committees, or other aspects of society management and outreach.

What will be in the handbook? The plan is to define the structure of the society so that the handbook adds depth and breadth to the organization. It will describe our membership, its governance, the meeting and its management, the awards we give out, and other policies. If we started from scratch on this project, it would not be completed by the end of my term in December. Fortunately, the resourcefulness of Jen Holland has primed the pump, so to speak, by providing me with a preliminary handbook based on what other societies do. This start-

ing material will be modified and molded into an ASMB-specific document. The authority of each elected officer and councilor will be explained along with her/his responsibilities and purview. Activities beyond planning the biennial meeting will be elaborated. Procedures that are now passed on mostly by word of mouth will be spelled out. In the case of the president, a list of "powers and duties" will be elucidated, and a timeline for developing and implementing new initiatives will be outlined.

In my opinion, the handbook will be most helpful for presidents-elect and new councilors. Currently, the President presides over one face-to-face meeting of the Executive Committee and the Council, usually held the day before our biennial meeting. This meeting is the first opportunity for the President to have an open discussion of her/his ideas and plans for the society. Since this meeting occurs very near the end of a President's term, implementation of new initiatives, no matter how important or exciting, is unlikely to occur before the term of service is completed. Perhaps the Executive Committee - Council meeting is the place where the President-elect (instead of the President) proposes ideas for her/his presidency and gets Council feedback. Then, during the ensuing presidential term, the plans can be carried out and progress can be reported at the next meeting two years hence. This suggestion and other ideas will be part of the discussion as we write the handbook.

At the very least, my goal is to provide future elected officers with a handbook of guidelines that will facilitate further development of our society and add value to an already exciting community. The handbook will evolve as future ASMB leaders add to and subtract from it as the society matures. I hope it will be a useful tool for the ASMB.

Thanks and best wishes,

Jean Schwarzbauer
ASMB President

Thanks to the efforts of a reliable and creative program committee, the organization of the 2012 joint meeting of the American Society for Matrix Biology and the Society for Glycobiology has moved into the critical phase in which you, our members , will provide the critical element: submission of your latest and greatest abstracts. Don't delay! As you can see from the included program description and the more detailed meeting website, we will intercalate a phenomenal roster of invited speakers with sixteen abstract-driven Concurrent Sessions that, combined with poster presentations, will showcase your creative activity.

The concept of the combined meeting stems from a successful, SfG-sponsored session in our 2010 Charleston event. Your program committee (David Roberts, Tom Barker, Elaine Davis, Linda Sandell, Ambra Pozzi, Jean Schwarzbauer , and Joanne Murphy-Ullrich) worked together with our counterparts in SfG (Hudson Freeze (SfG co-chair), Robert Haltiwanger, Linda Hsieh-Wilson, Jeff Esko, and Yu Yamaguchi) to develop a balanced meeting design with a very conscious effort to provide the combined audience with maximum opportunities for scientific exchange. We did not have to do much arm-twisting to obtain speakers once the program was set, since ASMB and SfG meetings are certainly recognized as premier events. As a result of assembling this roster, our R13 submission to the NIH recently received an outstanding score. Of course, we'll have to wait to learn how the priority score translates in to funding. We rely on this source to provide you with the best speakers and the highest level of meeting support for the young investigators who will carry our field forward. In addition, Maurizio Pacifici, together with our fearless FASEB account executive, Jen Holland, led a successful effort to engage numerous corporate sponsors to help underwrite the event.

We're well aware that San Diego is hardly a new venue for ASMB, but the repetition will be tempered by a smaller-scale hotel and a greener setting. There should be ample time for socializing, and we've been working hard make the final evening's banquet something that you won't want to consider as optional.

Another feature of this year's event is the inclusion of four satellite symposia: two glycomics groups and offerings from our colleagues in TERMIS and the Wound Healing Society. The sessions will be held on Sunday, November 11 in advance of the Opening Reception, and you should seriously consider coming in a day early for these bonus events. These symposia will offer highly complementary perspectives on matrix biology and glycobiology. Our website will keep you up to date on further developments in all aspects of the meeting. Stay in touch.

Jeff Davidson,President-elect

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)

Register Now

Early Bird rates end 7/2/12

www.asmbsfg2012.org

Important Dates

- **March 2012:** Registration and abstract submission opens
- **July 2nd:** End of early bird registration and regular abstracts
- **July 16-31:** Late breaking abstract submission
- **October 12:** Guest room reservation deadline
- **October 17:** Last day to register online

JOINT MEETING OF THE

Society for Glycobiology & American Society for Matrix Biology

Nov. 11-14, 2012 • Sheraton Hotel and Marina • San Diego, CA

Plenary Sessions

1. Stem and Progenitor Cells, Genetic Therapy and Regenerative Medicine
2. Biomaterials and Matrix Engineering
3. Biosynthesis, Secretion and Assembly
4. Immunology and Inflammation
5. Rare Glycosylation and Matrix Diseases
6. Development and Morphogenesis

Concurrent Sessions

1. Matrix and Carbohydrate Immunology
2. Matricellular Proteins
3. Receptors, Signaling, and Cytoplasmic Glycosylation
4. Vascular Biology, Elastic Fibers, and Related Diseases
5. Carbohydrate and Matrix Metabolism in Disease States
6. Basement Membranes
7. Genetics and Gene Expression
8. Mechanobiology & Biomedical Engineering
9. Stem/Progenitor Cells and Their Environment
10. Biosynthesis, Protein Folding, Secretion, and Matrix Assembly
11. Cancer Microenvironment
12. Chemical Glycobiology/Glycomics
13. Development
14. Fibrosis, Proteolysis, and Tissue Repair
15. Host-Pathogen Interactions

Organizers:
Jeff Davidson
Hudson Freeze
David Roberts
Jean Schwarzbauer
Robert Haltiwanger

ASMB American Society for Matrix Biology

SFG

Special Guest Symposia

Presented by partners of both ASMB and SFG. Including TERMIS and a special Glyco Analytical industry session shared by Qun Zhou (Genzyme) and John Briggs (Genentech)

For information on the full program, please visit: www.asmbsfg2012.org

Tell Us What Your Lab is Doing!

From the laboratory of Kurt Hankenson
University of Pennsylvania

The Hankenson laboratory is located in the School of Veterinary Medicine at the University of Pennsylvania. The laboratory collaborates closely with colleagues in both the Department of Orthopaedic Surgery of the Perelman School of Medicine and in the Department of Clinical Studies-Philadelphia, School of Veterinary Medicine. The laboratory focuses on determining how matricellular proteins direct mesenchymal progenitor cell differentiation during bone development and remodeling. We started by studying the role of thrombospondins, in particular thrombospondin-2 (TSP2). TSP2 is a homotrimeric member of the thrombospondin family that is most closely related to TSP1. In dermal wounds, mice lacking TSP2 have increased neovasculature in the granulation tissue and an increased rate of wound closure. An analogous function is seen during bone fracture healing. Mice lacking TSP2 have increased bone within the fracture callus at day 10 post fracture. Interestingly, these knockout mice also have reduced cartilage (a tissue that requires hypoxic conditions to form) and increased vasculature, suggesting that TSP2 is regulating tissue healing by partially regulating the oxemic tissue state.

To determine whether oxemic regulation is the driving mechanism behind the alterations in TSP2-null fracture healing we are currently measuring hypoxia and the rate of healing and bone formation in an ischemic fracture model. Preliminary findings suggest that mice deficient in TSP2 have enhanced bone regeneration.

Recent mechanistic work with TSP2 has focused on the potential role of TSP2 in regulating Notch signaling. Our collaborative studies with Michael Wang's laboratory at the University of Michigan, have demonstrated that TSP2 promotes Notch signaling in some cells. Interestingly, when we examined the effect of TSP2 on Notch signaling via Jagged-1 ligand in osteoblast progenitors, TSP2 inhibited Notch signaling. Thus, the modulatory role of TSP2 on Notch signaling appears to be highly contextual, consistent with our understanding of TSP2 biology. This initial work with Notch has led to a focus in the laboratory exploring the role of Notch signaling by Jagged-1 in bone development, maintenance, and regeneration.

Hankenson Lab: Rear, left to right: Derek Dopkin, Lorraine Mutyaba, Mike Dishowitz, Fengchang, Zhu; Front, left to right: Emily Miedel, Nicole Belkin, Allison Williams. Not shown, Mariya Sweetwyne.

In addition to our interest in TSP2 and Notch, our laboratory is investigating the role of a newer member to the TSP molecular family, R-spondin 2 (Rspo2). R-spondins, are so named because they contain thrombospondin repeats as well as a furin domain that can regulate Wnt signaling. Rspo2 knockout animals have extensive skeletal defects. Our lab has shown that expression of Rspo2 in a mesenchymal progenitor cell line increases the expression of osteogenic genes and results in increased mineralization, an indicator of osteogenesis. The expression of Rspo2 is regulated by a non-canonical Wnt protein, Wnt11. This has led us to also examine the role of Wnt11 in bone formation and maintenance. Surprisingly, we find that the loss of Wnt11 mice causes an increased and prolonged maintenance of trabecular bone in both Wnt11 null- and heterozygous female mice.

Together these studies, which developed from an interest in matricellular protein regulation of bone structure and function, are allowing us to build a library of mechanisms to direct osteoblastogenesis and bone healing.

Text Prepared by Dr. Mariya Sweetwyne, postdoctoral fellow in the Hankenson laboratory.

Taylor DK, Meganck, JA, Terkhorn S, Rajani R, Naik A, O'Keefe RJ, Goldstein SA, Hankenson KD. Thrombospondin-2 influences the proportion of cartilage and bone during fracture healing. J Bone Miner Res 2009 Jun 24(6):1043-1054.

Meng H, Zhang X, Hankenson KD, Wang MM Thrombospondin 2 potentiates notch3/jagged1 signaling. J Biol Chem 2009 Mar 20:284(12):7866-741.

Friedman MS, Oyserman SM, Hankenson KD Wnt11 promotes osteoblast maturation and mineralization through R-spondin2. J Biol Chem 2009 May 22:284(21):14117-25

Hankenson KD, Sweetwyne MT, Shitaye H, Posey KL Thrombospondins and novel TSR-containing proteins, R-spondins, regulate bone formation and remodeling. Curr Osteoporos Rep 2010 June 8(2):68-76

Interesting Science (contributed by ASMB members)

Identification of a mutation causing deficient BMP1/mTLD proteolytic activity in autosomal recessive osteogenesis imperfecta

Martínez-Glez V, Valencia M, Caparrós-Martín JA, Aglan M, Temtamy S, Tenorio J, Pulido V, Lindert U, Rohrbach M, Eyre D, Giunta C, Lapunzina P, Ruiz-Perez VL

Human Mutation 33:343-350 (2012)

Osteogenesis imperfecta (OI), characterized by decreased bone strength leading to increased frequency of fractures, ranges in severity from mild to perinatal lethal. Genes encoding the two collagen I [col(I)] chains became the first candidate genes for OI solely because col(I) is the most abundant protein component of bone. Surprisingly, given the many genes involved in bone formation/function, most autosomal dominant (AD) OI has indeed been shown to be due to mutations in these two genes. Recently, genetic lesions underlying rare autosomal recessive (AR) forms of OI have been elucidated. Of the seven AR loci described, three affect formation of a single 3-hydroxyproline in the col(I) triple helical domain, two encode chaperones involved in procollagen I secretion, and a sixth encodes transcription factor osterix, involved in osteoblast differentiation. A seventh locus encodes a col(I)-interacting protein of unknown function. Col(I) is synthesized as procollagen I, a precursor with N- and C-terminal propeptides that are cleaved to produce mature monomers. *In vitro* studies have implied that C-propeptide retention is incompatible with fibrillogenesis and thus viability. However, two recent studies indentified rare cases of mild AD-OI with procollagen I C-propeptide cleavage site mutations likely to affect kinetics of, rather than block, C-propeptide removal. Now Martínez-Glez et al. have described a missense substitution in locus *BMP1* that causes severe AR-OI combined with large umbilical hernias. *BMP1* encodes alternatively spliced RNAs for proteinases *BMP1* and mTLD, previously shown to cleave C-propeptides of fibrillar collagens. As in cases of mild AR-OI caused by C-propeptide cleavage site mutations, C-propeptide processing is decreased, but not absent in patients with the *BMP1* substitution. Martínez-Glez et al failed to detect C-propeptide cleaving activity in the aberrant mTLD. Thus, residual C-propeptide cleavage may be due to the proteinase mTLL1, which is closely related to BMP1/mTLD, but is encoded by a separate gene. Of interest is the severity of OI caused by the *BMP1* substitution, compared with the mildness of OI caused by C-propeptide cleavage site mutations. This contrast may be explained by previous findings that BMP1-like proteinases have manifold functions. These include biosynthetic processing of various proteins involved in ECM formation, including lysyl oxidase, important for cross-linking of collagen fibrils; small leucine-rich proteoglycans implicated in modulating fibrillogenesis; proteins that initiate mineralization of tissues; and TGFβ1, which can induce ECM formation. BMP1-like proteinases also activate BMP4, which is involved in dorsoventral patterning. Thus, the *BMP1* substitution may have various effects on bone biology. The large umbilical hernias of patients with the *BMP1* substitution are a feature not usually associated with OI, but are reminiscent of the previous observation that mice null for the corresponding *Bmp1* gene have persistent herniation of the gut resulting from failed closure of the ventral body wall. It will be of interest to determine the extent to which such herniations in mice and humans result from defective ECM and/or mild dorsoventral patterning defects arising from lowered levels of active BMP4.

Contributed by Dan Greenspan

Injection of amniotic fluid stem cells delays progression of renal fibrosis

Sedrakyan, S., S. Da Sacco, A. Milanesi, L. Shiri, A. Petrosyan, R. Varimezova, D. Warburton,
J. Am. Soc. Nephrol in Press (2012)

Alport syndrome is a basement membrane disease caused by mutations in any one of three type IV collagen chains (COL4A3, COL4A4, or COL4A5) that are found in the kidney glomerular basement membrane (GBM). It is a clinically important disease that causes kidney failure, in many cases in adolescents. There are ultrastructural lesions present in Alport GBM that are thought to promote or be indicative of glomerular damage. Because there are several good mouse (and dog) models of Alport syndrome, several types of therapies have been attempted. Theoretically, the best therapy would be one that results in either protection of the GBM or its normalization by restoring the missing collagen IV α3α4α5 network. Several groups have attempted the latter by transplanting wild-type bone marrow or infusing other types of cells (embryonic stem cells, blood cells) into Alport mice and have reported rescue of GBM composition accompanied by improved kidney function, but the results have been controversial and difficult to interpret. This paper from Laura Perin and colleagues is important because it clearly shows that treatment with pluripotent c-kit(+) mouse amniotic fluid stem cells (AFSCs) can lead to protection of kidney function and increase in life span without any ameliorative effect on GBM collagen IV identity, though there was improved GBM integrity. Injection of a million AFSCs into the left ventricles of Alport mice before disease onset reduced fibrosis in glomeruli and in the interstitial compartment of the kidney, and this was associated with down-regulation of transcription factors known to be important in the "pro-matrix synthesis" TGFbeta pathway. There was also a reduction in

M1-type pro-inflammatory macrophages, and an increase in the M2-type macrophages that promote tissue remodeling and repair. These results could have important implications for slowing kidney disease progression in human Alport patients and are certainly worthy of further investigation aimed at defining how the injected AFSCs are having these effects. Finally, these results reveal a commonality with other diseases of matrix or cell/matrix interactions, such as Marfan syndrome and muscular dystrophy, in which therapeutic approaches aimed at secondary effects, rather than the primary genetic defect, are quite promising, if not already proving effective in patients.

Contributed by Jeff Miner

Teriparatide as a chondroregenerative therapy for injury-induced osteoarthritis

Erik R. Sampson, Matthew J. Hilton, Ye Tian, Di Chen, Edward M. Schwarz, Robert A. Mooney, Susan V. Bukata, Regis J. O'Keefe, Hani Awad, J. Edward Puzas, Randy N. Rosier, Michael J. Zuscik

Science Transl Med 3:101ra93 (2011)

Articular cartilage is a long-lived and uniquely designed tissue that sustains the nearly friction-less movement of synovial joints such as knees and elbows through life. The tissue is able to do so because of its abundant extracellular matrix that is mainly composed of aggrecan multimers and collagen type II fibrils. The proteoglycans provide resilience and the collagen fibrils provide tensile strength. The surface zone of the tissue abutting the synovial cavity is specialized and secretes hyaluronan and lubricin that are essential lubricans in the synovial fluid. Unfortunately, articular cartilage is prone to several pathologies the most common of which is osteoarthritis (OA) and during which the tissue progressively loses its structure and function, with major health and healthcare consequences. The tissue has poor intrinsic regenerative and repair capacity and thus, there is an urgent need to find new therapeutic ways to invigorate and stimulate it as a possible treatment for joint disease. The study by Sampson et al. is thus a welcome and very important contribution to the field. Previous work had shown that parathyroid hormone (PTH) is able to stimulate matrix synthesis and prevent the phenotypic destabilization of chondrocytes in vitro. The authors have now found that gene expression of the type 1 PTH receptor is up-regulated in articular cartilage in OA patients and mice subjected to injury-induced knee OA. Because such up-regulation may represent an anabolic reparative attempt, the authors tested whether recombinant human PTH 1-34 (teriparatide) –an FDA-approved treatment for osteoporosis- could have beneficial effects. They administered systemic teriparatide (brand name Forteo) to mice immediately after or 8 weeks after the OA-inducing knee surgery. Knees were examined at 4, 8 and 12 weeks from injury by micro-computed tomography and biochemical and molecular procedures. The data indicate that immediate administration of the drug stimulated proteoglycan content and inhibited articular cartilage degeneration compared to vehicle-treated operated mice. Delayed treatment started at 8 weeks from surgery elicited a reparative effect on the tissue. The beneficial effects of teriparatide were revealed by decreases in expression of collagen X, Runx2, matrix metalloproteinase 13 and aggrecan cleavage product NITEGE all of which are indicative of articular cartilage destabilization and degenerative progression toward a phenotype resembling that of growth plate cartilage (a transient tissue that sustain skeletal growth until end of puberty and then disappears). As the authors poignantly conclude, the data strongly indicate that teriparatide could be used to stimulate matrix regeneration and slow down the often inexorable progression of OA. Incidentally, the data also reinforce evidence from several biomedical fields that drugs approved for a given pathology (osteoporosis in the present case) could actually have additional and equally important therapeutic uses.

Contributed by Maurizio Pacifici

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

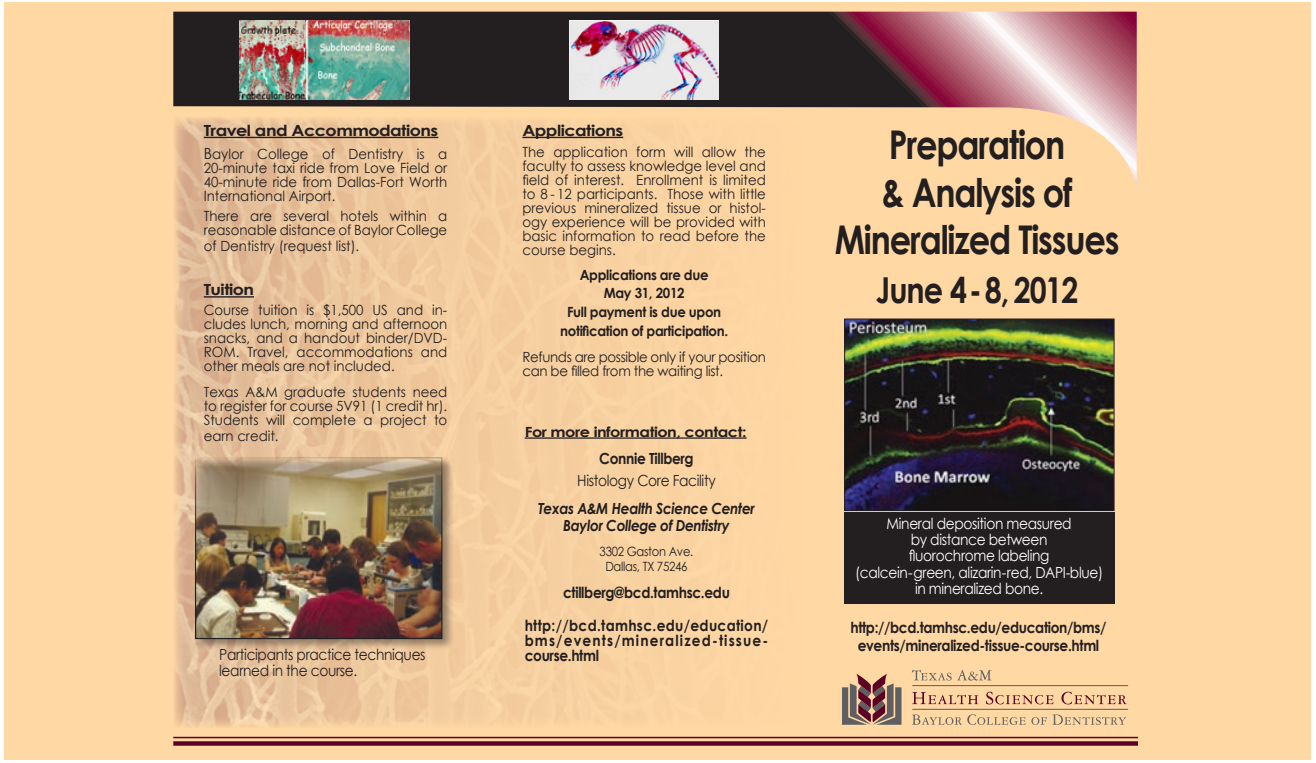
Preparation and Analysis of Mineralized Tissue

Baylor College of Dentistry, Dallas, Texas

The third annual course on the Preparation and Analysis of Mineralized Tissue at Baylor College of Dentistry (BCD) was held during the week of June 6 through June 10, 2011. The attendees learned about a variety of topics having to do with methods of preparing mineralized tissue for histological examination.

The twelve participants taking the course this year came from a variety of locations: six attendees from BCD included one postdoctoral fellow, three graduate students, and one in-coming dental student. Another member of the group was a high school student from Plano, TX. A research assistant from BCD's Department of Diagnostic Sciences was joined by two research assistants from the Institute for Regenerative Medicine at the College of Medicine, one of BCD's sister components. From farther away came a member of the Department of Pharmacology and Toxicology at Rutgers University. Two participants came from even farther, one from Brazil and the other from Poland.

The course was organized by Dr. Kathy Svoboda, Professor, and Ms. Connie Tillberg, Histologist/Lab Coordinator. The format for the course included instruction by Ms. Tillberg on various test procedures as well as presentations by BCD faculty on other analytical procedures and applications of the methodology. These faculty included Drs. Robert Spears, Bruno Ruest, Lynne Opperman, Peter Buschang, Paul Dechow, Phillip Kramer, Elias Kontogiorgos, and Jerry Feng. The course participants learned about perfusion and fixation, paraffin processing of hard tissue, methyl methacrylate processing, sample preparation for scanning and light microscopy, and mineralized tissue analysis using microCT. After listening to the instructors, the participants had the opportunity to have hands-on practice at performing some of the analytical methods. The week wrapped up with a question-and-answer session and an opportunity for the attendees to get help with trouble-shooting problems. The course was co-sponsored by the Associate Dean for Research and Advanced Education, BCD Biomedical Science Department and the American Association of Anatomists. The 2012 course is scheduled for June 4-8th. For more information contact Connie Tillberg at ctillberg@bcd.tamhsc.edu or go to the website :



http://bcd.tamhsc.edu/education/bms/events/mineralized-tissue-course.html

As an ASMB member, you receive a 33% discount on all new volumes of the **Biology of Extracellular Matrix** series, with a portion of royalties going to the Society. The first new volume in the series: "***Extracellular Matrix: An Overview***" is now available. The objective of this overview volume is to update and build upon topics discussed in previous volumes in this series as well as in classic ECM review texts, such as Betty Hay's *Cell Biology of Extracellular Matrix*. The new volume focuses on the major molecules that make up the ECM and will serve as an up-to-date reference for the beginner and matrix aficionado alike. The volume chapters are:

An Overview of Extracellular Matrix Structure and Function
Jürgen Engel and Matthias Chiquet

Fibronectin and Other Adhesive Glycoproteins
Jielin Xu and Deane Mosher

Collagens, Suprastructures, and Collagen Fibril Assembly
David E. Birk and Peter Brückner

Basement Membranes
Jeffrey H. Miner

Hyaluronan and the Aggregating Proteoglycans
Thomas N. Wight, Bryan P. Toole, and Vincent C. Hascall

Small Leucine-Rich Proteoglycans
Renato V. Iozzo, Silvia Goldoni, Agnes Berendsen and Marian F. Young

Microfibrils and Fibrillin
Dirk Hubmacher and Dieter P. Reinhardt

Elastin
Beth A. Kozel, Robert P. Mecham, and Joel Rosenbloom

Lysyl Oxidase and Lysyl Oxidase-Like Enzymes
Herbert M. Kagan and Faina Ryvkin

The Fibulins
Marion A. Cooley and W. Scott Argraves

Matricellular Proteins
David D. Roberts and Lester F. Lau

Other volumes in the series include:
Glycans in Disease and Therapeutics
Extracellular Matrix Degradation
Extracellular Matrix in Development (in production)
Evolution of Extracellular Matrix (in production)

Information about this and other volumes in the series can be found at www.springer.com/series/8422. The ASMB member discount can only be obtained when orders are placed directly at orders-HD-individuals@springer.com. Please confirm with your order that you are a member of the ASMB and that you would like to order the volume at the special member price.

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

7th European Elastin Meeting

September 1-4, 2012, Ghent, Belgium

The 7th European Elastin Meeting will be held this year in Ghent, Belgium. The program offers broad coverage of current progress and controversies in the structure and biology of elastin as well as in diseases of elastic fibers. Anne De Paepe and Olivier Vanakker, Chairs of the organizing committee, invite you to visit the meeting web site for information on registration, the shedule of events and other meeting information: http://www.elastin2012.be

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

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

Matrix Metalloproteinases Gordon Research Conference

May 19-24, 2013 Ill Ciocco Tuscany Resort
Barga, Tuscany, Italy

Proteases co-evolved with their substrates as integral components of specific networks or pathways in development and homeostasis. Elucidating their role in disease, resulting either from loss of function or over-expression, requires a sophisticated understanding within the context of biological networks. Furthermore, successful development of novel therapies relies on an integrated, multidisciplinary approach to investigating such networks and pathways.

The 2013 GRC on Matrix Metalloproteinases will develop these themes in the context of major functional networks, and will be inclusive of multiple organ/biological systems and a variety of disease pathways. The conference will feature fundamental and applied research on all aspects of MMPs, ADAM, ADAMTS, and astacin metalloproteinases, as well as their natural and synthetic inhibitors to highlight general principles as well as specific properties. The role of these metalloproteinases in molecular maturation and turnover in diverse biological systems and models is a major focus. In addition, disease-causing protease mutations, proteolytic mechanisms in acquired diseases, protease regulatory mechanisms at all levels, structural biology, developmental biology, genetics, and therapeutics will constitute the broad scope of the meeting.

Speakers will summarize the most exciting recent developments in their fields, identify the major unanswered questions, and share novel, unpublished data from their laboratories. The GRC on Matrix Metalloproteinases is an international forum that welcomes junior and established scientists from all disciplines to participate, and features scientists from both academia and industry. All applicants are encouraged to submit abstracts and a significant proportion of the oral program will be selected from submitted abstracts. Poster sessions as well as abundant time for social activities provide excellent opportunities for interactions. The informal nature of the conference promotes interdisciplinary interactions and stimulating discussions in a spirit of collegiality and collaboration.

The 2013 GRC on Matrix Metalloproteinases will again offer a Gordon Research Seminar (GRS, May 18-19) organized on the theme of the GRC, which will be held immediately preceding the GRC on Matrix Metalloproteinases. The GRS is a two-day program designed specifically for junior investigators, such as students, postdocs, and clinical trainees. The 2011 GRS was very successful and the attendees had expressed strong enthusiasm for this activity to continue. The Chair and Vice-chair of the 2013 GRS are Sean Gill (Univ of Washington) and Alisha Mendonsa (Vanderbilt University). The GRS offers unparalleled opportunities for peer-peer interaction, oral and poster presentations, and other activities focused on the needs of junior investigators. It is hoped that all GRS attendees will also participate in the GRC.

The Professional Development and Diversity Committee needs you!

The ASMB is looking for a committed society member to head our Professional Development and Diversity Committee. The chair of this committee is responsible for leading the society's efforts in advising, mentoring, and promoting the careers of junior investigators in matrix biology. The new chair will be involved in career development activities at the biennial meeting. As an example, the committee sponsored a Career Mentoring Breakfast at the 2010 ASMB meeting. This event was highly successful and will again be offered at the 2012 meeting. The new chair of this committee will be encouraged to devise activities to support young scientists throughout the year. If you are interested in matrix biology, the ASMB, and helping junior investigators, please contact Jen Holland or Jean Schwarzbauer at asmb@faseb.org.

News from the International Society for Matrix Biology

The purpose of the ISMB is to promote matrix biology research on a global scale. To do this, we don't hold standalone meetings as such, but prefer to contribute to major international meetings in matrix biology, such as the biennial ASMB meeting, to facilitate international exchange. Another major meeting is that of the Federation of European Connective Tissue Societies (FECTS), also held every two years, to take place this year in Poland (August 25-29, see www.fects.pl). ISMB provides direct financial support to these meetings and also international travel grants (available to members) to encourage the participation of young scientists. This year ISMB will be funding three such travel grants to attend the ASMB meeting and three travel grants to attend the FECTS meeting (see www.ismb.org for further details). We also provide awards, such as the Rupert Timpl award, presented at the FECTS meeting to a young scientist (working anywhere in the world) having made a major contribution to matrix biology research in the previous two years. Another award is the ISMB Distinguished Investigator award (again an international award), presented at the ASMB meeting, in recognition of lifetime achievements in matrix biology. Finally, we work closely with Elsevier in relation to the journal Matrix Biology.

The ISMB currently has about 300 members, from most parts of the world (USA, Canada, UK, Ireland, France, Germany, Netherlands, Belgium, Norway, Sweden, Finland, Switzerland, Hungary, Austria, Italy, Spain, Israel, China, Japan, Australia, New Zealand). Present members of ISMB Council are:

David Hulmes (President, Lyon, France)
Shireen Lamandé (Vice-President, Melbourne, Australia)
Peter Bruckner (Secretary/Treasurer, Münster, Germany)
Attila Aszódi (Munich, Germany)
Hans Peter Bächinger (Portland, USA)
David Birk (Tampa, USA)
Barbara Brodsky (Boston, USA)
Reinhard Fässler (Martinsried, Germany)
Billy Hudson (Nashville, USA)
Karl Kadler (Manchester, UK)
Johanna Myllyharju (Oulu, Finland)
Francesco Ramirez (New York, USA)
Sylvie Ricard-Blum (Lyon, France)
Liliana Schaefer (Frankfurt, Germany)
Sarah Wickström (Cologne, Germany)
John Whitelock (Sydney, Australia)

Funding for ISMB activities comes almost entirely from the membership. New members are always welcome, as well as industrial sponsors. See www.ismb.org for more information.

JOB OPENING

Postdoctoral Position at Johns Hopkins to Study Innate Immunity

An opening for post-doctoral position is available immediately in the lab of Dr. Shukti Chakravarti, Departments of Medicine, Cell Biology and Ophthalmology. The project would be to investigate regulation of innate immunity and inflammation by extracellular matrix (ECM) proteins. Previous work from this laboratory identified lumican, an ECM protein to regulate toll-like receptor mediated innate immune signals and host pathogen interactions. The candidate must be highly motivated, within 3 years of completion of a Ph.D in the areas of immunology, cell biology or biochemistry, experienced in mouse models and primary cell culture. Our laboratory is part of an exciting multidisciplinary and collaborative group of researchers with interests in corneal diseases like keratoconus, corneal infections and immune response.

To apply please submit your CV and two names of references to Shukti Chakravarti, Ph. D.
Email: schakra1@jhmi.edu

Thank you to our sustaining members!

Tom Barker, *Georgia Institute of Technology*
Rolf Brekken *UT Southwestern*
Renato Iozzo, *Thomas Jefferson University*
Robert Mecham, *Washington University*
William Parks, *University of Washington*
Barbara Smith, *Boston University*