NOVEMBER 2006

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ASBMB Today

Constituent Society of FASEB

AMERICAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY

Cells in Space

See page 18.

2007 ASBMB Annual Meeting

April 28 – May 2, 2007 • Washington, DC

Held in conjunction with EB 2007

Organized by: Benjamin F. Cravatt, The Scripps Research Institute, Michael K. Rosen, University of Texas Southwestern Medical Center and the 2007 ASBMB Program Planning Committee

Travel Award Application Deadline: November 30, 2006

Preliminary Program

Genome Dynamics

From Genome to Epigenome – Modification and Repair

- *Methylating and De-methylating DNA*
- *Recombining and Modifying DNA*
- *Making and Re-making DNA*
- *Telomeres and Telomerase*

The Chromosome Cycle

- *Centromeres and Kinetochores*
- *Chromatin Structure and Remodeling*
- *Chromosome Duplication and Cohesion*
- *Chromosome Segregation and Aneuploidy*

RNA

- *Molecular Recognition and Enzymology of RNA*
- *RNA-Based Gene Regulation*
- *Small RNAs*
- *RNA Modification: Mechanism and Function*

Protein Synthesis, Folding and Turnover

- *Molecular Mechanisms of Protein Biosynthesis*
- *Co- and Post-Translational Folding*
- *Protein Modification and Turnover*
- *Ribosome and Translation*

Structure and Design

Macromolecular Structure and Dynamics

- *Conformational Transitions and Protein Aggregation*
- *Experimental and Computational Dynamics*
- *Protein-Lipid Interface*
- *Structural and Mechanistic Evolution*

Enzymes – Mechanism and Design

- *Structural Enzymology*
- *The Role of Dynamics in Enzyme Catalysis*
- *Computational Studies of Mechanistic and Dynamical Aspects of Enzyme Reactions*
- *Enzyme Design*

Extracellular Matrix at Multiple Biological Scales

- *Extracellular Matrix at the Cellular Scale*
- *Extracellular Matrix at the Molecular Scale*
- *Extracellular Matrix at the Organism Scale*
- *Extracellular Matrix at the Tissue Scale*

Chemical Biology

- *Chemical Biology of Cell Death*
- *Fragment Based Drug Discovery*
- *Chemistry and Cell Biology of Natural Products*
- *Antibiotics for the 21st Century*

Cell Systems

Metabolism

- *Metabolic Sensing and Signaling*
- *Molecular and Cellular Aspects of Metabolic Disease*
- *Mitochondria in Health and Disease*
- *Aging and Metabolism*

Organelle Dynamics

- *Golgi Structure and Biogenesis*
- *Membrane Biogenesis*
- *Mitochondrial Dynamics*
- *Nuclear Dynamics*

Systems Biology

- *Modeling of Cell Systems*
- *Molecular Profiling of Cell Systems*
- *Proteomics of Cell Systems*
- *Mathematical Biology*

Minority Affairs Committee Sponsored Symposia

- *Best Practices in Program Assessment*
- *Infectious Diseases in Minority Populations – Hepatitis C*
- *Genetic Diseases in Minority Populations – Sickle Cell Anemia*
- *Infectious Diseases in Minority Populations – Tuberculosis*

Signaling

Biochemistry and Signaling of Lipids

- *Biogenesis, Transport and Compartmentalization of Lipids*
- *Chemical Probes of Lipid Systems*
- *Lipids as Transcriptional Regulators*
- *Specific Protein-Lipid Interactions*

Signaling Pathways Controlling Cell Structure and Fate

- *Cytokine and Growth Factor Signaling*
- *DNA Damage Signaling*
- *Cell Cycle*
- *Signaling to the Cytoskeleton*

Public Affairs Advisory Committee Sponsored Symposium

Sponsored by EB participating societies

- *NIH at the Crossroads: How Diminished Funds Will Impact Biomedical Research and what Scientists Can Do About it*

Education and Professional Development Committee Sponsored Symposia

- *Classroom of the Future II*
- *Science at Undergraduate Institutions*
- *Graduate Student/Postdoctoral Starting Faculty Transitions*
- *Preparing for a Successful Career in Industry*

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ASBMB Today

AMERICAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY

NOVEMBER 2006
Volume 5, Issue 8

features

ON THE COVER:

18 A Soyuz launch similar to the one used to send T-cells to the international space station.

Credits: ESA/Starsem.

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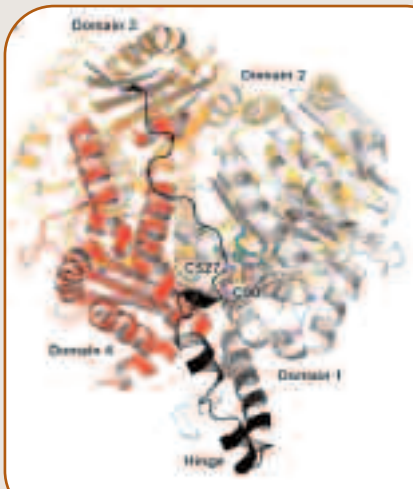

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Update on Peer Review

We hear so much about the cost of health care spiraling out of control, and one important part of dealing with this problem is biomedical research. Many Americans, while supporting biomedical research, do not always understand that a stagnant NIH budget means that much important research does not get done, research that would likely lead to breakthroughs in combating disease and developing new treatments. Making sure people in your communities understand this connection is one of the reasons I have urged repeatedly in this space in recent months for you to promote the benefits of NIH science locally, especially to your Member of Congress and Senators.

The impact on the peer review system of this difficult funding environment is dramatic. According to the CSR, since 2002, the number of total applications submitted to the NIH increased from 55,000 to almost 80,000 in 2005, and the number of research project grant (RPG) applications increased from 30,000 to 45,000. Likewise, in 2002, investigators applying for RPGs wrote an average of 1.2 grant applications each, but in 2005 RPG applicants were writing 1.4 grants each. This is both a result of, and a contributing factor to, plummeting success rates, and in turn, necessitates ever more people willing to review grants. According to CSR, the number of reviewers has increased from 8,000 in 1999 to 18,000 in 2005.

ASBMB has been active in helping to address this problem by asking you, our members, to serve on study section if asked, even if you have already served. More than 700 of you agreed to do so, and this has been appreciated by Center for Scientific Review (CSR) Director Toni Scarpa, and Dr. Donald Schneider, the Director of the Division of Molecular and Cellular Mechanisms, the Division that houses the Biological Chemistry and Macromolecular Biophysics (BCMB) Integrated Review Group, which reviews biochemistry and some molecular biology grants.

We have been interacting with Drs Scarpa and Schneider on a number of issues of interest to ASBMB members. Because of the breadth of the current study sections, there is a concern that proposals are not receiving adequate peer review, and we have asked the CSR to increase the number of study sections in biochemistry. Several new study sections have been approved by the Peer Review Advisory Committee, and will be constituted in time for the June, 2007 round of reviews (Feb. and Mar. application deadlines). We are delighted that the CSR has been responsive to our recommendations for more study sections. This is clearly a step in the right direction, and we hope that those of you who will be

Tell Us What You Think

We appreciate receiving letters that are suitable for publication regarding issues of importance or commenting on articles appearing in *ASBMB Today*. Letters should be sent to the editor, John Thompson, at the address found at left. Letters must be signed and must contain the writer's address and telephone number. The editor reserves the right to edit all letters.

Dr. Heidi E. Hamm
ASBMB President

asked to serve on these new study sections will do so!

A central recommendation of the CSR reorganization task force headed by then National Academy of Science President Bruce Alberts was constant oversight of the review groups in each IRG by leading scientists in the field. At the time, Dr. Alberts described this as a "great once-in-a-lifetime opportunity to create a system that won't be just locked in place, but can continually be evaluated by outside experts—and in which modern science, which is changing so rapidly, can really be adequately be supported and tracked." Currently, however, the IRGs are reviewed only once per 5 years. Especially with a dramatically reorganized system, oversight is necessary often at the beginning to ensure the system is functioning as planned. We fear that on a 5-year oversight schedule, several more years will pass until there is a comprehensive review of the reorganization in these areas. Every two years, there is a CSR internal review of each IRG, and BCMB was the first one that was scheduled for this year. We appreciate this scrutiny, but we continue to push for more ongoing review from the scientific community as well. We all have a stake in making peer review work the best that it can, even in times of tremendous overload and tumbling success rates.

A number of other positive changes are occurring in the CSR. A wonderful new program this year is the shortened review cycle with "pink sheet" coming very quickly after the study section has met, so that investigators can resubmit within 4 rather than 8 months. Already 140 investigators have taken advantage of this accelerated review process. A number of electronic enhancements of the review process are being instituted, including telephone- and video-enhanced discussions, and asynchronous electronic discussions. In addition, there is an enforced rate of triage of applications so that study section meetings don't last as long. The goal of these changes is to make the peer review process less onerous, and thus increase the participation of the community.

You have all heard that applications will be electronic starting in Feb, 2007. One dramatic change in the works related to this is a shorter application. Again, the goal is to increase the number of reviewers of each grant, and thereby increase the fairness of the process and bring deeper expertise to the review.

Also up for discussion in the near term are novel mechanisms to identify significant innovative and high-impact research.

Certainly, when the NIH budget is flat (or falling, when biomedical inflation is taken into consideration) and success rates are low, there are extraordinary stresses on all aspects of the system. No matter how good peer review is, it cannot work adequately when success rates for first RPG applications are as low as 9% (in 2005 according to published data, with an aggregate success rate of 22% for RPGs in 2005). Though we appreciate all the changes being made at CSR to introduce improvements in the process, we also need at least modest increases in the NIH budget to capitalize on the incredible opportunities to bring breakthroughs in biomedical research to bear on the unsolved health problems of the nation. Thus, we need to make the case to Congress of the importance of biomedical research to our economy and our future. The crisis we are facing will not be solved until this case is made.

Renew Your Membership Online

ASBMB 2007 dues renewal notices have been mailed to all members. You can now make payment online at the ASBMB website: www.asbmb.org, by clicking on "membership" and then "renew your dues now."

Your membership includes a free subscription to our monthly magazine, *ASBMB Today,* plus free subscriptions to *JBC Online* and *MCP Online*. You also receive special member rates for *The Journal of Lipid Research* and *Trends in Biochemical Sciences,* as well as the print versions of *JBC* and *MCP.*

ASBMB members may also register for the Annual Meeting at discounted rates. In addition, you can order your 2007 edition of the *Annual Review of Biochemistry* through ASBMB.

If you have any questions, please email membership@asbmb.org.

by Peter Farnham, CAE, ASBMB Public Affairs Officer

109th Congress Wraps Up (Almost),

Members of Congress left Washington in late September to campaign for reelection with a number of crucial bills still on their "to do" list, virtually guaranteeing a lame duck session in November. Only two appropriations bills (Homeland Security and Defense) have passed the Congress; the National Institutes of Health and National Science Foundation have not been given a budget; and although a quite laudable NIH reauthorization bill has passed the House, its prospects in the Senate this year are dim at best.

What is worse, it is unclear that any progress will be made on any of these bills after the election. The House and Senate are both "in play," with the Democratic party making its strongest bid since 1994 to regain control of at least one if not both bodies. A lame duck session with control of one or both houses due to change in January 2007 would likely be even less productive than such sessions usually are.

Appropriations

Ten spending bills are awaiting action when Congress returns, and the Senate leadership wants to wrap these up in a week-long session ending about November 17, when the current continuing resolution expires (a CR is a temporary measure funding the government until the regular appropriations bills can be passed). Few observers are optimistic that this highly ambitious schedule is achievable; most expect the session to last until after Thanksgiving.

The top priority among the House leadership is avoiding an omnibus spending bill that rolls all unpassed bills into one huge measure. The Senate is less concerned about this, however, and given time constraints and differences between the House and Senate versions of the appropriations bills that have passed, it may be the only viable alternative. A long term CR is another possibility, but this would maintain all spending at this year's level (and eliminate earmarks), and if Democrats take control of one or both houses this election, it would give them an opportunity to rewrite the bills in January.

NIH Budget Continues to Languish

NIH, coming off a year when it received its first actual cut in a generation, is not expected to do much better this year, thus continuing the erosion of the massive infusion of funds it received during the 5-year doubling campaign that ended in 2003. NIH has lost about 10% of its 2003 purchasing power due to inflation, and even under the best circumstances it is hard to imagine that this will be made up in the near future.

The House version of the NIH bill flat-funds the agency at the President's request level, approximately $28.25 billion at the program level (after various taps, etc.). The Senate bill includes about $220 million above the President's request and comes in at about $28.45 billion. This is only a 0.78% increase and doesn't begin to meet biomedical inflation, projected to be about 3.5% this year.

There are efforts underway in both houses to increase the NIH appropriation. Rep. Mike Castle (R-DE) has organized a group of about two dozen centrist Republicans, and in a September 27 letter to Majority Leader John Boehner (R-OH) asked him to ensure that the House version of the Labor/HHS bill had a total of $7 billion more than the President's request for the bill. This follows through on an effort begun last spring when Castle succeeded in getting an additional $4 billion into the bill. He is now asking that the remaining $3 billion be provided. Although no additional money is guaranteed for NIH under this agreement, if $3 billion is provided it will make it easier for appropriators to provide NIH with a little additional money.

In the Senate, our long-time allies Arlen Specter (R-PA) and Tom Harkin (D-IA) have circulated a "dear colleague" letter urging the Senate to support a level of funding for the L/HHS bill that is "no less" than the 2005 level, in effect asking for an additional $2 billion to be provided. Like the House effort, this does not guarantee additional money for NIH. However, if the Senate goes along, it makes it possible to provide additional money during final negotiations.

Lame Duck Session a Certainty

Our long-time allies in the Senate, Arlen Specter (at left) and Tom Harkin have circulated a "dear colleague" letter urging the Senate to support a level of funding for the L/HHS bill that is "no less" than the 2005 level.

Regardless of all this, however, NIH at best can expect something approximating flat funding this year, which means another year in which inflation continues to take its toll on NIH purchasing power.

NSF a Rare Bright Spot

The National Science Foundation is one of the rare bright spots in science funding this year, with the President having asked for about an 8% increase to around $6 billion. NSF is a key agency in the President's American Competitiveness Initiative, a package of legislative proposals aimed at shoring up the nation's innovative, technological, and educational enterprises. Both the House and Senate have approved this level of funding, although an overall appropriations bill has not been approved yet.

NIH Reauthorization Bill

Every federal agency is supposed to be reauthorized periodically, but this is a requirement honored more in the breach than in actuality, especially in recent years. However, Rep. Joe Barton (R-TX), chairman of the powerful Energy and Commerce Committee, has undertaken the task of trying to reauthorize every agency under the committee's jurisdiction, and the first agency he has addressed is the NIH, last reauthorized in the early 1990s.

A draft reauthorization bill was circulated in the summer of 2005 that provoked considerable outcry from the biomedical community. However, over the past year, Barton's staff has worked very closely with FASEB and other components of the biomedical community, and finally, in late September, a bill was passed by the House that FASEB, the Association of American Universities, and the Association of American Medical Colleges all endorsed.

The bill's major elements include an authorization of 5% increases at NIH in each of the next three years (a level for which Barton will fight). In addition, a "common fund" is established that would grow to 5% of the NIH budget and then be maintained at that level. The common fund would be used to support trans-NIH initiatives, and most of it will be dispensed in the form of individual investigator grants. Under the earlier draft, the common fund would have grown to about 15% of the NIH budget, and there was no language directing NIH to spend it on individual grants.

The bill also provides for a "Scientific Management Review Group" to review and recommend any changes in the NIH's structure. The process also calls for public input into any plans to restructure the agency. Finally, an agency-wide reporting system is created that will increase transparency for researchers and other members of the public, hopefully spurring research.

Barton's staff, and Barton himself, were remarkably open to negotiations during discussions of various drafts of the bill in the months preceding the bill's introduction, and the bill represents a solid consensus between congress and the community on how best to manage NIH. The bill passed the House in late September on a near unanimous vote.

No comparable bill has been introduced in the Senate; thus the bill will probably die at the end of the coming lame duck session because it is unlikely that the Senate will take up such a bill given the amount of time it will have. However, the bill will clearly be on the fast track in the House next year regardless of which party has control, and prospects are good in 2007 for NIH to be reauthorized for the first time since the early 1990s.

FASEB Supports Animal Enterprise Terrorism Act

"It is time to protect our researchers, their institutions and those who do business with them from the dangerous tactics of (animal rights) extremists," declared Dr. Leo Furcht, FASEB President in a September 20 letter addressed to the Senate Judiciary committee. The letter was in support of S.3880, the Animal Enterprise Terrorism Act (AETA), which increased protections for animal research facilities and their employees targeted by animal rights (AR) extremists. On September 30th, the last day of their Congressional session before November's election, the Senate passed S. 3880 by unanimous consent. This represents a tremendous victory for the animal research community.

FASEB has long supported the AETA and in fact helped pass its predecessor, the Animal Enterprise Protection Act, which was signed into law in August, 1992. Unfortunately, this law proved inadequate: while it covered physical property damage to animal facilities, it did not cover disruption caused by threats or harassment campaigns launched against employees and their families. AR extremists have become infamous for causing economic damage by attacking not just the animal facility itself, but all associated businesses (i.e. – accountants, banks, insurance brokers), as well. The AETA would extend protections to these secondary or tertiary targets, increase the penalties for attacking animal enterprises, and provide law enforcement with the statutory tools needed to prosecute coordinated harassment campaigns or threats made across state lines. Moreover, the bill provided enhanced protection against the most insidious tactic used by AR extremists: personal campaigns launched against researchers, employees and their families, including death threats, continual home and neighborhood harassment, vandalism, publishing of personal information on the internet, and even arson or bombings. Although many of these acts are minor crimes, few are more than misdemeanors, and current law would not allow prosecutors to package together all of the parts of a continuous campaign, particularly if it crossed state lines.

When the AETA was originally introduced, by Senator James Inhofe (R-OK) and Representative Thomas Petri (R-WI), it seemed unlikely to move forward. Although there were a series of high profile hearings in which AR activists Jerry Vlasak loudly advocated the murder of researchers to end the use of laboratory animals, there was little bipartisan support for the bill (known as S.1926 in the Senate at that time). Democratic members of Congress, while appalled by Vlasak himself, were worried about potential infringement on civil liberties, and remained unconvinced that AR extremism was a real threat, despite testimony by the Federal Bureau of Investigation (FBI) that the number and magnitude of cases was rapidly increasing. FASEB joined forces with the National Association for Biomedical Research (NABR) as part of the Animal Enterprise Protection Coalition. Through letters and meetings, the groups sought to convince Congress of the necessity of these protections for biomedical research and those researchers who used animal models.

Then, two major breakthroughs occurred. One of the most controversial provisions of the bill would allow FBI and other law enforcement agencies to use wiretapping as a tool to find and prosecute AR extremists. This provision was removed from the AETA and folded into the renewal of the PATRIOT act in March, 2006, eliminating much of the objection to the AETA itself. Then, in early September, Dr. Dario Ringach, a neurobiologist at UCLA made headlines when he conceded to AR extremists after a long harassment campaign against himself and his family, saying that he would cease using primates in his experiments. This was after AR extremists aimed a Molotov cocktail at a fellow UCLA primate research (the group mistakenly left the bomb on the doorstep of the researcher's elderly neighbor). Although the lethal device malfunctioned and fortunately no one was injured, the apparent willingness of AR extremists to indiscriminately cause harm was enough to cause Dr. Ringach to send an email to the group saying simply, "You win." This was enough to gain a revised AETA (now S.3880) a Democratic cosponsor in California Senator Dianne Feinstein, critical to its movement out of the Senate Judiciary and on to passage by the Senate.

The House version of the AETA remains stalled in the House Judiciary committee. However, FASEB and NABR are hopeful it will move forward in the lame duck session of Congress following the election. It is likely that the bill, should it leave the committee, would be passed by the full House and signed into law by President Bush. If that does not happen, passing the AETA again in the new Congress may present new challenges.

Carrie D. Wolinetz, Ph.D.
FASEB Office of Public Affairs

Carol W. Greider Shares Lasker Award for Basic Medical Research

ASBMB member Carol W. Greider, along with Elizabeth H. Blackburn and Jack W. Szostak, has been chosen to receive the 2006 Albert Lasker Award for Basic Medical Research. The award honors them for the prediction and discovery of telomerase, the RNA-containing enzyme that synthesizes the ends of chromosomes, protecting them and maintaining the integrity of the genome. In so doing, the scientists unearthed a biochemical reaction that guards cells against chromosome loss and identified the molecular machinery that performs this feat. The work resolved perplexing observations about chromosome termini and explained how cells copy their DNA extremities.

Dr. Carol Greider

In the 1930s, scientists surmised that telomeres ensure the propagation of chromosomes during cell division and prevent them from inappropriately melding with one another. The physical nature of these structures and how they are constructed eluded researchers until Blackburn, Greider, and Szostak performed their groundbreaking investigations in the late 1970s and 1980s. Blackburn showed that simple repeated DNA sequences comprise chromosome ends and, with Szostak, established that these repeats stabilize chromosomes inside cells. Szostak and Blackburn predicted the existence of an enzyme that would add the sequences to chromosome termini.

Greider and Blackburn then tracked down this enzyme—telomerase—and determined that each organism's telomerase contains an RNA component that serves as a template for the creature's particular telomere DNA repeat sequence. Szostak found that budding yeast unable to perform the telomerase reaction lose their telomeres—and chromosomes—over multiple generations. Eventually, the organisms stop dividing. In addition to providing insight into how chromosome ends are maintained, Blackburn, Greider, and Szostak's work laid the foundation for studies that have linked telomerase and telomeres to human cancer and age-related conditions.

Given by the Albert and Mary Lasker Foundation for "outstanding contributions in basic and clinical medical research," the Lasker was first awarded in 1945. It has since become known as "America's Nobel" and is considered by many as the nation's most prestigious honor for basic and clinical medical research, primarily because of the extremely rigorous process of nomination and selection conducted by a jury of the world's top scientists. Seventy-one Lasker winners have gone on to win the Nobel Prize.

ASBMB 2007 Theme Meeting: Organelle Dynamics

Organizers: Matthew Shair, Daniel Kahne, Jodi Nunnari, and Yixian Zheng

The biochemistry and molecular biology of organelles, especially with respect to their dynamic properties, will be the focus of the Organelle Dynamics Theme in Washington at the 2007 ASBMB meeting. The plenary speakers in each of the sessions will discuss their studies on membrane biogenesis, mitochondria dynamics, Golgi biogenesis and organization, and dynamics of the nucleus. Each of the four plenary sessions will be accompanied by a Young Investigator Award to encourage post-doctoral fellow and graduate student participation. Lectures will focus on advances in understanding how these organelles are constructed, organized, maintained, and function given their dynamics. In some cases, these lectures will emphasize the use of chemistry to study organelle dynamics.

The Organelle Dynamics Theme is organized in four sessions:

Membrane Biogenesis (Chair: Daniel Kahne, Harvard University)

Mitochondrial Dynamics (Chair: Jodi Nunnari, University of California, Davis)

Golgi Structure and Biogenesis (Chair: Matthew Shair, Harvard University)

Nuclear Dynamics (Chair: Yixian Zheng, Caernegie Institution)

The Membrane Biogenesis session will feature lectures on mechanisms of constructing membranes, with an emphasis on chemical aspects of membrane assembly. Daniel Kahne will discuss his group's efforts to understand membrane biogenesis from a chemical perspective and using chemical probes to study this problem. Natividad Ruiz from Princeton University will cover biogenesis of the outer membrane of *E. coli*. Hajime Tokuda from the University of Tokyo will discuss his efforts to understand how lipoproteins are sorted at the outer membrane of Gram-negative bacteria.

Dr. Matthew Shair

The session entitled Mitochondrial Dynamics features plenary lectures by Richard Youle of the NINDS, David Chan from Caltech, and Jodi Nunnari of the University of California, Davis. This symposium will cover the latest research to study mitochondrial dynamics, especially mitochondria biogenesis. Richard Youle will discuss the role of Bcl-2 family proteins in mitochondrial morphogenesis. David Chan will deliver a lecture on the dynamics of mitochondria in neurodegeneration, and Jodi Nunnari will report on molecular machines responsible for mitochondrial division and fusion.

A session entitled Golgi Structure and Biogenesis will focus on the latest research probing how the Golgi assembles and how it maintains its structure (and function). Jennifer Lippincott-Schwartz from NIH will report on Golgi secretory transport by rapid partitioning within a continuous two-phase membrane system. Graham Warren of the Yale School of Medicine will present a lecture on the biogenesis of the Golgi apparatus in a protozoan parasite, and Matthew Shair from Harvard University will deliver a lecture on the discovery and use of a new small molecule (named dispergo) that causes reversible separation of Golgi cisternae into small particles.

A fourth session in Organelle Dynamics focuses on the dynamics of the nucleus. This session features cutting-edge lectures by Katherine Ullman (Huntsman Cancer Institute), Ueli Aebi (University of Basel), and Yixian Zheng (Carnegie Institution). Katherine Ullman will report on her group's work on studying nuclear remodeling during cell division. Ueli Aebi will deliver a lecture on the role of nuclear actin in nuclear dynamics, what form it is in, and what it is used for. Finally, Yixina Zheng will discuss morphogenesis of the mitotic spindle and its connection with the interphase nucleus.

These sessions provide insight into some of the latest findings on the biochemistry, molecular biology, and chemistry of organelles, especially as they relate to their dynamic properties. The organizers encourage graduate students and post-doctoral fellows and junior faculty to submit posters and compete for the four Young Investigator Awards associated with the Organelle Dynamics Theme for 2007. We look forward to seeing you at the meeting!

Sarah Elgin to Receive ASBMB Award For Exemplary Contributions to Education

Dr. Sarah Elgin, Professor of Biochemistry and Molecular Biophysics and Professor of Education at Washington University (WU) in St. Louis, will receive the 2007 ASBMB Award for Exemplary Contributions to Education.

As a scientist, Elgin has made numerous important contributions to the study of the roles of chromatin structure and genome organization in gene regulation resulting in more than 160 research papers and other publications. She was also elected as a Fellow of AAAS (1998) and Chair of the Gordon Conference on Chromatin Structure and Function (1996). In addition, Elgin has served on national review panels and several editorial boards.

Elgin has successfully combined an active and productive research career with a commitment and passion for education both at the undergraduate and precollege levels. She has received numerous honors and awards and regularly teaches undergraduate and beginning graduate courses, including chromatin structure and gene expression, molecular genetics, and upper level laboratory courses in genetics. Throughout her career, she has consistently involved undergraduates, high school teachers and students, predocs, postdocs, and visiting fellows in her research and served as a mentor to them.

The following quote from a former student of Elgin's attests to the strong influence she has had in encouraging students, particularly young women, to succeed in science:

"I believe that the best way to learn is by example, and Dr. Elgin is the best example I know of a teacher, a scientist, a mentor, and a role model for women in science. I love science and would like to stay in academia where I can both do research and teach. I sincerely believe that I wouldn't be where I am today with the goals I have without the influence of Dr. Elgin."

Elgin has also been an active proponent of science education at the K-12 level. In the late 1980s and early 1990s she initiated a new science education partnership between WU and a predominately minority school district in St. Louis. The program provides professional development opportunities for teachers while supplying materials and equipment that help teachers to do hands-on investigations with their students. She continues to be deeply involved in teaching courses and running workshops in genetics aimed at both K-8 teachers and high school teachers.

With her impressive vision and boundless energy, Elgin has emerged as a national leader in undergraduate science education. She was one of the two founding editors of CBE Life Sciences, the on-line education journal of the American Society for Cell Biology. She has been a member of the steering committee of the Coalition for Education in the Life Sciences, a national coalition of professional societies in the biological sciences that have joined together in an effort to improve undergraduate education in the life sciences.

From 1992 through 2004, Elgin served as the director of four HHMI undergraduate science education grants at WU. Thanks to her efforts, her institution was one of the most successful in this highly competitive national program. Her program supported undergraduate research, curriculum development, and K-12 outreach, involving faculty from the Division of Biology and Biomedical Sciences, Chemistry, Physics, Mathematics, and Education. Under her leadership, the HHMI grants have supported 586 undergraduate researchers, many of whom have been co-authors in more than 120 papers in major journals. In addition, the HHMI grants she has directed have provided science education opportunities for some 1,100 precollege teachers and 13,000 students.

Dr. Sarah Elgin and student

In 2002 Elgin was one of only 20 scientists nationally to be named an HHMI Professor in our inaugural competition for this program. She used the award to broaden the undergraduate curriculum by bringing the tools and thinking of genomics into the classroom and to develop appropriate ways to introduce genomics into the secondary school biology curriculum.

Hunter, Pawson to Receive 2007 Herbert Tabor/

Dr. Tony Hunter and Dr. Tony Pawson have been selected to share the 2007 Herbert Tabor/*Journal of Biological Chemistry* Lectureship Award. The award will be conferred at the 2007 ASBMB annual meeting held in conjunction with Experimental Biology, April 28–May 2 in Washington, DC, and the Herbert Tabor/JBC Lecture will open the meeting on Saturday, April 28, 2007, at 6 p.m. The Lectureship was established in recognition of the many contributions of Herbert Tabor to the *Journal of Biological Chemistry* and the Society. The award honors the recipients with a cash prize as well as paid travel and expenses to present a lecture at the 2007 meeting. Past recipients of the award were Dr. Robert J. Lefkowitz in 2004, Dr. Michael S. Brown and Dr. Joseph L. Goldstein in 2005, and Dr. Charles C. Richardson in 2006.

Dr. Tony Hunter

Dr. Tony Pawson

About Tony Hunter

Hunter, who was born in Ashford, Kent, England, attended Caius College at the University of Cambridge, receiving his B.A. in 1965. Subsequently, he conducted graduate studies in the Department of Biochemistry at the University of Cambridge, receiving his Ph.D. in 1969 for work on mammalian protein synthesis. In 1968 he was appointed a Research Fellow of Christ's College at the University of Cambridge and worked for three years in the Department of Biochemistry doing independent research on the mechanism of initiation of protein synthesis in eukaryotes, discovering that this requires a specialized methionyl tRNA. In 1971 he joined the Salk Institute in La Jolla, California, as a Research Associate working under Walter Eckhart on polyoma virus DNA replication.

Hunter spent 1973 to 1975 back at the Cambridge Department of Biochemistry where he discovered how tobacco mosaic virus expresses its coat protein using a subgenomic RNA before rejoining the Salk Institute as an Assistant Professor in 1975. There he set out to identify tumor virus transforming gene products, starting with the tumor (T) antigens of polyoma virus and then turning his attention to Rous sarcoma virus (RSV). In 1979, in the course of studying the polyoma virus middle T antigen and the RSV v-Src protein, he discovered that middle T and v-Src both exhibit a previously unknown protein kinase activity that phosphorylates tyrosine. He went on to show that normal cells have tyrosine kinases, including c-Src and the EGF receptor, and that tyrosine phosphorylation is important in signal transduction processes that control many aspects of normal cell growth as well as in oncogenesis.

These findings led to the development of tyrosine kinase inhibitors as drugs for the treatment of certain types of cancer. Hunter has spent most of the last 30 years studying protein kinases and phosphatases and the role of protein phosphorylation in cell growth, oncogenesis, and the cell cycle.

His current interests are mechanisms of signal transduction through protein phosphorylation, ubiquitination and sumoylation events that are involved in cell proliferation and growth control, and in cell cycle checkpoint activation in response to DNA damage, using cultured mammalian cells, mice, Xenopus egg extracts, *C. elegans* and budding and fission yeast as experimental systems. His recent work has highlighted the importance of crosstalk and feedback loops in the PI-3 kinase-Akt-mTOR cell growth pathway, has elucidated mechanisms of activation of the ATM protein kinase in response to double strand DNA breaks, and has revealed a critical role for apical aPKC localization in asymmetric division of neuronal precursors in the spinal cord.

Hunter has received many awards for his work on tyrosine phosphorylation, including the Katharine Berkan Judd Award, General Motors Cancer Research Foundation Mott Prize, Gairdner Foundation International Award, Biochemical Society Hopkins Medal, Keio Medical Science Prize, City of Medicine Award, American Cancer Society Medal of Honor, Landon-AACR Prize for Cancer Research, and the Wolf Prize in Medicine. He is a Fellow of the Royal Society of London, an Associate Member of the European Molecular Biology Organization, a Fellow of the American Academy of Arts and Sciences, and a Member of the National Academy of Sciences, Institute of Medicine, and American Philosophical Society.

Journal of Biological Chemistry Lectureship Award

He is currently an American Cancer Society Research Professor, Professor and Director of the Molecular and Cell Biology Laboratory at the Salk Institute for Biological Studies, and Adjunct Professor in the Division of Biological Sciences at the University of California, San Diego.

About Tony Pawson

World-renowned signal-transduction researcher Pawson developed an interest in biochemistry with the help of his high school teacher, Michael Baron, who shared his passion for the biochemical mechanism of cellular metabolism with his students. Pawson became fascinated by the notion that one could understand how complex organisms work at a cellular level and went on to study biochemistry at Cambridge. There, he was fortunate enough to study under the guidance of Tim Hunt, then working on protein synthesis. Hunt would eventually go on to win the Nobel Prize in 2001.

At Hunt's suggestion, Pawson went to work on his Ph.D. at the Imperial Cancer Research Fund in London where he met other outstanding scientists working on establishing the identity of a gene that can cause a normal cell to become cancerous. That gene was called the v-src retroviral oncogene. Pawson decided to specialize in the genetics of oncogenes that create oncoproteins, big complicated molecules that transmit biochemical signals to affect virtually every aspect of cellular behavior, thereby developing cancer.

Pawson did his post-doctoral research at the University of California at Berkeley from 1976 to 1980 where he became interested in tyrosine kinase, which at the time was very poorly understood.

At a cellular level communication controls our very essence

In 1981, Pawson accepted a position as an Assistant Professor at the University of British Columbia and moved to Vancouver. Once established in his own lab, he worked in close collaboration with other scientists. Together, they tried to identify the regions of the protein that are essential for its transforming activity. Later, they found that the tyrosine kinase domain was critical for its cancerous properties.

Eventually, in 1985 Pawson moved to Toronto to join the Samuel Lunenfeld Research Institute at Mount Sinai Hospital. Since then, Pawson and his lab associates have understood many aspects of signal transduction—how signals are conveyed from receptors at the cell membrane to their targets within the cell. However, the entire picture of this complex mechanism is yet to be discovered.

The Science

Communication is one of the activities responsible for our success in every sphere of our lives: work, school, sport, relationships. At a cellular level communication controls our very essence—our lives. In molecular biology the basic process of communicating involves getting signals from outside of a cell to inside, and it is called signal transduction.

Any external physical or chemical signal, such as temperature change, electricity and light waves, or signals from hormones or neurotransmitters, stimulates a specific response and causes biochemical reactions within the cell. In a healthy organism, cells function in an orderly manner. But what happens if the cell does not respond or receives an unusual signal? If this happens the organism may develop diseases.

For example, bad intracellular communication reduces immunity to various disorders. Cancer is the classic example of cells responding to an aberrant signal that causes them to grow in an uncontrolled fashion. The process can begin with a genetic mutation that affects the creation of signal transduction proteins that tell cells when to divide.

Pawson has spent 25 years studying how cells grow and communicate with each other. He discovered which specific protein interactions control signal transduction. Further, he recognized the importance of tyrosine kinases, which are responsible for transmitting the commands to hormones that regulate cellular reproduction and metabolism. These discoveries allowed the development of new drugs that block the action of tyrosine kinases and thus halt the proliferation of some types of cancer cells.

Pawson's discoveries contribute to every aspect of biomedical research including immunology and cancer research; for a decade, he has been one of the top 25 cited scientists in this field.

The Four Dimensions of Macromolecular Structure

Organizer: Joseph Noel, HHMI, The Salk Institute for Biological Sciences

The macromolecular structure and dynamics theme at ASBMB 2007 will explore our increasing appreciation for the role of time, defined across wide scales, in the emergence and execution of macromolecular activities in biological systems. Specifically, protein function derives not only from three-dimensional structure but also from structural changes over time. These time scales range from millions of years during which proteins evolve and acquire new functions to fleetingly fast stints during which proteins and protein complexes fold and assemble, catalyze chemical reactions, or parse biological information. This year's theme brings together an internationally recognized group of scientists to expand upon the discovery and contribution of conformational transitions and protein aggregation to normal and aberrant cellular function, the development and application of experimental and computational methods to investigate protein dynamics, the unique physiochemical environment of the lipid-protein interface, and the evolutionary underpinnings of protein structure and mechanism. Moreover, the macromolecular structure and dynamics theme synergizes with many other themes in 2007 that explore the relationship between biological structures and dynamics.

The conformational transitions and protein aggregation session chaired by Dr. Roland Riek (Salk Institute) will focus on the role of protein misfolding and amyloid formation in both normal cellular function and diseases such as Alzheimer and Parkinsons. Riek will discuss his structural studies of amyloid fibrils associated with neurodegenerative diseases and prions. Dr. Jeff Kelly (Scripps Research Institute) will explore the chemical biological aspects of amyloids, and Dr. Beat Meier (ETH Zurich) will discuss the application of solid-state methods to unraveling the structural features of protein aggregates.

The session on experimental and computational dynamics chaired by Dr. Lewis Kay (University of Toronto) will take a journey into the "excited" states of proteins. Although a three-dimensional static structure provides a description of the ground state of the molecule in question, macromolecular function is, in many cases, highly dependent on excursions to excited molecular states and hence intimately coupled to flexibility. In this session, studies of protein dynamics will be presented using a variety of different methodologies. Dr. Sunney Xie (Harvard University) will describe single molecule fluorescence experiments probing the dynamics of individual protein molecules that provide information about the distribution of molecular properties that cannot be obtained in ensemble-based approaches. Kay will provide illustrations of how solution-based NMR spectroscopy can be used to provide a view of excited states of proteins. Dr. J. Andrew McCammon (University of California, San Diego) will describe some of his recent molecular dynamics simulations that probe the relation between dynamics and function in complex biological systems.

The protein-lipid interface session chaired by Dr. Charles Sanders (Vanderbilt University) will center on the unique physical-chemical environment provided by biological membranes and how this mosaic milieu contributes to protein function. Dr. Susan Buchanan (NIDDK, NIH) will describe the structure of an energy-dependent small molecule (iron) transporter from *E. coli*, the so-called colicin I receptor (Cir), alone and in complex with colicin Ia, a large protein that misappropriates the transport system to cause cell death. Dr. John Bushweller (University of Virginia) will provide a composite structural and dynamic picture of the integral membrane enzyme DsbB, which serves as a key component of the system of proteins responsible for catalyzing disulfide bond formation in the periplasm of Gram (-) bacteria. Sanders will describe the architecture of a structurally unique integral membrane kinase solved by NMR, namely the 40-kDa prokaryotic diacylglycerol kinase.


Dr. Joseph Noel

The structural and mechanistic evolution session chaired by Dr. Joseph Noel (Salk Institute) will focus on recent successes in deciphering the principles impacting evolutionary change in proteins and protein networks in living systems. Dr. Antony Dean (University of Minnesota) will describe the biochemical architecture of an ancient adaptive landscape. Noel will discuss the utility of specialized metabolism in providing clues for the identification and manipulation of evolutionary determinants linking catalytic specificities in enzymes. Dr. Rama Ranganathan (University of Texas Southwestern Medical Center) will articulate how evolution guides the design of functional proteins.

These four sessions will provide a sampling of the role that time plays in biological systems particularly at the atomic level where the underlying chemistry of macromolecules is exploited for a myriad of functional outputs.

George William Schwert, 1919-2006

George William Schwert died on September 19, 2006 at Central Baptist Hospital. Schwert was born on January 27, 1919 in Denver, Colorado, son of the late George William and Agnes Buhler Schwert. He attended public schools in Denver and Minneapolis and graduated, summa cum laude, from Carleton College in 1940. He received his Ph.D. from the University of Minnesota in 1943.

Schwert worked for one year as a research biochemist at Sharp and Dohme, Inc. and then enrolled in the Naval Reserve where he served as a LTJG in the Communication Division and was a "plankowner" on the newly commissioned destroyer, USS Duncan, which operated in the Pacific Theater.

In 1945, he was appointed Instructor in the Department of Biochemistry in the Duke University School of Medicine and, in 1957, he was made Professor in that Department. In 1959, he became the founding Chairman of the Department of Biochemistry in the College of Medicine at the University of Kentucky. He served as Chairman of the department until 1974, and as Professor until he retired in 1985.

Schwert participated in various committees and councils at the University of Kentucky and was chairman of the Senate Council for one term. He contributed numerous research papers and reviews to professional journals and was a member of the editorial board of the *Journal of Biological Chemistry* from 1965 to 1969 and from 1972 to 1977. Schwert was also an emeritus member of the American Society of Biochemistry and Molecular Biology, the Biochemical Society [Great Britain], and the American Chemical Society.

A long time member of Walnut Hill Church, Schwert served terms as Treasurer and as Senior Warden. For many years he enjoyed the Lexington Philharmonic Orchestra and he served on the Board of the Orchestra. He spent many summers in the mountains of southwestern Colorado. His other recreational interests included travel and model railroads.

Dr. Geroge W. Schwert

Deceased Members

ASBMB would like to recognize the following members who passed away earlier this year:

Annette Baich
Robert Costa
Walton H. Marsh
Nancy G. Nossal
John G. Pierce
Karl A. Piez

Roger Kornberg Wins Nobel Prize in Chemistry

ASBMB member Roger D. Kornberg of Stanford University School of Medicine won this year's Nobel Prize in Chemistry for showing how genes convey their messages in cells to copy functions like making proteins. The Nobel Prize committee cited Dr. Kornberg, 59, for visually showing how information encoded in a cell's DNA blueprint is read and duplicated into what is called messenger RNA. This messenger RNA, in turn, takes the information out of the nucleus to outer areas of the cell where it is used to construct proteins that control cell functions.

Kornberg, who said he was "simply stunned" by the award, is the son of Dr. Arthur Kornberg, who shared the Nobel in medicine in 1959 for his work in DNA information transfer. The Kornbergs are the sixth father and son to win Nobel Prizes. The younger Dr. Kornberg said that while others suggested he might win the prize this year, he viewed it as "improbable."

But the elder Dr. Kornberg, 88, said he was not entirely surprised. "I'm disappointed it's been so long in coming," he said with a smile at a Stanford news conference.

The work is important for medicine, because disturbances in that process are involved in illnesses like cancer, heart disease, and various kinds of inflammation. And learning more about the process is key to using stem cells to treat disease.

Kornberg, 59, a Professor at the Stanford University School of Medicine, had just spent two days traveling from Europe to his home in California when he learned of the honor.

© The Nobel Foundation.

Dr. Roger Kornberg

"When the telephone first rang I was completely bewildered," he said in a telephone interview with journalists in the Swedish capital on October 4, the day after winning the award. "I'm still shaking. I hope I will be able to calm down shortly."

The Kornbergs are the sixth father and son to both win Nobel Prizes. One father and daughter, Pierre Curie and Irene Joliot-Curie, won Nobel Prizes in physics and chemistry, respectively. Marie Curie, Irene's mother and Pierre's wife, won two Nobel prizes, for chemistry and physics.

Photos courtesty of Linda A. Cicero / Stanford News Service

Roger Kornberg with researchers from his lab, from left, Craig Kaplan, Dave Bushnell, Kornberg, Karl-Magnus Larsson, Andreas Ehrensberger, Henrik Spahr, Maia Azubel.

Nobel Prize winner Roger Kornberg will be speaking at the ASBMB sponsored symposia, Transcriptional Regulation by Chromatin and RNA Polymerase II, November 2-5 at Kiawah Island, South Carolina. The title of his address will be "Chromatin and Transcription."

Roger Kornberg's prize-winning work produced a detailed picture of transcription in eukaryotes, the group of organisms that includes humans and other mammals, the Royal Swedish Academy of Sciences said in its citation.

Kornberg shed light on how information is taken from genes and converted to molecules called messenger RNA. These molecules shuttle the information to the cells' protein-making machinery. Proteins, in turn, serve as building blocks and workhorses of the cell, vital to its structure and functions.

Since 2000, Kornberg has produced actual pictures of messenger RNA molecules being created, a process that resembles building a chain link by link. The images are so detailed that individual atoms can be distinguished.

"In an ingenious manner Kornberg has managed to freeze the construction process of RNA half-way through," the Nobel committee said. "That let him capture the process of transcription in full flow, which is "truly revolutionary," the committee added.

The committee said that Kornberg did fundamental work over 20 years on how the information stored in genes is copied and transferred to other parts of the cell. The committee also said he was the first to create pictures of that process. Using a method called X-ray crystallography, he was able to have a computer assemble freeze-frame images of the enzyme at work.

''We've completed the central part of the puzzle,'' said Kornberg. ''Now we want to create a moving picture of the process from beginning to end.''

Dr. Jeremy M. Berg, director of the National Institute of General Medical Sciences at the National Institutes of Health, said that honoring Dr. Kornberg showed the importance of taxpayer-supported basic research not focusing on a specified goal. The institute has financed Dr. Kornberg's work since 1979, even when it was unclear whether the research would be successful, Dr. Berg said.

Kornberg's father, Arthur, shared the 1959 Nobel Prize for Medicine with Severo Ochoa for studies of how genetic information is transferred from one DNA molecule to another. The younger Kornberg said he remembered traveling to Stockholm with his father for the Nobel Prize award ceremonies.

"I have always been an admirer of his work and that of many others preceding me. I view them as truly giants of the last 50 years. It's hard to count myself among them," he said. "Something so remarkable as this can never be expected even though I was aware of the possibility. I couldn't conceivably have imagined that it would become reality."

Nobel Laureate Roger Kornberg (right) with his father, Arthur Kornberg, who was awarded the Nobel Prize in Physiology and Medicine 1959.

Dr. Kornberg, who said he had vivid memories of visiting Stockholm as a 12-year-old to see his father receive his Nobel, said the $1.4 million award should not mean a big change in his family's lives.

''On the salary of a professor, I have mostly debts that will be settled after paying lots of taxes,'' he said. ''Then I'll probably replace my 20-year-old automobile. I don't see much after that."

Roger Kornberg and his father, Arthur, join in congratulating Stanford's Andrew Z. Fire, who shared the 2006 Nobel Prize in Medicine with Craig C. Mello.

Roger Kornberg's father, Arthur Kornberg, has a long history with ASBMB, having served as president of what was then the American Society of Biological Chemistry in 1965. Most recently he authored a *Journal of Biological Chemistry Classic*, Remembering Our Teachers, which can be accessed on the *JBC* website, www.jbc.org/. In the introduction to that article, he wrote:

"The spotlight on the biochemistry stage moves rapidly, leaving a star of yesterday in the dark and virtually forgotten. A substance, a procedure, or a biochemical event named after the star is eventually renamed and what seemed an assurance of immortality is gone. This has been the fate of the Cori cycle, the Cori ester, and of Carl and Gerty Cori as well. In journals (and even more so in textbooks) discoveries are described in a logical pattern unlike the sequence of erratic and serendipitous events which led to them but seldom with references to the authors included. In the hope that we might profit from reflecting on how our scientific lives and work have been shaped by past stars, I want to consider on how they (among them my teachers, Carl Cori, Gerty Cori, and Severo Ochoa) affected mine."

He was also the subject of an article, Arthur Kornberg's Discovery of DNA Polymerase I, by ASBMB Science Editor Nicole Kresge, Robert D. Simoni, and Robert L. Hill, J. Biol. Chem. 2001 276: 3-11.

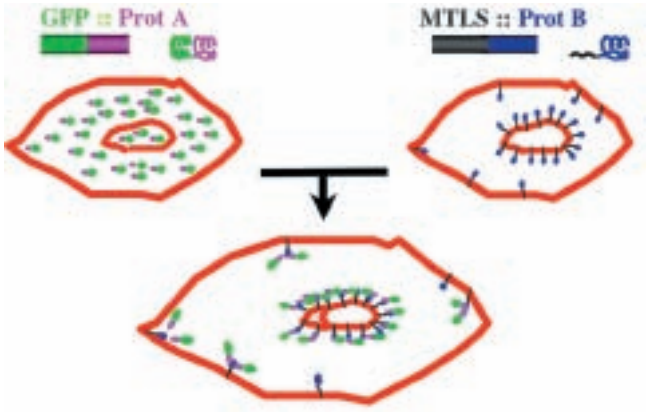
At the 2006 Annual Meeting he also appeared in one of the special video interviews shown throughout the meeting in the ASBMB Lounge. That video can be seen at mms://media.asbmb.org/ASBMB/akornberg-323.wmv.

Article by Nobel Laureate in November MCP

The November issue of *Molecular and Cellular Proteomics* (MCP) contains an article by Dr. Andrew Z. Fire, who was awarded the 2006 Nobel Prize in Physiology or Medicine. Fire shared the Prize with Craig C. Mello for their discovery of RNA interference (RNAi).

The paper, entitled "A Differential Cytolocalization Assay for Analysis of Macromolecular Assemblies in the Eukaryotic Cytoplasm," is available free on the MCP website. In the paper, Fire and colleagues report on their development of a differential cytolocalization assay (DCLA) that allows the observation of cytoplasmic protein-protein interactions *in vivo*. In the DCLA assay, interactions are visualized as a relocalization of a GFP-tagged "prey" by a membrane bound "bait."

The researchers tested the assay in *C. elegans*, using it to probe interactions among proteins involved in RNA interference (RNAi) and nonsense mediated decay (NMD) pathways. Several previously documented interactions were confirmed with the assay, and many new ones were observed. Fire and co-workers also used the assay to test a subset of the RNAi and NMD interactions in animals mutant for proteins central to each mechanism, thereby identifying several key associations that can occur *in vivo* in the absence of a functional process.



A schematic of Fire's differential cytolocalization assay (DCLA).

Previously Approved Drugs May Be Helpful in Fatal Pediatric Disorder

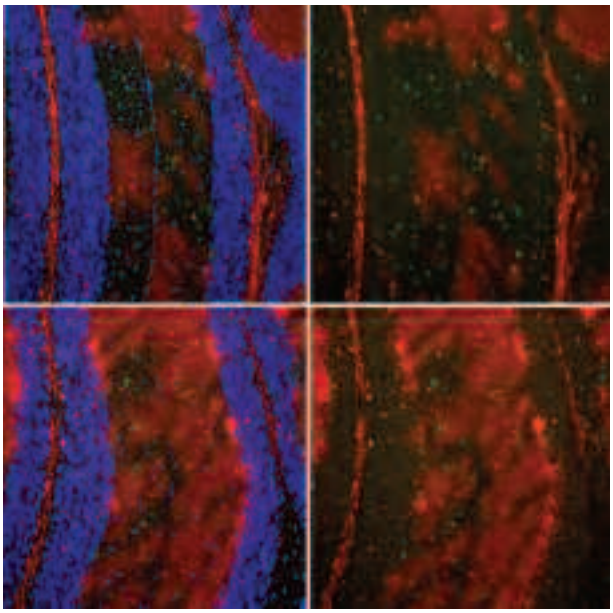
According to a report in the September 12 issue of the *Proceedings of the National Academy of Sciences*, a progressive neurodegenerative disorder that is often fatal within the first two decades of life may be treatable via a receptor already targeted by approved drugs.

Scientists working with a mouse model for Niemann-Pick type C (NPC) disease showed that experimental treatments appear to be acting through the pregnane X receptor (PXR). Found in the cell nucleus, PXR regulates the activity of a cluster of genes that helps rid the body of toxins.

NPC belongs to a broader class of diseases known as lysosomal storage disorders, which affect more than 30,000 people worldwide. In these disorders, disposal and recycling within lysosomes become jammed, preventing proper degradation of compounds and leading to a build up.

In NPC, cholesterol builds up in the lysosomes. Cells use compounds made from cholesterol to sense and regulate their own internal cholesterol levels. Thus the lysosomal jam disrupts the creation of these byproducts, impairing the cell's ability to sense its own cholesterol levels. As a result, cells continue to make and take in cholesterol even after their cholesterol levels are already dangerously high.

Using a mouse model of NPC, Dr. Daniel S. Ory, Associate Professor of Medicine and of Cell Biology and Physiology at Washington University School of Medicine, began to test the effects of T0901317, a synthetic oxysterol ligand that activates genes involved in cholesterol breakdown and removal. At the same time, Dr. Synthia H. Mellon of the University of California-San Francisco was treating the same mouse line with the neurosteroid allopregnanolone. Both efforts met with moderate success. However, when the treatments were combined, a single dose 7 days after birth extended the average mouse lifespan from 78.8 days to 135.7 days.



Treatment with compounds that activate nuclear receptors (bottom panel) promotes Purkinje cell (red) survival in the cerebella of mice deficient in NPC1 protein. Photograph courtesy of S. Joshua Langmade.

The researchers determined that the treatments were acting by increasing the activity of a gene regulated by PXR. To establish definitive proof of the link between PXR and successful treatment of NPC, Ory and his colleagues have now created a mouse line that has NPC but lacks the gene for PXR. If the T0901317-allopregnanolone treatment does not help those mice, then the link between PXR and NPC will be confirmed.

There are currently several drugs available that target PXR, including the antiseizure drug dilantin, the antibiotic rifampicin, and the herbal compound St. John's Wort, all of which may potentially be used in clinical trials for NPC.

ASBMB member Daniel S. Ory is an Associate Professor of Medicine, Cell Biology and Physiology, at the Washington University School of Medicine. He received his B.A. from Harvard University (1982) and M.D. from Harvard Medical School (1986). Ory worked at Brigham and Women's Hospital in Boston, Massachusetts General Hospital, Whitehead Institute for Biomedical Research, and Barnes-Jewish Hospital in St. Louis prior to joining the faculty of the Washington University School of Medicine in 1995.

Ory is the recipient of many awards, including the 2006 SCCOR Award from the National Institutes of Health and the 2003 Washington University/Pfizer Biomedical Research Award. A member of several professional societies including the American Society for Clinical Investigation and the American Association for the Advancement of Science, Ory has authored or co-authored over 26 publications. He also holds three patents and many editorial responsibilities. Currently, work in his laboratory focuses on the identification and characterization of genes that function in the uptake, intracellular transport, and export of lipoprotein-derived cholesterol.

Dr. Daniel S. Ory

Scientist-Astronaut Sends T-cells into Space

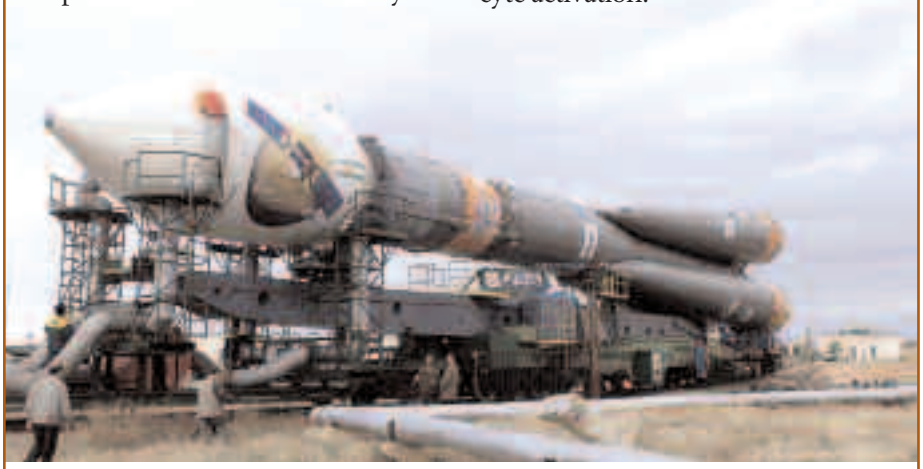
Millie Hughes-Fulford, a former astronaut and current researcher at the San Francisco VA Medical Center, traveled to the Cosmodrome space-launch site at Baikonur, Kazakhstan, this past September to prepare a crucial experiment designed to demonstrate how the human immune response is suppressed in the weightless environment of space. Hughes-Fulford then sent human T-cells up to the International Space Station (ISS) aboard ISS Soyuz 13.

"We're doing this experiment because many astronauts are immunosuppressed during flight. Their T-cells stop working in microgravity," said Hughes-Fulford. "This experiment will tell us for the first time exactly which genes involved in the normal immune response aren't activated in space."

ASBMB member Millie Hughes-Fulford received her Bachelor of Science degree in Chemistry and Biology from Tarleton State University in 1968 and her Ph.D. from Texas Woman's University in 1972. Upon completing her doctorate degree, Hughes-Fulford joined the faculty of Southwestern Medical School at the University of Texas, Dallas, as a postdoctoral fellow. In 1973 she became a Research Chemist in Veterans Administration Medical Center, and in 1977 she joined the Biochemistry Department at the University of California, Berkeley. Selected as a payload specialist by NASA in January 1983, Hughes-Fulford flew in June 1991 aboard STS-40 Spacelab Life Sciences (SLS 1). She was a major in the U. S. Army Reserve Medical Corps until 1995.

Dr. Millie Hughes-Fulford

Hughes-Fulford is currently an Adjunct Professor at the University of California Medical Center at San Francisco and is a principal Investigator at the San Francisco Department of Veteran's Affairs Medical Center where she continues her research. As the Director of the Laboratory for Cell Growth and Scientific Advisor to the Under Secretary of Veteran's Affairs, she studies the control of human prostate cancer growth and the regulation of bone and lymphocyte activation.



The Soyuz launcher during transport to the launch pad.Credits: ESA/STARSEM-S.Corvaja.

The problem of immunosuppression in microgravity was first noted during the Apollo moon mission series in the 1960s and 1970s, when 15 out of 29 Apollo astronauts developed infections during their missions or soon after landing. Subsequent experiments aboard Skylab and several space shuttle missions, including Hughes-Fulford's, confirmed that T-cells do not activate properly in microgravity.

"In this experiment, we're looking at why they're not working," said Hughes-Fulford. "Normally, for T-cells to be activated, certain genes have to be expressed in a certain order in a signaling pathway. Aboard the ISS, we hope to find exactly which genes are not being expressed in microgravity."

The experiment was carried to the International Space Station inside a specially designed incubator called Kubik, which was made to fit precisely under the cosmonaut's seat in the Soyuz spacecraft. Kubik contains a compartment for weightless experiments as well as a centrifuge that can accelerate cells in a range from 0.2 to 2 earth gravities.

On board the space station, European Space Agency astronaut-scientist Thomas Reiter simultaneously activated T-cells in the weightless compartment and in the centrifuge for four hours. "By activating the cells, he simulated the activation that normally occurs in response to infection," Hughes-Fulford explained. "He set up the whole cascade that would normally turn on the T-cells. Except we know that some of the genes will not turn on because they're in a weightless environment."

At the end of the experiment, the T-cells were safely packaged and then sent back to Earth aboard the return-

Continued next page

Solution to Bacterial Mystery Promises New Drugs

A 25-year quest to identify the first biochemical step that many disease-causing bacteria use to build their membranes has led to a discovery that holds promise for effective, new antibiotics against these bacteria. A report on this finding appears in the September 1 issue of *Molecular Cell*.

"We identified a biochemical process that uses a previously unrecognized molecule as a raw material to make phospholipid," explains Dr. Charles Rock, a member of the St. Jude Children's Research Hospital's Department of Infectious Diseases and senior author of the paper. "That discovery solved a mystery that has puzzled researchers for 25 years."

As bacteria grow and divide, they must make additional membrane using a series of biochemical reactions. The first step in this process is the acylation of glycerol-3-phosphate, which is then used in the formation of phosphatidic acid, the key intermediate in membrane phospholipid synthesis.

Scientists have used *E. coli* for many years as a model to understand how bacteria make membrane phospholipids. However, Rock's research now shows that the first step in membrane biosynthesis in *E. coli* is entirely different from that found in most strains of pathogenic bacteria.

In *E. coli*, acylation of the 1-position of glycerol-3-phosphate is carried out by PlsB. However, the majority of bacteria lack a *plsB* gene, and in others it is not essential. Rock and his colleagues showed that Gram-positive bacteria use a two-step pathway with a new fatty acid intermediate for the initiation of phospholipid formation. The bacteria first use PlsX to produce a unique activated fatty acid by catalyzing the synthesis of fatty acyl-phosphate from acyl-acyl carrier protein. Then, PlsY transfers the fatty acid from acyl-phosphate to the 1-position of glycerol-3-phosphate.

"The biochemical pathway that uses PlsX and PlsY is the most widely distributed bacterial pathway for initiating the production of phospholipids," explained the study's first author, Dr. Ying-Jie Lu, of the St. Jude Department of Infectious Diseases. "It turns out that *E. coli* is more of an oddball rather than in the mainstream when it comes to how it makes membranes."

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ing Soyuz craft. Hughes-Fulford is currently analyzing the results in her lab in San Francisco.

"Our expectation is that the T-cells in the centrifuge–basically, under artificial gravity–will be activated normally, and the T-cells in microgravity will not be activated," she predicted. "We will compare them side by side and discover, for the first time, exactly which genes did not turn on in microgravity."

Hughes-Fulford placed an earlier version of the same experiment aboard the space shuttle Columbia on shuttle mission STS-107. At the end of that mission on February 1, 2003, the Columbia broke up upon reentry into Earth's atmosphere, killing all seven crew members and destroying all experiments aboard.

"We cannot go to Mars, or even to the Moon over the long term, without knowing more about why T-cells are not working," said Hughes-Fulford. "When we learn that, we can start looking for possible treatments."

Dr. Charles Rock. Biomedical Photography, St. Jude Children's Research Hospital.

ASBMB member Charles O. Rock is a Professor in the Department of Molecular Sciences at the University of Tennessee and a member of the Department of Infectious Diseases at St. Jude Children's Research Hospital. He earned his B.S. in Zoology from Colorado State University in 1971 and his Ph.D. in Lipid Biochemistry from the University of Tennessee at Oak Ridge in 1976. Between 1976 and 1979 he conducted two postdoctoral fellowships, one at Oak Ridge Associated Universities and one at Yale University. After working as a Research Associate for one year at the University of Illinois, Rock became an Assistant Member in the Department of Biochemistry at St. Jude Children's Research Hospital in 1980. He was promoted to Associate Member in 1985 and became Full Member in 1992. Rock was also an Assistant Professor in the Department of Biochemistry at the University of Tennessee from 1981 to 1985 and an Associate Professor in the Department of Molecular Sciences from 1987 to 1993.

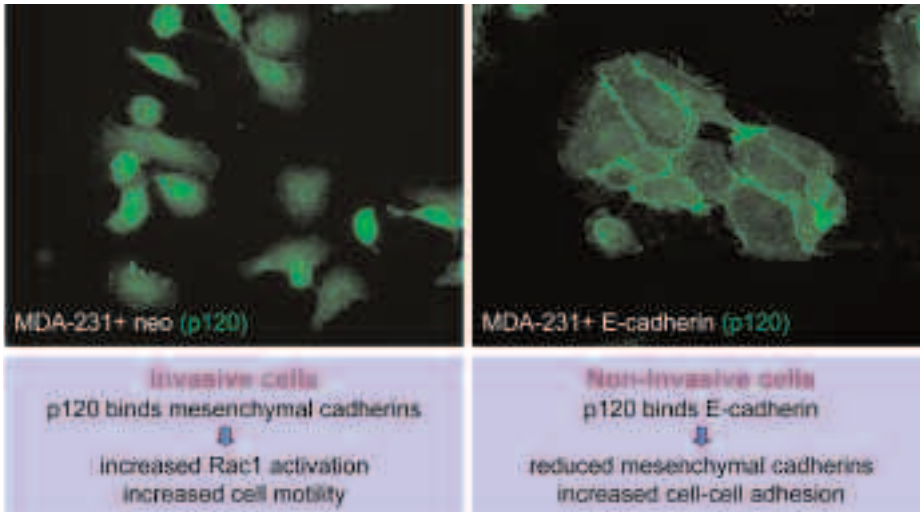
Rock's research interests include control points in fatty acid biosynthesis, regulation of membrane phospholipid metabolism, and small molecule therapeutics. In addition to serving on the editorial boards of several journals, he has chaired several scientific sessions at national meetings.

Protein Can Both Stop and Promote Metastasis

According to researchers at the Mayo Clinic, a protein known to be a key component of the glue that holds cells together also is involved in breaking them apart and promoting their movement when tumors begin to spread to other parts of the body.

The study, published in the September 25 issue of the *Journal of Cell Biology*, helps illuminate the very first steps involved in metastasis and suggests that a future designer drug might be able to block the beginning of this dangerous process or stop it once it starts.

"Our data show that p120 catenin is a key player in both suppressing invasion and promoting it," says the study's senior author, Dr. Panos Anastasiadis, a Mayo Clinic cancer researcher. "This is very exciting because the findings open up a whole new field of discovery for novel therapeutics that should be applicable to most types of tumors."



Recruitment of p120 catenin (green) to E-cadherin or mesenchymal cadherins regulates the sessile versus motile behavior of epithelial cells. Modified from Yanagisawa and Anastasiadis (2006) J. Cell Biol. 174, 1087-1096.

The study looked at how p120 catenin interacts with different cadherin proteins in cancer cells. The cadherins are a class of cell surface proteins involved in cell adhesion. The molecules' extracellular domains interact with other cadherin proteins on adjacent cells, whereas their intracellular domains bind to catenins, which regulate a cell's shape and function.

The best understood cadherin is E-cadherin, which provides tight connections between epithelial cells. The other cadherins used in the study belong to a group collectively called "mesenchymal" cadherins, which provide a looser bond between the cells that sparsely populate the connective tissue.

Sometimes, such as during human development or wound repair, epithelial cells need to travel to other areas, and to do this, they undergo a process known as "epithelial-mesenchymal transition" (EMT). The cell reduces its production of E-cadherin proteins and increases expression of mes-

Continued next page

ASBMB member Panagiotis Z. Anastasiadis is Senior Associate Consultant at the Mayo Clinic Comprehensive Cancer Center and Assistant Professor in the Department of Cancer Biology at the Mayo Clinic in Jacksonville. He received his B.S. in Biology at the Aristotelian University of Thessaloniki in Greece (1987) and his Ph.D. in Cellular and Clinical Neurobiology at Wayne State University (1993). Anastasiadis was a postdoctoral fellow at the Henry Ford Hospital in Detroit and at Vanderbilt University prior to becoming an Assistant Professor at the Sealy Center for Cancer Cell Biology in 2001. He joined the Mayo Clinic in 2003.

Anastasiadis has authored or co-authored over 30 publications and is a member of several professional societies including the American Society for Cell Biology. His honors and awards include the 2000 Vanderbilt-Ingram Cancer Center award and a 1992 Thomas C. Rumble University Graduate Fellowship. Currently, his research focuses on the role of cadherins and catenins in human cancer. He is especially interested in cadherin-mediated signaling events that suppress cell motility and invasiveness and promote the reorganization of the actin cytoskeleton.

Dr. Panos Anastasiadis (left) and co-author Dr. Masahiro Yanagisawa (right).

NIH Announces 2007 Pioneer Award Competition

NIH Director Elias A. Zerhouni launched a new round of competition for the NIH Director's Pioneer Award on October 12. This signature program supports exceptionally creative scientists who take highly innovative, and potentially transformative, approaches to major challenges in biomedical research.

Dr. Elias Zerhouni

"We hope this opportunity stimulates even more investigators to send us their boldest, most imaginative concepts," said Zerhouni. "The Pioneer Award supports individual scientists rather than specific projects and allows recipients to pursue promising new research directions that could have unusually great impact. This program is one way we are exploring the funding of scientists whose ideas might be too novel, span too diverse a range of disciplines, or be at too early a stage to fare well in the traditional NIH peer review process."

Each Pioneer Award provides $2.5 million in direct costs over five years. NIH funded 35 scientists in the first three years of the program, which is part of the NIH Roadmap for Medical Research. In September 2007, the agency expects to make between five and ten new Pioneer Award grants.

Scientists at all career levels and engaged in any field of research may apply for the Pioneer Award, as long as they are interested in exploring biomedically relevant topics.

"We hope to see a diverse applicant pool again this year. Toward that end, we continue to encourage applications from women, members of groups that are underrepresented in biomedical research, and individuals in the early to middle stages of their careers," said Jeremy M. Berg, Director of the National Institute of General Medical Sciences and a leader of the Pioneer Award program.

The centerpiece of the streamlined, electronic application process is an essay on the investigator's vision for addressing a biomedical challenge, the importance of the problem, and the person's qualifications to engage in groundbreaking research. The application period opens on Friday, December 1, 2006, and closes on Tuesday, January 16, 2007.

Application instructions are at http://grants.nih.gov/grants/guide/rfa-files/RFA-RM-07-005.html. More information on the Pioneer Award is at http://nihroadmap.nih.gov/pioneer.

Continued from previous page

enchymal cadherins, thus effectively loosening the anchors that keep the cell bound to its neighbors.

Cancer, unfortunately, has adopted this strategy in order to spread, Anastasiadis says. "When the function of E-cadherin is lost in a cell, it can break free from its neighbors and travel to settle elsewhere," he says. "This means that E-cadherin normally helps suppress invasion."

But researchers have noted that the p120 catenin protein seems mysteriously two-faced: although it normally strengthens cell-cell bonding, in some cases it can also negatively affect cell adhesion. They also have found that over-production of p120 increases a cell's ability to move. But the significance of these observations had eluded scientists.

In this study, Anastasiadis and his colleagues provide an answer as to why p120 acts this way, which helps explain how the EMT shift between E-cadherin and mesenchymal cadherins allows cancer cells to break away from tissue and spread.

They found that p120 prefers to bind to E-cadherin rather than to mesenchymal cadherins. So, in normal epithelial cells, p120 always associates with the more abundant E-cadherins. But when E-cadherin production is lost during the progression of cancer, p120 catenins begin binding to mesenchymal cadherins. And when that happens, the researchers found that p120 unexpectedly switches on a cascade of events that promote cell movement.

"We show that E-cadherin suppresses invasion, at least in part, by binding to p120 protein in the cell," Anastasiadis says. "If E-cadherin is missing, p120 is free to bind to mesenchymal cadherins, setting off a process that leads to metastasis."

Thus, p120 acts as a rheostat that promotes either stability when associated with E-cadherin or motility when it interacts with mesenchymal cadherins, he says.

The investigators say that further research is needed to see if p120 functions the same way in living tissue as it does in laboratory cell culture, and they add that other pathways are likely involved in the transition to metastasis. But, if the results continue to hold up, it might be therapeutically possible to selectively shut down the pro-invasive function of p120 on mesenchymal cadherins while keeping the pro-adhesion function of p120 in normal epithelial cells.

NIH Director Announces 2006 Pioneer Award Recipients

Five-Year, $2.5 Million Grants Support Highly Innovative Research

NIH Director Elias A. Zerhouni has named 13 recipients of the 2006 NIH Director's Pioneer Award.

Now in its third year, the Pioneer Award is a key component of the NIH Roadmap for Medical Research. The program supports exceptionally creative scientists who take highly innovative approaches to major challenges in biomedical research.

"The 2006 Pioneer Award recipients are a diverse group of forward-thinking scientists whose work could transform medical research," said Zerhouni. "The awards will give them the intellectual freedom to pursue exciting new research directions and opportunities in a range of scientific areas, from computational biology to immunology, stem cell biology, nanotechnology, and drug development."

The 2006 awardees, who will each receive $2.5 million in direct costs over five years, are:

Kwabena A. Boahen, Stanford University associate professor of bioengineering, who will develop a specialized hardware platform for the detailed simulation of the inner workings of the brain's cortex.

Arup K. Chakraborty, Massachusetts Institute of Technology Robert T. Haslam Professor of Chemical Engineering, Chemistry, and Biological Engineering, who will combine the application of theoretical methods rooted in statistical physics and engineering with experiments to determine principles governing the emergence of autoimmune diseases.

ASBMB member Lila M. Gierasch, University of Massachusetts, Amherst, professor of biochemistry and molecular biology and chemistry, who will investigate protein folding in the complex environment of a cell and explore how diseases may arise from folding mistakes.

Rebecca W. Heald, University of California, Berkeley, associate professor of molecular and cell biology, who will study how cells scale the size of their internal organelles.

Karla Kirkegaard, Stanford University School of Medicine professor and chair of microbiology and immunology, who will identify and validate targets for antiviral drugs leading to suppression of the growth of drug-resistant variants of dengue, West Nile, hepatitis C, and polio viruses.

Thomas J. Kodadek, University of Texas Southwestern Medical Center at Dallas professor of internal medicine and molecular biology, who will develop a chemistry-based approach to monitor and manipulate the immune system.

Cheng Chi Lee, University of Texas Health Science Center at Houston associate professor of biochemistry and molecular biology, who will refine technologies for the suspended animation of non-hibernating mammals.

Evgeny A. Nudler, New York University School of Medicine professor of biochemistry, who will develop new types of antimicrobial drugs and vaccines to treat and prevent drug-resistant infections.

Gary J. Pielak, University of North Carolina at Chapel Hill professor of chemistry, who will study proteins involved in neurodegenerative diseases at the atomic level inside living cells.

David A. Relman, Stanford University associate professor of microbiology and immunology and of medicine, who will explore the roles in health and disease of microbial communities indigenous to humans.

Rosalind A. Segal, Dana-Farber Cancer Institute associate professor of neurobiology, who will focus on identifying the way complex sugars work to maintain neural stem cells in the developing and adult brain.

James L. Sherley, Massachusetts Institute of Technology associate professor of biological engineering, who will work to develop routine methods for the production of human adult stem cells from liver, pancreas, hair follicles, and bone marrow.

Younan Xia, University of Washington, Seattle, professor of chemistry, who will develop nanomaterials as new tools for understanding and controlling cell communication.

NIH selected the 2006 Pioneer Award recipients through a special application and evaluation process. After NIH staff determined the eligibility of each of the 465 applicants, the first of three groups of distinguished experts from the scientific community identified the 25 most highly competitive individuals in the pool. The second group of outside experts then interviewed the 25 finalists at NIH in August 2006. The Advisory Committee to the Director performed the final review and made recommendations to Zerhouni based on the evaluations by the first two groups.

Choosing To Be an Editor

By Deborah Sweet

I can't really remember why or when I first started thinking about becoming an editor because I can't remember a time working in the lab when I wasn't considering it as a possibility. From my postgraduate time onwards, I had doubts about whether I wanted to pursue the standard academic track. I did think vaguely about a variety of possible career routes (such as medicine or law), but I've always really enjoyed science, and biology in particular, so it was hard for me to imagine doing something that wasn't closely involved with biological research. For a variety of reasons (which I will discuss below), editing really appealed to me.

Although it was stressful at times, I quite enjoyed being an undergraduate student and having my entire life be immersed in learning about a wide range of science. Once I started working in the lab full time, I realized that I enjoyed reading, writing, and thinking about science just as much as actually doing it myself. I also found that, compared to the average bench scientist, I was quite good at communicating science effectively, especially in writing. This became even more noticeable once I was a postdoctoral researcher. I (frankly) got somewhat bored with my own projects because they moved so slowly, and I often found other people's experiments more interesting than my own. I really enjoyed writing papers, review articles, and short summaries of recent papers for the "Headlines" section of *Trends in Cell Biology*. I also enjoyed helping other people write their grants and papers and found I was quite frequently asked to read and critique pieces because my colleagues valued my comments. The idea of doing this type of thing every day, and getting paid for it, seemed very appealing to me. I was also really attracted to the idea of being able (and in fact needing) to keep up with scientific advances across a wide range of fields. It seemed to me that editorial work would emphasize aspects of the research enterprise that I enjoyed, minimize ones that I wasn't as enamored with, and match well with my particular strengths. I found it very easy to imagine myself really enjoying daily life as an editor.

There were, of course, arguments against moving away from academic science. I did enjoy many aspects of bench science, and I was concerned that I would really miss doing research myself. I also had to contend with feeling that I was in some way giving up, or "copping out," and running away from a challenge rather than standing up to it because leading a successful research group clearly was a challenge. There was, and still is (quite rightly) a lot of publicity about the relatively small number of senior women scientists, and moving away from bench science felt like a step in the wrong direction if I wanted to support efforts to change the overall gender balance. I had also been fortunate enough to study and work in very high quality institutions, and I was concerned that insecurity regarding my ability to get a position in an equally stimulating environment was clouding my decision. These feelings weren't helped by various colleagues, particularly more senior ones, who found it difficult to understand why anyone would want to do anything other than academic science. Even after I had made the decision to switch, it took several years for me to stop feeling slightly defensive about not continuing down the academic path.

I also thought about the general organization of my working life. I've never really had a problem with short-term deadlines; studying for exams or working towards a grant submission deadline never seemed especially onerous to me. Therefore, publishing deadlines seemed equally manageable, and even motivating. However, I found the standard scientific funding structure of a grant—which lasted approximately three years and then required one to get more funding in order to continue—quite stressful. Many of my colleagues had no such concerns; they were convinced that it would all work out, and they could just focus on the science, and it may well be that most people who work in research feel that way. However, it seemed to me that I would feel considerably more secure, and thus happier, if I had a job with a more standard structure.

I discussed all of these points at length with a number of my colleagues and friends, so many of them were not at all surprised when I took my first editing job. While I was a graduate student, I went for an informational interview with Carol Featherstone, then the Editor of *Trends in Cell Biology*, and she was very honest about the positives and negatives of editing as a profession. The argument that tipped the balance for me was thinking that if I did not take the opportunity to try something that seemed appealing on multiple levels, I would always wonder what would have happened if I had. When I was offered my first editing position, I decided I would try it for a year or so, and then if I found it really wasn't for me I would move back to research by doing another postdoctoral fellowship. Ten years on I am still here, albeit having moved around. I'm very glad I took the plunge, and at this point I have no regrets at all.

DoD Awards Center of Excellence Grant to V. Craig Jordan

Dr. V. Craig Jordan of Fox Chase Cancer Center has received a $10.7 million grant from the Department of Defense Breast Cancer Research Program. The grant is for a Breast Cancer Center of Excellence focused on developing a new treatment model for breast cancer to reverse the eventual development of resistance to anti-estrogen therapy. The five-year multidisciplinary project, intended to encompass both laboratory research and clinical trials, involves scientists and physicians at Fox Chase and three other institutions, representing four task teams.

Dr.V. Craig Jordan

Jordan is vice president and scientific director of medical science at Fox Chase and holds its Alfred G. Knudson Jr., M.D., Ph.D., Chair in Cancer Research. He is known as the "father of tamoxifen" for his seminal work that led to the validation of tamoxifen as a therapy targeted to treat breast cancer and the first-ever drug to prevent breast cancer.

Much of Jordan's 35-year research career has focused on "designer estrogens" such as tamoxifen and newer drugs. Classed as selective estrogen-receptor modulators, or SERMs, they act like the hormone estrogen in some ways but not in others. These drugs can bind to the hormone receptors found in breast cells and thus block the effects of natural estrogen which can promote breast cancer.

Clapham Wins Bristol-Myers Squibb Cardiovascular Research Award

David E. Clapham, the Aldo R. Castañeda Professor of Cardiovascular Research, Professor of Neurobiology at Harvard Medical School, Director of Cardiovascular Research at Children's Hospital Boston, and an Investigator of the Howard Hughes Medical Institute, has been selected to receive the Bristol-Myers Squibb Freedom to Discover Award for Distinguished Achievement in Cardiovascular Research.

Clapham is being recognized for his seminal contributions to understanding the control of heart rate at the molecular and cellular level and for his determined and creative efforts in the probing of the regulation of the cardiac G protein-gated potassium channel. He was selected to receive the Distinguished Achievement Award by an independent panel of his peers. He will receive a $50,000 cash prize and a silver commemorative medallion.

"Dr. Clapham's pioneering research on potassium channels, and more recently on calcium channels, should lead the way to important new treatments for serious cardiac arrhythmias, benefiting patients around the world," said Richard E. Gregg, M.D., vice president, Clinical Discovery, Bristol-Myers Squibb. "He has given us extraordinary insights into the complex cascade of signaling events that occurs in cells and controls many basic biologic processes, telling us how to better control the electrical impulses in our hearts."

Dr. David Clapham

Olson Honored by Biophysical Society

Rice University biochemist Dr. John Olson is the winner of this year's Emily M. Gray Award from the Biophysical Society. The award is the international professional organization's top award for education and outreach.

Olson, the Dorothy and Ralph Looney Professor of Biochemistry and Cell Biology, has won five teaching awards at Rice, including three George R. Brown Awards. Olson was also recognized for his mentoring. Since arriving at Rice in 1973, he has advised 22 students as they earned their Ph.D.s and currently is mentoring four more doctoral candidates

Olson said his service as director of large National Institutes of Health-sponsored graduate student training programs — such as the Houston Area Molecular Biophysics Predoctoral (HAMBP) Training Grant program — likely played a role in his winning the Gray Award. HAMBP provides fellowships for students from Rice, Baylor College of Medicine, M.D. Anderson Cancer Center, the University of Houston, the University of Texas Medical School at Houston and the University of Texas Medical Branch–Galveston.

Dr. John Olson

As winner of this year's Gray Award, Olson will present the keynote address at the student symposium at the Biophysical Society's annual meeting next March in Baltimore.

Lila M. Gierasch Receives NIH Pioneer Award

Research into how proteins fold and why they misfold has garnered a $2.5 million award from the National Institutes of Health (NIH) for Lila M. Gierasch, Distinguished Professor of Biochemistry and Molecular Biology and Chemistry at the University of Massachusetts. Gierasch and 12 other awardees received 2006 NIH Director's Pioneer Awards at a September ceremony in Washington D.C.


Dr.Lila M. Gierasch

The Pioneer Award is given to exceptionally creative scientists who take highly innovative approaches to major challenges in biomedical research. Gierasch will use the award to continue her investigations of protein folding in the complex environment of a cell and explore how diseases may arise from folding mistakes. Now in its third year, the Pioneer Award is a key component of the NIH Roadmap for Medical Research.

"The 2006 Pioneer Award recipients are a diverse group of forward-thinking scientists whose work could transform medical research," said NIH Director Dr. Zerhouni. "The awards will give them the intellectual freedom to pursue exciting new research directions and opportunities in a range of scientific areas, from computational biology to immunology, stem cell biology, nanotechnology, and drug development."

Fuchs Given Steven C. Beering Award

Dr. Elaine Fuchs received the 2006 Steven C. Beering Award for Advancement of Biomedical Science, from the Indiana University School of Medicine.

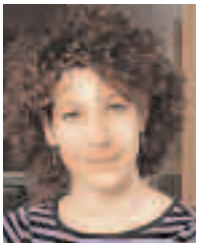
Established through contributions of faculty, alumni and friends of the School of Medicine as a tribute to the former dean, the Beering Award honors an internationally recognized individual for outstanding research contributions to the advancement of biomedical or clinical science. The award winner receives a medal and a check for $10,000, and spends about three days on campus, during which one or two additional lectures to smaller groups are planned.

Fuchs is Rebecca C. Lancefield Professor and Head of the Laboratory of Mammalian Cell Biology and Development at The Rockefeller University. Fuchs, who is also a Howard Hughes Medical Institute investigator, is a world leader in skin biology and its human genetic disorders, which include skin cancers and life-threatening genetic syndromes such as blistering skin disorders.


Dr.Elaine Fuchs

Bassler to Receive Eli Lilly and Company Research Award

The American Society for Microbiology has announced that Dr. Bonnie Bassler will receive its 2006 Eli Lilly and Company Research Award. The Eli Lilly Award is the Society's oldest and most prestigious prize. It recognizes fundamental research of unusual merit in microbiology or immunology by an individual on the threshold of his or her career.


Dr. Bonnie Bassler

Bassler, Professor of Molecular Biology at Princeton University and an investigator for the Howard Hughes Medical Institute, is being honored for her research in bacterial cell-cell communication and for leading contributions to the understanding of the molecular mechanisms underlying this communication process, called quorum sensing.

The award consists of a cash prize of $5,000, a commemorative piece, and travel to the American Society for Microbiology General Meeting where Bassler will deliver the Eli Lilly Award Lecture.

Laura Kiessling Selected For Harrison Howe Award

The ACS Rochester Section has selected Dr. Laura L. Kiessling, Professor of Chemistry and Biochemistry at the University of Wisconsin, Madison, and director of the Keck Center for Chemical Genomics, as the 2005 recipient of the Harrison Howe Award. The award, which consists of a plaque and an honorarium, was established in 1946 to honor Harrison E. Howe, one of the founders of the Rochester Section.

Kiessling is being recognized for her work at the interface of chemistry and biology and specifically for her work on the synthesis and application of materials that illustrate the biological role of carbohydrates, and the understanding and study of signal transduction pathways.


Dr. Laura Kiessling

By Nicole Kresge, Science Editor

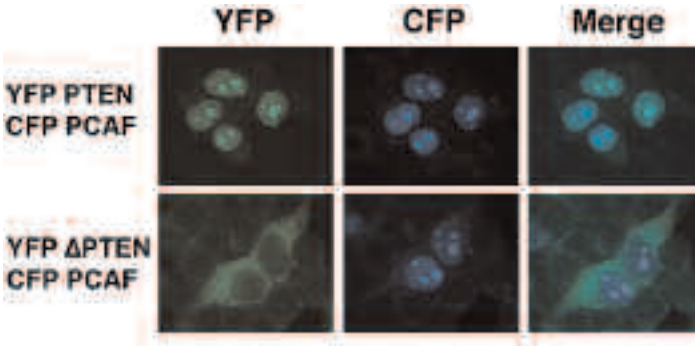
ASBMB Bio Bits

PCAF Modulates PTEN Activity

Koichi Okumura, Michelle Mendoza, Robert M. Bachoo, Ronald A. DePinhol, Webster K. Cavenee, and Frank B. Furnari

J. Biol. Chem. 2006, 281: 26562-26568

PTEN is an important tumor suppressor in human cancer that also plays a role in the regulation of cell migration, growth, and apoptosis. These functions are mediated by the protein's lipid phosphatase activity, which inhibits the phosphatidylinositol 3-kinase (PI3K) signaling pathway. Now, this paper establishes that PTEN lipid phosphatase activity is inhibited when the protein is acetylated by the histone acetyltransferase PCAF. The authors show that expression of PCAF results in increased acetylation of two lysine residues within the catalytic cleft of PTEN, a structure essential for PI3K specificity. Furthermore, reduction of endogenous PCAF activity using short hairpin RNA results in a loss of PTEN acetylation and restores the ability of PTEN to downregulate PI3K signaling and to induce G1 cell cycle arrest. The authors also demonstrate that acetylation-resistant PTEN mutants are able to retain their PI3K signaling in the presence of PCAF.



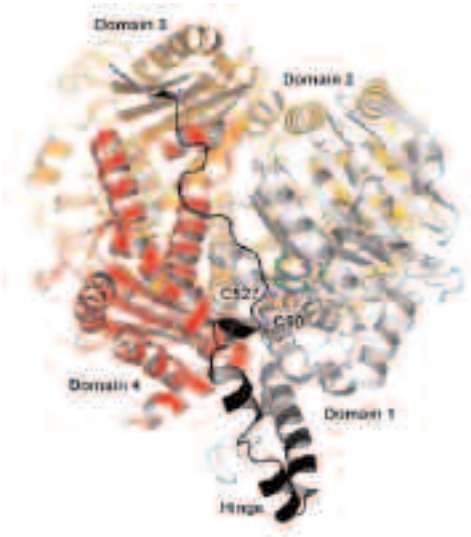
PTEN and PCAF co-localize in cells.

Degradation of the Amyloid beta-Protein by the Novel Mitochondrial Peptidasome, PreP

Annelie Falkevall, Nyosha Alikhani, Shashi Bhushan, Pavel F. Pavlov, Katrin Busch, Kenneth A. Johnson, Therese Eneqvist, Lars Tjernberg, Maria Ankarcrona, and Elzbieta Glaser

J. Biol. Chem. 2006 281: 29096-29104

In Alzheimer disease, amyloid beta-protein polymerizes into insoluble fibrils in the brain. Although the extracellular accumulation of these plaques has been the focus of most molecular studies associated with Alzheimer disease, lately, increasing attention has focused on intracellular events, including the role of the mitochondria in the disease. In this paper, the authors report that a novel mitochondrial metalloendopeptidase named presequence protease, or PreP, degrades amyloid beta-protein. They produced recombinant hPreP and characterized its proteolytic activity both *in situ* and *in vitro*. Anti-hPreP antibodies caused complete inhibition of proteolytic activity against amyloid beta-protein. The researchers also investigated the degradation pattern of the amyloid beta-protein using mass spectrometric analysis and found that hPreP used several unique cleavage sites. These results indicate that mitochondrial degradation of amyloid beta-protein by hPreP may be important in the pathology of Alzheimer disease.

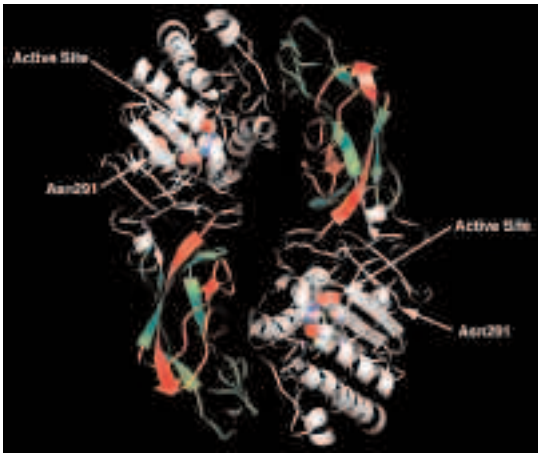


A model of hPreP with amyloid beta-protein bound to the active site.

A Systematic Review and Meta-Analysis of the Relationship between Lipoprotein Lipase Asn291Ser Variant and Diseases

Yaomin Hu, Wei Liu, Rong Huang, and Xiaoying Zhang

J. Lipid Res. 2006 47: 1908-1914.



A model of the LPL homodimer.

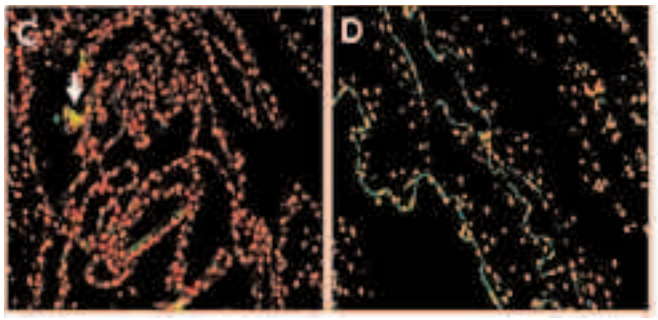
Lipoprotein lipase (LPL) is a key enzyme that hydrolyzes triglyceride contained in the core of both chylomicrons and VLDL particles. Because of its role in lipid breakdown, the loss of LPL function may cause dyslipidemia. In this JLR paper, the authors attempted to summarize the associations between the Asn291Ser variant in the LPL gene and dyslipidemia, the risk of type 2 diabetes mellitus, and coronary heart disease. In addition, the relationships between the Asn291Ser variant and other metabolic diseases such as obesity and high blood pressure were investigated. To do this, the authors systematically reviewed 21 articles by meta-analysis. They found that the Asn291Ser variant is a risk factor for dyslipidemia and that this risk increases in severity with age and weight gain. The analysis also indicated that the LPL Asn291Ser variant is associated with type 2 diabetes mellitus and coronary heart disease.

Proteomic Analysis of the Extraembryonic Tissue from Cloned Porcine Embryos

Jung-Il Chae, Seong-Keun Cho, Jung-Woo Seo, Tae-Sung Yoon, Kyu-Sun Lee, Jin-Hoi Kim, Kyung-Kwang Lee, Yong-Mahn Han, and Kweon Yu

Mol. Cell. Proteomics 2006 5: 1559-1566

Cloned animals developed from somatic cell nuclear transfer (SCNT) embryos are useful resources for agricultural and medical applications. However, the birth rate in cloned animals is very low, and animals that survive often have developmental defects. In this report, the authors examined the differentially regulated proteins in extraembryonic tissue from 26-day-old SCNT porcine embryos to determine the developmental differences between normal and SCNT animals. The tissue from SCNT embryos was abnormally small compared with the control animals. In a proteomic analysis, the authors found that 12 proteins were up-regulated in SCNT tissue, whereas 27 proteins were down-regulated. The up-regulated proteins included apoptosis marker proteins, and the down-regulated proteins included antioxidant enzymes. The results indicate that the low birth rate in cloned animals could be caused by abnormal apoptosis in the extraembryonic tissue during early pregnancy.



SCNT placenta (D) contains more apoptotic cells (yellow and green) than normal placenta (C).

by John D. Thompson, Editor

China Eyes Biotech as Scientists Return after Training in the U.S.

The *San Francisco Chronicle* reports that China, which catapulted into the top ranks of global manufacturing, is now planning to become a biotechnology power.

Kevin Chen, VP of Bioduro Co. Ltd., a San Diego biotech contract research company with a growing operation in Beijing, told *The Chronicle*, "There's no reason the things that make China so powerful in manufacturing cannot apply to knowledge-driven industries." Among the things he referred to is the return to home of Chinese scientists trained in the U.S.

T. J. Deng, Bioduro's director of analytical chemistry, who worked for several years with Wilmington, North Carolina, pharmaceutical company PPD Inc., said one of the factors supporting the growth is that Chinese scientists working in the United States are returning to China in large numbers. "There are huge growth opportunities and a great lifestyle here, and people like me are seeing that," said Deng, who is from the central Sichuan province. "The skills and experience they bring is a crucial element [because] without Western-trained people Chinese outsourcing firms would find it hard to get American clients."

China's biotech strategy mirrors the one it used to become a dominant force in manufacturing. It is using the biotech contract research industry, in which companies take on projects for foreign clients, as a springboard to develop the expertise and technical prowess that will help Chinese companies compete internationally. The goal is to build a world class biotechnology sector that develops a steady stream of proprietary products for the global market.

So far, Western pharmaceutical and biotech firms have not flocked to China. Global pharmaceutical firms will outsource about $3.5 billion in research this year, with less than 5% of that earmarked for China, according to Kalorama Information.

China's main barriers to growth in biotech include concerns over its lax intellectual property rights, language barriers, and quality issues. However, that's not inhibiting companies such as Bioduro from betting big on China's research potential. Officials of the company, which does only contract work, say they can do high quality biotech research for about 30% of what it would cost in the U.S. That is a powerful argument for an industry in which it generally costs about $1 billion to discover and market a new drug.

Puerto Rico Plans Biotechnology Hub to Boost Research, Economic Growth

Speaking at the island's second annual Biotechnology Week, Puerto Rico Governor Aníbal Acevedo-Vilá introduced Puerto Rico as Bio Island and outlined the island's commitment to becoming a global hub for the life sciences and biotechnology.

With more than two million square feet of manufacturing space, Puerto Rico's biotechnology sector has generated more than $3.5 billion in capital investments from the world's largest life sciences corporations. Included in these commitments are global companies such as Amgen ($2 billion), Eli Lilly ($1 billion), Abbott Laboratories ($500 million) and Bristol-Myers Squibb ($200 million).

The island's Bioprocess Training and Development Complex is expected to bring hands-on educational opportunities and new types of cutting-edge research to Bio Island. In addition, a new molecular science facility will focus on research in biology, nanotechnology, genomics, molecular neurosciences, and infectious disease prevention and is a joint venture of the Commonwealth of Puerto Rico, the National Institutes of Health, and University of Puerto Rico. The University of Puerto Rico Cancer Center, a joint venture with the M.D. Anderson Cancer Center, will be the first of its type to study cancer prevention and treatment specifically for the Hispanic community.

"Our status as a U. S. Commonwealth has enabled Puerto Rico to offer the best of all worlds," said the governor. "Puerto Rico has combined the special benefits of an off-shore location with the important protections of doing business in the United States." The island is already the leading pharmaceutical exporter to the United States, with most of the top 20 drugs prescribed in the U. S. manufactured on the island.

For Your Lab/For Your Lab/For Your Lab

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by John D. Thompson, Editor

University of Arizona Seeking Funds for Med School Expansion

The University of Arizona (UA) will ask the state for $13 million more for the expansion of the College of Medicine to Phoenix in an effort to increase the number of medical students and expedite the development process. "This state needs doctors and scientists, but we've still got a long way to go," said Regents President Robert Bulla at a recent Arizona Board of Regents meeting.

Instead of a class of 24 medical students in 2008 as originally planned, 64 students will be admitted to the Phoenix campus during its second year. Students and faculty began moving into the first building completed in the last week of September, according to Regent Gary Stuart. The UA will be asking the state for $6 million specifically for the Phoenix medical school and $1.5 million for the Phoenix pharmacy school. Other funds would go toward the overall biomedical campus.

President Robert Shelton hailed the medical school as "the cornerstone for a much larger effort," referring to the collaboration with ASU and other groups in Phoenix. The entire biomedical campus will house extensions of the colleges of medicine, nursing, pharmacy, and public health. One hundred and fifty students are expected to be enrolled in the Phoenix medical school within five years.

Amaranth Gets $7.5 Million for Development of Bioabsorbable Stents

Amaranth Medical, Inc., a developer of bio-absorbable stents for peripheral and coronary vascular applications, has announced the closing of a $7.5 million Series A financing arranged by Bio*One Capital of Singapore and Charter Life Sciences of Palo Alto, California.

Proceeds from the financing will be used to advance the company's bioabsorbable stents into human clinical trials. The company has developed a bio-absorbable urinary stent, which will enter clinical trials shortly. The company's bio-absorbable vascular stents are expected to enter clinical trials in 2007, initially for the treatment of peripheral vascular disease (PVD). PVD is a major disease in developed countries, afflicting from 12 to 20% of people over age 65 in countries such as the United States, where PVD currently afflicts 8 million people.

Current treatment techniques, including metal stents, are only partially effective, and severe PVD, if not effectively treated, can result in limb amputation. Approximately 200,000 amputations take place in the United States each year as a result of limb ischemia. Metal stents have a history of high fracture rates in the treatment of PVD, and Amaranth's polymeric stents are expected to be much more durable than metal stents. Amaranth's stents also have the advantage of being bio-absorbable, so that stent treatment of the PVD affected vessel can be repeated if the vasculature closes again after time (restenosis), whereas re-treatment of the same portion of the vasculature is extremely difficult or impossible after treatment with a metal stent.

Sanofi Called Best Bet for Bristol-Myers

According to *The Times* of London, Sanofi-Aventis, the world's third-largest pharmaceuticals group, has been tipped as the most likely buyer for Bristol-Myers Squibb, the embattled $48 billion American firm that has signaled it might be willing be listen to offers. Healthcare analysts think that Sanofi is the most credible of a string of potentially interested suitors that includes Pfizer, Schering-Plough, and AstraZeneca.

Bristol-Myers is reeling following the departure in late September of its CEO of five years, Peter Dolan. Following a string of problems at the group during his leadership. Dolan was replaced by board member James Cornelius. Dolan's problems culminated in an ill-fated patent deal with Apotex, a Canadian company that had produced a cheap generic rival to the blockbuster blood-thinner treatment Plavix. Bristol-Myers is Sanofi-Aventis's American sales partner for Plavix.

Bryan Garnier, a health and life sciences analyst at Bryan Garnier, said: "With no permanent new leader, a highly regarded pipeline and an anemic stock price, BMS looks like a perfect takeover target. In our view, Sanofi-Aventis is the most likely suitor, but such a transaction is not realistic before 2007." Buying Bristol-Myers would catapult Sanofi ahead of Pfizer as the world's largest drugs maker, with annual sales of $55 billion.

Career Opportunities

FACULTY POSITION

Baylor University

The Department of Biomedical Sciences at Baylor College of Dentistry, Texas A&M University System Health Science Center, Dallas, is seeking outstanding candidates for a full-time position at the Assistant to Associate Professor level for either the tenure or non-tenure educator track. A PhD in Biochemistry or a related science area is required. Non-tenure educator track applicants must demonstrate considerable teaching experience (preferably in a dental school setting) with a record of excellent student evaluations. Tenure-track applicants must demonstrate the ability to establish an independent research program and procure extramural funding. The successful tenure-track candidate will participate primarily in a team-taught Biochemistry course to first year dental students and graduate students; for a non-tenure educator track candidate, the teaching load will include participation in additional courses. Current departmental research strengths include genetics and developmental biology of the craniofacial region and inflammation/pain. Applications will be reviewed as they are received and will continue to be accepted until the position is filled.

Applications should include a curriculum vitae, summary of current research activities, statement of career goals and teaching philosophy along with the names and contact information of at least three individuals for letters of recommendation to be submitted in electronic format to: Dr. Bob Hutchins, Search Committee Chair, Department of Biomedical Sciences, TAMUSHSC, 3302 Gaston Avenue, Dallas, TX 75246; Email: bhutchins@bcd.tamhsc.edu

ASSISTANT PROFESSOR POSITION

Biochemistry and Structural Biology

Department of Molecular and Cellular Biology, Harvard University

As part of a broad expansion in the Life Sciences at Harvard University, the Department of Molecular and Cellular Biology has an opening for the tenure-track position of Assistant Professor in the fields of **biochemistry and structural biology**. Our department covers a broad range of topics, including molecular biology, cellular biology, developmental biology, neurobiology, molecular evolution, systems biology, biochemistry, and structural biology. The department provides access to many core facilities, including imaging, proteomics, genomics, and bioinformatics. Harvard University is part of the NE-CAT consortium at Argonne National Laboratories.

We strongly encourage applications from women and minority candidates. Applications should include: curriculum vitae, reprints of publications, and a statement of present and future research plans (1-3 pages). Complete applications and three letters of recommendation, solicited by the applicant, should be received not later than 10 November 2006.

Submit applications to:
BSB@mcb.harvard.edu or to:
J. Blackbourn/MCB Search Committee
Department of Molecular and Cellular Biology
Harvard University
7 Divinity Avenue, Rm. 167
Cambridge MA 02138

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ASSISTANT PROFESSOR OF CHEMISTRY

Eastern Connecticut State University

Full-time tenure track position in the Department of Physical Sciences, Eastern Connecticut State University. Ph.D. in Biochemistry, Bio-Analytical Chemistry, Chemistry, or Environmental Chemistry required. Post-doctoral experience preferred with strong evidence of quality teaching. Applicants with expertise in environmental or forensic chemistry are especially encouraged to apply. Teaching expectations include advanced biochemistry and chemistry courses as well as introductory chemistry courses designed in support of the integrative Liberal Arts Core curriculum. The successful candidate will support the department's growing biochemistry program through course development, program assessment and involving undergraduates in research. Send CV, transcript of all graduate work, a statement of teaching philosophy and research interests, documentation of teaching ability, and three current letters of recommendation to Tim Swanson, Department of Physical Sciences, Goddard Hall, Eastern Connecticut State University, Willimantic, CT 06226. Search will continue until position is filled.

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PROFESSOR AND CHAIR

Department of Biochemistry

University of Nebraska-Lincoln

The University of Nebraska-Lincoln is seeking an individual with an outstanding research program and excellent interpersonal skills who can provide energetic and creative leadership for the research, teaching, and public service activities of its **Department of Biochemistry**. A competitive start-up package is available for this full time, 12-month appointment. The Department of Biochemistry is rapidly growing and includes 15 budgeted and 10 affiliated faculty members. The research programs in the Department are currently supported by annual grant and contract awards exceeding $5 million. The Department houses the NIH-funded Redox Biology Center and has established strengths in biomedical research, plant biochemistry, structural biology, bioinformatics, and classical enzymology. The Department is located in the state-of-the-art George W. Beadle Center, which is also the home of the NIH-funded Nebraska Center for Virology, the Plant Science Initiative, the Center for Biotechnology, and key core research facilities. To learn more about the Department, please visit the website http://biochem.unl.edu.

To apply for this position, access the web site http://employment.unl.edu. Search for position number 060852. Complete the faculty academic administrative information form. Attach a letter of application, curriculum vitae, and the contact information for three professional references. Review of applications will begin on November 16, 2006, and continue until the position is filled.

The University of Nebraska is committed to a pluralistic campus community through affirmative action and equal opportunity and is responsive to the needs of dual career couples. We assure accommodation under the Americans with Disabilities Act; contact Linda Arnold at 402-472-3802 for assistance.

Calendar of Scientific Meetings

NOVEMBER 2006

Transcriptional Regulation by Chromatin and RNA Polymerase I I

November 2–6 • Kiawah Island, South Carolina
Organizer: Ali Shilatifard, Saint Louis, University School of Medicine, Email: shilatia@slu.edu

43rd Japanese Peptide Symposium/4th Peptide Engineering Meeting

November 5–8 • Yokohama, Japan
www.peptide-soc.jp/43JPS4PEM.html
E-mail: hmihara@bio.titech.ac.jp

Fall Workshop: The Present and Future of Quadrupole Ion Trap Mass Spectrometry

November 9-10 • Catamaran Resort, San Diego
Program Chairs: Victor Ryzhov and Richard Vachet
For information contact: ASMS
505-989-4517; asms@asms.org; www.asms.org

NIH 4th Symposium — Functional Genomics of Critical Illness and Injury
Surviving Stress: Organ Systems to Molecules

November 13-14 • Bethesda, Maryland,
Preliminary agenda and detailed guidelines for abstracts are available at: www.strategicresults.com/fg4
Register online through Thursday, October 19. There will be no on-site registration.
Deadline for abstract submission is September 8.

Annual meeting of the Society for Glycobiology

November 15-18 • Los Angeles
Contacts: Linda Baum, President; lbaum@mednet.ucla.edu
Kelley Moremen, Secretary; moremen@uga.edu
Website: www.glycobiology.org

The 19th Annual Tandem Mass Spectrometry Workshop

November 29–December 2 • Lake Louise, Alberta, Canada
www.csms.inter.ab.ca/louise.htm
E-mail: mnlouise@telusplanet.net; Ph: 403-335-3707

DECEMBER 2006

Second ISN Special Neurochemistry Conference: Neural Glycoproteins and Glycolipids

December 1-5 • Antigua, West Indies
For information contact:www.isnantigua2006.org/

19th World Diabetes Congress

December 3–7 • Cape Town, South Africa
www.idf2006.org/

American Society for Cell Biology 46th Annual Meeting

December 9-13 • San Diego
Ph: 301-347-9300; Email: ascbinfo@ascb.org
Website: www.ascb.org

JANUARY 2007

Sanibel Conference

January 19-22 • Sundial Beach Resort, Sanibel Island, Florida
Imaging Mass Spectrometry
Program Chairs: Richard Caprioli, Ron Heeren, and Markus Stoeckli, For information contact: ASMS
505-989-4517; asms@asms.org; www.asms.org

MARCH 2007

U.S. HUPO 2007

March 4-8 •Seattle
For information contact:www.ushupo.org
Email: USHUPO@USHUPO.org; Ph: 505-9899-4876

RNAi2007: The Expanding Roles of Small RNAs

March 29-30 • St. Anne's College
Woodstock Road, Oxford, UK
Organizer: Dr. Muhammad Sohail
Ph: +44(0)1865 275231
Fx: +44(0)1865 275259 (Switchboard)
Email: Muhammad.sohail@bioch.ox.ac.uk
www.libpubmedia.co.uk/Conferences/RNAi2007/Home.htm

Association for Biomolecular Resource Facilities

Mar 31-April 3 •Tampa Convention Center, Florida
For information contact:www.faseb.org/meetings/default.htm
Email: ncopen@faseb.org; Ph: 301-634-7010

APRIL 2007

Second Workshop on Biophysics of Membrane-active Peptides

April 1–4 • Lisbon Science Museum, Portugal
The Lisbon Science Museum includes a 19th century lab and lecture room. Conference call for papers: special theme issue of J Pep Sci. Symposia: Membrane-translocating peptides / Cell penetrating peptides, Membrane-permeabilizing peptides / Antimicrobial peptides, Fusogenic peptides, and Structure and Dynamics in peptide-membrane interaction, Plenary lectures: Jöel Schneide: Bioactive properties of peptide surfaces. Robert Hancock: Antimicrobial peptides. Stuart McLaughlin: Electrostatic interaction of basic peptides with acidic lipids in membranes.
Abstract submission, January 15, 2007, Early registration, January 15, 2007, Faculty of Sciences, University of Lisbon, Miguel Castanho, Ph.D.
www.biophysicsmap.com; E-mail: castanho@fc.ul.pt

American Society for Biochemistry and Molecular Biology Annual Meeting in Conjunction with EB2007

April 28–May 2 • Washington, DC
Contact: ASBMB 2007, 9650 Rockville Pike, Bethesda, MD 20814-3008
Ph: 301-634-7145
Email: meetings@asbmb.org
Website: www.asbmb.org/meetings

2nd International Congress on Prediabetes and the Metabolic Syndrome

April 25–28, 2007 • Barcelona, Spain
www.kenes.com/prediabetes2007;
Email: prediabetes2007@kenes.com

MAY 2007

7th International Symposium of the Protein Society

May 12–16, 2007 • Stockholm-Uppsala, CA Sweden
www.proteinsociety.org/pages/page02b.htm
E-mail: cyablonski@proteinsociety.org
Tel.: 301-634-7277

JUNE 2007

55th ASMS Conference on Mass Spectrometry

June 3-7 •Indianapolis
For information contact: ASMS, 505-989-4517
asms@asms.org; www.asms.org

Mitosis Spindle Assembly and Function A FASEB Summer Research Conference in Honor of Dr. B. R. Brinkley

June 9 -14 •Hyatt Grand Champions Resort and Spa, Indian Wells, California
Applications from students and post-docs are especially welcome! For additional information contact the organizers:
Dr. Conly L. Rieder, rieder@wadsworth.org or
Dr. Robert E. Palazzo, palazr@rpi.edu.

76th Annual EAS Congress European Atherosclerosis Society

June 10-13 • Helsinki, Finland
The Congress aims to create a stimulating atmosphere for exchange of the latest scientific and clinical knowledge in the field of atherosclerosis and cardiovascular diseases.
Deadline for submission of abstracts: November 30, 2006
For more information contact:Kenes International, EAS 2007
17, rue du Cendrier; P.O. Box 1726
CH-1211 Geneva 1, Switzerland
Ph: +41 22 908 0488; Fax: +41 22 732 2850
Email: eas2007@kenes.com
Website: www.kenes.com/eas2007

20th American Peptide Symposium—20th Jubilee Peptides for Youth

June 22-27 • Montreal, Canada
For information: www.americanpeptidesociety.com/index.asp?
Email: 20thAPS@UMontreal.ca
Ph: 819-564-5346

JULY 2007

XXIst Congress of the International Society on Thrombosis and Haemostasis

July 6–12, 2007 • Geneva, Switzerland
www.isth2007.com

SEPTEMBER 2007

48th International Conference on the Bioscience of Lipids

September 4–8, 2007 • Turku, Finland
www.icbl2007.abo.fi

5th Euro Fed Lipid Congress

September 16–19, 2007 • Goteborg, Sweden
www.eurofedlipid.org/meetings/goeteborg/index.htm

OCTOBER 2007

34th Annual Conference of the Federation of Analytical Chemistry and Spectroscopy Societies (FACSS)

October 12–18 • Memphis Convention Center, Memphis, TN
Contact: FACSS, PO Box 24379, Santa Fe, NM 87502.
Ph: 505-820-1648; Fax: (505) 989-1073
Email: facss@facss.org; www.facss.org

SEPTEMBER 2008

The 35th Annual Conference of the Federation of Analytical Chemistry and Spectroscopy Societies (FACSS)

Sept. 28 – October 2 • Reno Hilton Hotel, Reno, NV
Contact: FACSS, PO Box 24379, Santa Fe, NM 87502
Ph: (505) 820-1648
Fx: (505) 989-1073
Email: facss@facss.org
FACSS Worldwide Website: www.facss.org

Molecular & Cellular Proteomics (*MCP*) is pleased to announce the 4th special issue dedicated to

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Guest editors: Steven A. Carr and Julio E. Celis

Issue Date: October 2006

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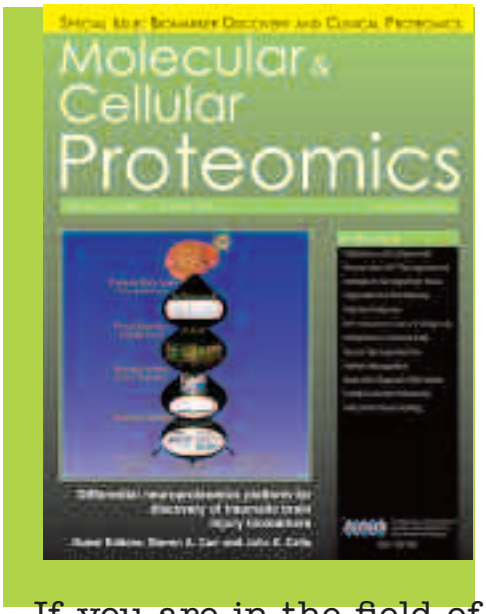
Proteomics is a powerful, cutting-edge discipline that has enormous potential for diagnosis and treatment of human diseases.

This special issue will include articles from presentations at the 2005 Asilomar Conference on "Biomarker Discovery and Clinical Proteomics" organized by Steven Carr and Leigh Anderson, several invited contributions, as well as four research reports selected from direct submissions to the journal. The issue is organized in three sections covering the following topics: 1) biomarkers of disease and conditions, 2) proteomic data analysis, and 3) methodologies.

www.mcponline.org

snapshot of accepted papers

- *Challenges in biomarker discovery.*
- *Protein biomarkers in a mouse model of extremes in trait anxiety.*
- *Novel differential neuroproteomics analysis of traumatic brain injury in rats.*
- *Proteomics in clinical trials and practice: present uses and future promise.*
- *Proteomics of breast cancer: principles and potential clinical applications.*
- *Proteomic based development of biomarkers in cardiovascular disease: Mechanistic, clinical and therapeutic insights.*
- *A platform for experimental pattern recognition.*



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ASBMB

American Society For Biochemistry And Molecular Biology