JUNE 2006

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ASBMB Today

Constituent Society of FASEB

AMERICAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY

NEW FEATURE:
See page 14 for ASBMB journal highlights

How Protein Folding Controls Cellular Routing

page 12

Schedule Announced

The ASCB 46th Annual Meeting

December 9-13, San Diego, CA

Mary Beckerle, President ■ Anthony Bretscher, Program Chair ■ Arshad Desai, Local Arrangements Chair

KEYNOTE SYMPOSIUM

Saturday, December 9

Frontiers in Cell Biology—6:00 pm

Thomas R. Cech, Howard Hughes Medical Institute

SYMPOSIA

Sunday, December 10

Coordination of Adhesion and Migration—8:00 am

Denise Montell, Johns Hopkins Medical School
Clare Waterman-Storer, The Scripps Research Institute
Kenneth Yamada, National Institute of Dental & Craniofacial Research/NIH

Deciphering Evolution—10:30 am

Sean Carroll, University of Wisconsin–Madison/HHMI
Eric Jarvis, Duke University Medical Center
David Kingsley, Stanford University School of Medicine/HHMI

Monday, December 11

Mechanisms in Mitosis—8:00 am

Rebecca Heald, University of California, Berkeley
Lucille Shapiro, Stanford University School of Medicine
Ronald D. Vale, University of California, San Francisco/HHMI

Developmental Decisions—10:30 am

Hans Clevers, Netherlands Institute for Developmental Biology
Elliot Meyerowitz, California Institute of Technology
Susan Strome, Indiana University

Tuesday, December 12

Membrane Assembly and Dynamics—8:00 am

Gillian Griffiths, University of Oxford
Janet Shaw, University of Utah
Marino Zerial, Max Planck Institute of Molecular Cell Biology & Genetics

From Cellular Mechanisms to Therapeutic Intervention—10:30 am

Susan Lindquist, Whitehead Institute for Biomedical Research
Christine Seidman, Harvard Medical School/HHMI
Xiaodong Wang, University of Texas Southwestern Medical Center/HHMI

Wednesday, December 13

Functional Networks—8:00 am

Susan Mango, University of Utah
Kevan Shokat, University of California, San Francisco
Tian Xu, Yale University School of Medicine/HHMI

Stem Cell Biology—10:30 am

George Q. Daley, Children's Hospital Boston
Elaine Fuchs, Rockefeller University/HHMI
Margaret Fuller, Stanford University School of Medicine

MINISYMPOSIA

Apoptosis
Eileen White, Rutgers University
Junying Yuan, Harvard Medical School

Applications of Biosensors
Atsushi Miyawaki, RIKEN Brain Science Institute
Alice Ting, Massachusetts Institute of Technology

Cancer Mechanisms
Lisa Maria Coussens, University of California, San Francisco
Mary J.C. Hendrix, Children's Memorial Research Center/Northwestern University Feinberg School of Medicine

Cell Cycle
Mary Dasso, National Institute of Child Health & Human Development/NIH
Jonathon Pines, The Wellcome Trust/Cancer Research UK

Cell Migration
Diane L. Barber, University of California, San Francisco
Gregg G. Gundersen, Columbia University College of Physicians & Surgeons

Computational Applications in Cell Biology
Douglas A. Lauffenberger, Massachusetts Institute of Technology
Alex Mogilner, University of California, Davis

Cytoskeleton, Adhesion and Disease
Kathleen J. Green, Northwestern University Feinberg School of Medicine
Alpha S.K. Yap, University of Queensland

ECM and Cell Signaling
Jean E. Schwarzbauer, Princeton University
Christopher Turner, SUNY Upstate Medical University

Endo- and Exocytosis
Todd Graham, Vanderbilt University
Margaret Scott Robinson, CIMB/The Wellcome Trust

Epigenetics and Chromatin Remodeling
Peggy Farnham, University of California, Davis
Andrew Feinberg, Johns Hopkins University School of Medicine

Epithelial Organization and Morphogenesis
Andrea I. McClatchey, Massachusetts General Hospital
Ulrich Tepass, University of Toronto

GTPases in Cellular Traffic
Francis Barr, Max Planck Institute of Biochemistry
Shou-ou Shan, California Institute of Technology

Host Pathogen Interactions
Jorge Galan, Yale University School of Medicine
Francoise Gisou Van Der Goot, University of Geneva Medical School

Imaging
J. Richard McIntosh, University of Colorado
Eva Nogales, University of California, Berkeley/HHMI

Immune Cell Adhesion and Recognition
Andrey Shaw, Washington University School of Medicine
Colin Watts, University of Dundee

Intermediate Filaments and Disease
Don W. Cleveland, University of California, San Diego
Colin Stewart, NCI-Frederick

Kinetochores and Centrosomes
Michel L.F. Bornens, Institute Curie, Paris
Peter Todd Stukenberg, University of Virginia School of Medicine

Life at the Microtubule Plus End
Anna Akhmanova, Erasmus University
Kevin Vaughan, University of Notre Dame

Mechanisms of Actin Dynamics
Bruce Lane Goode, Brandeis University
Dorit Hanein, The Burnham Institute

Mechanisms of Cell Polarity
Patrick Brennwald, University of North Carolina at Chapel Hill
Chris Q. Doe, University of Oregon/HHMI

Membrane Traffic in Disease
Esteban Carlos Dell-Angelica, University of California, Los Angeles School of Medicine
Daniel Klionsky, University of Michigan

Microtubule Motors
Erika L.F. Holzbaur, University of Pennsylvania
Claire E. Walczak, Indiana University

Motile and Sensory Cilia
Kathryn Anderson, Memorial Sloan-Kettering Cancer Center
Elizabeth F. Smith, Dartmouth College

Myosin-based Movement
Folma Buss, Cambridge University
Arturo DeLozanne, University of Texas

Neural Degeneration and Regeneration
Zhigang He, Harvard University
Stephen Strittmatter, Yale University School of Medicine

Nuclear Pore and Traffic
Michael P. Rout, Rockefeller University
Katherine S. Ullman, University of Utah

Organelle Inheritance and Maintenance
Liza A. Pon, Columbia University College of Physicians & Surgeons
Michael Schrader, University of Marburg

Regulation of the Cytoskeleton
Keith W.T. Burridge, University of North Carolina at Chapel Hill
Anne J. Ridley, Ludwig Institute for Cancer Research

RNA and Development
Oliver Hobert, Columbia University College of Physicians & Surgeons/HHMI
Roy Parker, University of Arizona/HHMI

Signaling in Development
Marcos Gonzalez-Gaitan, Max Planck Institute of Molecular Cell Biology & Genetics
Alexandra Joyner, New York University School of Medicine/HHMI

Stem Cells
M. Kathryn Barton, Carnegie Institution of Washington
Linheng Li, Stowers Institute of Medical Research

Synapse Assembly and Plasticity
Ann Marie Craig, University of British Columbia
Nancy Y. Ip, Hong Kong University of Science & Technology

For more information, contact the ASCB at (301) 347-9300, ascbinfo@ascb.org or www.ascb.org.

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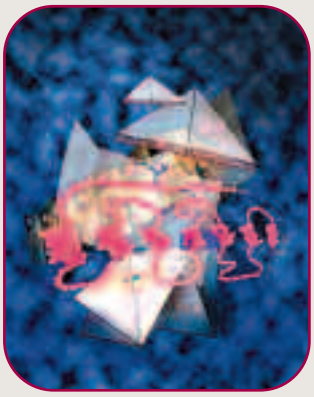

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ASBMB 100 YEARS

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Comments

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Exploring the Biochemical Basis of Evolution?

To the Editor:

In his last book, America's leading evolutionist, the recently deceased Stephen Jay Gould, lamented his "relative ignorance" of "the nature of genomes" and the "realm of the smallest."[1] Indeed, it is possible that few evolutionists are fully aware of recent biochemical advances that are of great relevance to evolutionary theory.[2] Thus, at face value, we should have welcomed Professor Michael Behe's book, "Darwin's Black Box: The Biochemical Challenge to Evolution" as one of the few written on this subject by a professional biochemist.[3] Unfortunately, as Professor Sjoerd Bonting points out in the April issue of ASBMB Today, Behe's arguments are severely flawed. However, Bonting does not point out that Behe also ignores many of the issues that evolutionists have long considered fundamental—issues to which biochemistry and molecular biology have made major contributions. These include the questions: what are species, how do they usually originate, and does this origination occur genetically or non-genetically? Indeed, had Behe fully considered these issues his title might more appropriately have been "Darwin's Black Box: The Biochemical Basis of Evolution."

Donald R. Forsdyke,
Department of Biochemistry,
Queen's University, Kingston,
Ontario, Canada K7L3N6

(1) Gould, S. J. (2002) The Structure of Evolutionary Theory. Harvard University Press.
(2) Forsdyke, D. R. (2006) Evolutionary Bioinformatics. Springer, New York.
(3) Behe, M. J. (1996) Darwin's Black Box: The Biochemical Challenge to Evolution. The Free Press, New York.

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.

Reference Omitted

A reference was omitted from a paragraph in Professor Kendric C. Smith's letter, "Ethics in Science: what has happened to it?" in the May issue of ASBMB Today. The paragraph and the reference in question follow:

"Recombinational DNA repair accounts for 50% of survival after UV irradiation, and excision repair accounts for 50%, i.e., not the 100% that some authors would have you believe (for a review on recombinational DNA repair, and the citation of some of these bad papers, see reference).

"Smith, K.C., Recombinational DNA Repair: the ignored repair systems. BioEssays 26:1322-1326 (2004)."

Thanks for the Memories!

Dr. Judith Bond

June 2006 is busting out all over, and is the last month of my ASBMB Presidency. The two years have flown by and are chock full of great memories. It was a very great pleasure to be serving in the role of President in these transitional years between the first and second century of the Society and the *JBC*. It has reaffirmed for me what a great tradition/discipline we belong to, as members of the Society and as practitioners/devoted members of the discipline, and how fundamentally important our science is to advances in medicine, agriculture, the environment, safety, and the quality of life worldwide.

ASBMB Finance Committee. From left to right are Judith Bond, Director of Publications Anthony Pegg, Larry Solomonson, Treasurer-Elect Merle Olson, ASBMB Comptroller Steve Miller, JBC Editor Herbert Tabor, Treasurer Kenneth Neet, Ken Mann, Gerry Hart, President-Elect Heidi Hamm, and ASBMB Executive Officer Barbara Gordon.

It is difficult to envision how scientific publications and communications/information exchange will evolve in the next century. The information and computer sciences are changing so rapidly, that to keep up with the technology is challenging, no less predicting years ahead. It seems certain that paper communications are dwindling, and that globalization of all aspects of our discipline is growing.

It is clear that our Society and our Journals are in good hands. We have a strong and committed staff and scientific leadership. We go into the next century with Heidi Hamm as President and a devoted Council.

Our Journals are of the highest quality with outstanding editorial boards and Herb Tabor, Editor, and Bob Simoni, Deputy Editor, of *JBC*, Ed Dennis, Editor, and Joe Witzum, Deputy Editor, of *JLR*, and Ralph Bradshaw and Al Burlingham, Co-Editors, of *MCP* at the helm.

We have a host of committees, composed of volunteers and staff that work behind the scenes to fulfill our missions. The Program and Meetings Committees have worked to organize high quality annual and small meetings. The Educational and Professional Development and Minority Affairs Committees support activities for the next generation of scientists and for diversity. The Public Affairs Advisory Committee deals with issues in the public arena that affect the lives of our members. The Publications Committee handles the policies and overarching issues of our Society's journals. The Awards and Nominating Committees select individuals for recognition and leadership positions in the Society. One of the Committees that has been working to sustain the Society and Journals in these changing times is the Finance Committee. Ken Neet has done an excellent job of chairing this committee for the last six years, and this year he turns over the reigns to Merle Olson.

ASBMB Council. From left to right in back are William R. Brinkley, Linda J. Pike, Juliette Bell, Suzanne R. Pfeffer, J. Ellis Bell, Kuan-Teh Jeang, Dennis R. Voelker, William Sly, Edward A. Dennis, William L. Smith, Joan Conaway, F. Peter Guengerich, and Anthony E. Pegg. In front, from left, are Robert Copeland, Kenneth Neet, Judith Bond, Heidi Hamm, Merle S. Olson, and Herbert Tabor.

It is true that we have many challenges to face, for example, procuring funding for research, educating the public and our political leaders about the importance of science and science education, and understanding and curing diseases. I am optimistic that with the brains, energy and vision of biochemists and molecular biologists we will find solutions, continue to be creative, make advances, and contribute to the quality of life through our science. So, thanks to all for a very memorable two years, and best wishes for the future.

Judith S Bond
President, ASBMB

by Peter Farnham, CAE, ASBMB Public Affairs Officer

Open Access Bill Introduced in Senate—

A Senate bill requiring most scientists to submit their federally funded papers to agency databases within six months of publication was introduced May 2. The sponsors of the legislation, the Federal Research Public Access Act of 2006, are Senators John Cornyn (R-TX) and Joe Lieberman (D-CT).

The bill greatly expands the public access concept in government. Currently, only the National Institutes of Health (NIH) has an active policy regarding public access to federally-funded research (although language to this effect has been on the books at almost all federal agencies for many years). The NIH's current policy is that scientists are encouraged (not required) to submit an electronic copy of any final manuscript that has been accepted by a peer-reviewed journal and funded in whole or in part by NIH, to the NIH research database, PubMed Central, and make it publicly accessible as soon as possible, but no later than within 12 months following publication. The Cornyn/Lieberman bill, S.2695, makes a number of important changes in this policy.

First, the bill would make the submission policy mandatory. A scientist would be *required* to submit an electronic copy of his or her final manuscript to a digital library, either to one maintained by the funding agency or to another repository that permits free public access. Further, the work must become publicly available after six months, rather than twelve. Finally, the bill would expand the number of federal agencies affected to all those that funded at least $100 million year in extramural research.

Both Cornyn and Lieberman made public statements about the bill. "Our legislation," Cornyn said, "is a simple, common sense approach that will advance the public's access to the research it funds." Cornyn said the bill helps three groups as well as the general public. First, patients can find articles discussing the latest information on treatment and prognosis for their diseases. Second, students will have access to research articles to complete assignments and further their studies. Finally, researchers' findings will be disseminated more quickly and broadly, thus sparking further discovery and innovation.

"We wrote this legislation to give taxpayers access to scientific discoveries and advancements that they are paying for," said Lieberman. "The goal is to share information, avoid duplication of effort and help spur new ideas which down the road can mean new treatments and cures for researchers, medical professionals and patients in Connecticut."

Mila Becker, Director of Government Relations at the American Society for Hematology, said that "The bill is vast in terms of who it would affect," but she and other observers are not sure what immediate impact it is going to have, as the Senate has many other, more pressing issues on its plate at this time. But Becker noted that the bill having been introduced "proves the whole movement toward public access is alive and well."

Martin Frank is the coordinator of DC Principles for Free Access to Science, a coalition of about 50 scientific publishers (including ASBMB) that advocates on public access issues. Frank spoke with *ASBMB Today* about the many problems associated with the bill. "The bill does not take into account the potential impacts upon society publishers that have served as long-standing filters and evaluators of scientific research, including the ability to provide adequate and unbiased peer review."

A reduced ability for some publishers to innovate may be a further result of the bill, Frank said. "Many of the non-profit publishers in biomedical research were in the forefront in putting publications on-line. Some of the publishers of research in fields other than biomedical are not as advanced as the biomedical publishers, and will thus have less money to spend on innovation."

Frank also raised cost issues. "Mandatory submission of manuscripts and publications within six months will negatively impact quarterly and 'niche' journals because of difficulties associated with recovery of subscriptions costs. The bill also has the potential for shifting costs from reader to the author, with an associated diminution of funds the author has available for research."

Finally, as many as 11 federal agencies will be required to arrange for their funded research to be deposited in some type of repository, either set up by the agencies themselves or in another suitable one. The additional infrastructure needed will eat up precious resources that could be better used to support actual research.

But Watch House Appropriations

The bill has been referred to the Homeland Security and Government Affairs Committee. Senator Lieberman is the ranking minority member there.

Watch Events in the House

In spite of the attention that the Cornyn-Lieberman bill has garnered in the press, most observers do not expect it to make much progress toward becoming law any time soon. However, an equally if not more important move may be afoot in the House. Rep. Ernest Istook (R-OK), following his pattern on this issue in recent years, has reportedly prepared language to include in the report that will accompany the 2007 Labor/HHS appropriations bill when it is reported out of subcommittee.

We have also learned that the chairman of that subcommittee, Rep. Ralph Regula (R-OH), has developed an interest in mandating public access within a shorter amount of time than the current 12-month policy at NIH. We understand that Regula is also very bothered by the low compliance rate (about 4 %) at NIH.

Another player in the debate is, of course, NIH Director Elias Zerhouni. He has expressed public sympathy with scientific publishers and has stated his support for a 12-month limit (the original public access policy promulgated by NIH called for a six-month limit, subsequently expanded to 12 in the final version). We also understand that there is more sympathy in the Senate for the 12-month limit than in the House, in spite of the Cornyn-Lieberman legislation having been introduced.

GOP Moderates, with Community Support, Force Changes in Budget Resolution

Although the House has still not approved a 2007 budget resolution, there will be a minimum of $5 billion more for health and education programs than there would have been under the President's February proposal, thanks to the efforts of a determined group of moderate Republicans led by Rep. Mike Castle (R-DE) and Nancy Johnson (R-CT)—and also due to the public and vocal support of hundreds of affected groups, including ASBMB.

As we reported in last month's issue, GOP moderates have continued to refuse to support any budget resolution that does not include at least $7.1 billion in additional health and education spending over the President's proposal. Since then, ASBMB and almost 800 other organizations have signed a letter to the House leadership urging support for Castle's position. As the letter notes, "While our organizations represent a wide array of domestic priorities, we are united in our effort to advance the bipartisan goal of adding $7 billion in discretionary funding for health, education, labor enforcement, job training and social services programs as the budget process moves forward. On behalf of our millions of constituents, we strongly urge you to provide at least $7 billion in additional federal support…This is essential to sustain the well-being and prosperity of our nation." ASBMB also supported a broader FASEB effort to contact society members in key congressional districts to urge support for the Castle position.

The letter and constituent contacts, along with the pressure two dozen dissident Republicans can bring to bear on a new leadership team in a closely divided House, apparently worked. The leadership met with Castle in early May and offered to move $5.1 billion out of defense and homeland security spending and into social spending, of which more than $4 billion would be included in the Labor/HHS appropriations bill (which funds the National Institutes of Health).

Castle issued a statement calling the leadership's offer a "significant step forward," and a "refreshing approach." However, he remains committed to a $7.1 billion increase for health, education and community programs, which he noted "would take us to FY06 levels while also accounting for 2 % inflation. It is my hope we can continue to negotiate the difference that remains so we can adequately fund these important programs."

ASBMB members in Delaware are urged to contact Rep. Castle and thank him for his efforts.

Stem Cell Researchers' Dilemma: Seek Vote Now or Wait for New Congress

May 24, 2006 marked the one year anniversary of the landmark passage of the Stem Cell Research Enhancement Act (H.R. 810) by the U.S. House of Representatives. Many stem cell advocates are using this date to build momentum for passing the Senate version of the House bill, or the Castle-DeGette bill, as it is often referred to, in recognition of its chief sponsors, Representatives Mike Castle (R-DE) and Diana DeGette (D-CO). The Senate version (S.471) was introduced by Senators Arlen Specter (R-PA) and Tom Harkin (D-IA), and as of this writing has 40 additional bipartisan sponsors. The legislation would expand federal funding eligibility to existing stem cell lines, created from discarded in vitro fertilized embryos, regardless of when they were derived. It seems likely that if this bill were introduced in the Senate, it would pass. However, there are a number of extenuating circumstances that may make bringing the Senate bill to the floor a detrimental scenario for the research community.

The first, and sadly perhaps best, scenario is that the bill passes and is vetoed immediately by President George W. Bush. The President has threatened to veto any stem cell legislation that expanded his existing federal policy, and it is almost certain he would carry through on that threat. It remains to be seen if the House and Senate would have enough votes to override a veto, but with very few days in the legislative calendar in this election year, and a number of pressing issues facing the Congress, including appropriations, immigration, and the continuing war in Iraq, it is improbable that stem cells will remain a top priority.

What remains perhaps a greater danger to the research community is that the Senate stem cell bill would be sent to the floor as a package of bills. Among these would be a bill opposing somatic cell nuclear transfer (SCNT) for the purposes of reproductive cloning and maybe research, as well, and a bill recently introduced by Senator Sam Brownback (R-KS), the Human Chimera Prohibition Act of 2006 (S.1373). This bill would criminalize a number of experiments involving human-animal chimeras, including the production of hybrid embryos through hetergenuous gamete fertilization and production of "a non-human life form engineered such that it contains a human brain or a brain derived wholly or predominantly from human neural tissues." Although many in the scientific community would undoubtedly agree there exist ethical boundaries in chimeric science that should not be crossed, some provisions of the bill could prohibit existing lines of inquiry. These include the introduction of a human nucleus into a non-human oocyte and the production of human gametes by a non-human species. The latter has been proposed as a potential method for solving the SCNT related problem of having enough human eggs to make SCNT a feasible technology.

Unfortunately, while FASEB and the rest of the research and stem cell advocacy community have done an excellent job of educating Congress and the public about the importance of embryonic stem cells, education about chimeras remains an uphill battle. The co-mingling of human and animal cells inspires a frightening vision straight out of science fiction in many non-scientists, and explaining the details of the science is proving difficult. President Bush himself, in his State of the Union address, called for passage of "legislation to prohibit the most egregious abuses of medical research…creating human-animal hybrids." If the Brownback bill comes to the floor, accompanying the stem cell bill, both of them may pass, but only the chimera prohibition will survive the Presidential veto. This situation leaves stem cell advocates in an untenable situation: push for the Senate stem cell bill in this Congress, knowing it may result in unintended negative consequences; or let it go for now, and begin from square one in passing the House bill in the next Congress following the election. A year ago we were celebrating a hard won victory with passage of the Castle-DeGette bill; this year's anniversary will be far more bittersweet.

Deceased Members

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

David W. Allmann
Laurence Cezanne
Nicholas R. Cozzarelli
Bernard J. Finkle
Lawrence Grossman
Beverly M. Guirard
Robert J. Kadner
Andre S. Meyer
Cecile M. Pickart
Martin A. Rizack
Hugh Robertson
John R. Sokatch
Kay Tanaka
Alvin Taurog
Harold Werbin
William L. Williams

JBC Institutes Submission Fee

After considerable thought, the leadership of the ASBMB has decided to introduce a submission fee for the *Journal of Biological Chemistry*. The $60 fee will be instituted in September 2006. The intent is to offset the cost of processing submitted manuscripts. The fee will be charged at the time the manuscript is submitted to the Journal and must be paid before the manuscript is reviewed.

The decision to institute the submission fee was in part due to the changing financial profile of our scientific publications. In addition, there has been a recent decrease in the percentage of manuscripts accepted (down from about 45% to near 30%) and an increase in production costs. On the basis of financial projections and the options we have available for financial

stability, as well as discussions with the JBC Associate Editors, the ASBMB Publications Committee, and the ASBMB Council, we feel the decision to institute the $60 submission fee is warranted. The fee is miniscule compared to the cost of doing the research, and all are aware these days that there are considerable costs for handling manuscripts, even those that do not meet the JBC Guidelines.

The ASBMB staff will continue to examine the pricing, subscriptions, marketing and advertising of our journals. There may well be ways to increase revenues and all avenues to do this will be explored. We will further investigate the cost structure for our journals and possibilities for going forward, and bring new options to the table for the long-range fiscal health of our journals and Society.

Judith Bond, ASBMB President
Kenneth E. Neet, ASBMB Treasurer
Nancy J. Rodnan, Director of Publications

Cell Division Rewind Button Found

Scientists have found a way to reverse the process of cell division, previously thought to be unstoppable. The discovery could have important implications for the treatment of cancer, birth defects and numerous other diseases and disorders. The findings appear in the April 13 issue of the journal *Nature*.

"No one has gotten the cell cycle to go backwards before now," said principle author Gary J. Gorbsky, a scientist with the Oklahoma Medical Research Foundation (OMRF). "This shows that certain events in the cell cycle that have long been assumed irreversible may, in fact, be reversible."

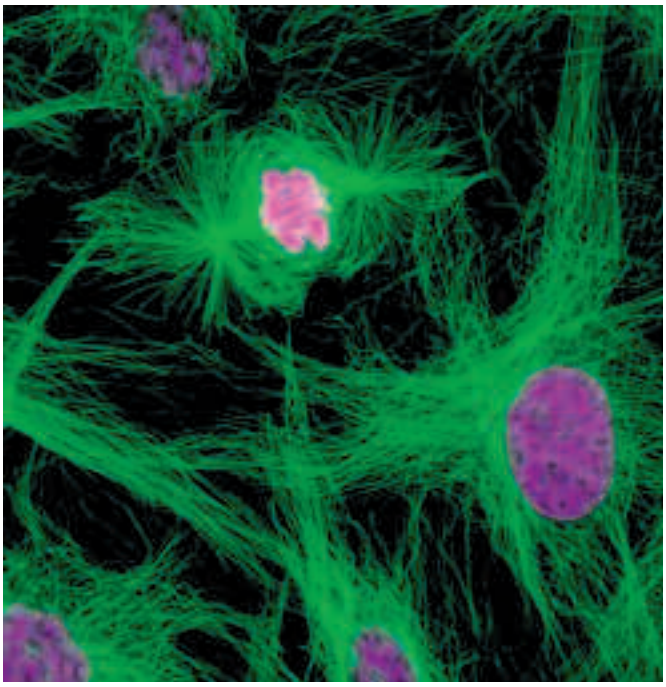
Gorbsky and his OMRF colleagues were able to block the degradation of a protein called cyclin B which normally plays a key role in instigating cell division. This caused the cell cycle to go backwards, sending duplicate chromosomes back to the center of the original cell, which had been thought impossible, rather than continuing the division process. This appeared to be because cyclin B was still present in the cell when, under natural circumstances, it should not be.

But the researchers had to act at a particular point, or the intervention failed to reverse cell division. This suggests many other factors involved in regulating cell division.

"Our studies indicate that the factors pointing cells toward division can be turned and even reversed," Gorbsky said. "If we wait too long, however, it doesn't work; so we know that there are multiple regulators in the cell division cycle. Now we will begin to study the triggers that set these events in motion."

The findings may prove important to controlling the development and metastasis of certain cancers. It also holds promise for the prevention and treatment of birth defects and a wide variety of other conditions.

"Dr. Gorbsky's results provide elegant proof that the cell cycle must be precisely controlled," said Dr. Rodger McEver, OMRF vice president of research. "Now he and his lab can work toward developing innovative methods to probe and better understand the complex process of cell division."



Xenopus S3 tissue culture cells labeled for microtubules (green) and for DNA (magenta). The cell in the upper middle area is in mitosis. The culture was treated 10 min earlier with flavopiridol, which is causing the cell to begin to exit mitosis.

Photograph courtesy of Joseph Mills.

Dr. Gary J. Gorbsky

ASBMB member Gary J. Gorbsky holds the W.H. and Betty Phelps Chair in Developmental Biology and heads the Molecular, Cell and Developmental Biology (MCDB) Research Program at OMRF. He is also Adjunct Professor in the Department of Cell Biology and a member of the Cancer Institute at the University of Oklahoma Health Science Center. He obtained his B.S. from the College of William and Mary in 1976, and both his M.S. and Ph.D. from Princeton University in 1978 and 1982, respectively. Gorbsky carried out his PhD studies with Dr. Malcolm Steinberg and his postdoctoral research was done with Dr. Gary Borisy and Dr. Hans Ris at the University of Wisconsin. He became Assistant then Associate Professor in the Department of Cell Biology at the University of Virginia, and then went on to become Professor of Cell Biology at the University of Oklahoma Health Sciences Center. In 2003 he joined the OMRF as Program Head of MCDB.

Viral Protein Helps Infected T-cells Stick to Uninfected Cells

New research shows that a protein made by a cancer virus causes infected immune cells to cling to other immune cells, enabling the virus to spread.

The virus, the human T lymphotropic virus type 1 (HTLV-1), is transmitted mainly when infected cells known as T lymphocytes, or T cells, touch uninfected T cells.

The finding helps explain how this cell-to-cell transmission happens. It suggests that an HTLV-1 protein known as p12 activates infected T cells and causes them to become sticky and adhere to other T cells.

The greater stickiness happens because the p12 viral protein causes special adherence proteins found on the surface of T cells to cluster in large groups—something that normally happens when T cells touch to communicate with one another during an immune response.

The findings also suggest that a drug that inhibits the p12 protein might also help prevent HTLV-1 transmission.

The research, published in the May issue of the *Journal of Immunology*, was led by scientists with The Ohio State University Comprehensive Cancer Center and the OSU College of Veterinary Medicine.

"This study indicates that the p12 protein plays an important role in programming infected cells for cell-to-cell transmission," says principal investigator Michael D. Lairmore.

"It shows that this virus takes advantage of something that T cells do normally; but, in this case, the virus is stimulating the interaction with other T cells rather than a normal immune response."

HTLV-1 infects an estimated 15 to 25 million people worldwide. About 5 percent of those infected develop adult T cell leukemia or lymphoma (ATLL), an aggressive disease characterized by a long latent period and the proliferation of T cells. The infected cells are spread from person to person during sexual activity and by blood and breast milk.

In the body, the HTLV-1 mainly targets immune-system cells known as CD4 T lymphocytes. These immune cells coordinate immune responses in part through physical contact with other immune cells. The cells adhere to one another using a protein known as LFA-1, which is found on the cells' surface.

In this study, Lairmore and his collaborators examined the influence of the p12 protein on LFA-1 adhesion. The researchers compared cells infected with HTLV-1 that lacked the p12 protein to cells that were infected by normal HTLV-1.

They found that the p12 protein not only activated the T cells, but caused the cells infected with normal HTLV-1 to have far greater adherence than cells infected with viruses that lacked p12 in a standardized adherence test.

In addition, they showed that the greater adherence did not occur because the infected cells made more of the LFA-1 protein, but rather because already existing LFA-1 protein molecules gathered into large clusters on the cell surface.

(LFA-1 proteins float in the cell membrane like buoys in semisolid gelatin. They can move across the surface of the cell and form clusters when directed to do so by signals from within the cell.)

"Our study is the first to show that HTLV-1 p12 not only enhances the activity of infected T cells, but that it promotes the spread of the virus from cell to cell by causing LFA-1 receptors to cluster on the cell surface," Lairmore says.

ASBMB member Michael D. Lairmore is currently Chairperson of the Department of Veterinary Biosciences and the Associate Director of Basic Sciences for The Ohio State University, Comprehensive Cancer Center. He earned his D.V.M. from the University of Missouri (1981) and his Ph.D. from Colorado State University (1987). His studies led to his appointment to the National Centers for Disease Control (CDC) in Atlanta, Georgia in 1987. Lairmore joined the OSU faculty in 1990, and rose to the rank of full professor while establishing an international reputation as a researcher and educator. He has authored or co-authored over 121 scientific publications on topics that include novel vaccine approaches to overcome barriers to effective immune responses against retroviruses, clarification of which target cells express the CD4 AIDS receptor in the human placenta, and the first report of an infectious molecular clone of HTLV-1. Lairmore is the recipient of numerous academic awards including the Pfizer Award for Research Excellence, National Omega Tau Sigma Career Award, Phi Zeta Honor Society and Research Award, and the David White Research Award. He is a member of several editorial boards including *The Journal of Virology*.

Dr. Michael Lairmore

Inhibition of Iron-Metabolizing Enzyme Reduces Tumor Growth

By Nicole Kresge, Staff Science Writer

A report in the April 21 issue *Journal of Biological Chemistry* (281, 11332-11346) shows that inhibition of heme oxygenase-1, an enzyme involved in iron metabolism, reduces Kaposi sarcoma tumor growth. This discovery, which was featured as a Paper of the Week, could result in the production of new drugs to treat this and other viral cancers.

Kaposi sarcoma is the most frequent tumor in AIDS patients and is caused by infection of the patients with the Kaposi sarcoma-associated herpes virus. The Kaposi sarcoma virus genome contains sequence that encodes for a protein called viral G protein-coupled receptor (vGPCR) that plays a key role in the development of tumoral lesions.

Interestingly, a study done in early 2004 showed that the cellular production of a protein called heme oxygenase-1 could be turned on by the Kaposi's sarcoma-associated herpesvirus. Heme oxygenase-1 is an enzyme that is expressed in spleen and liver and is responsible for breaking down heme, a molecule that consists of an iron atom surrounded by a large ring of other atoms. Further evidence of the connection between heme oxygenase-1 and the Kaposi's sarcoma virus came when elevated levels of the protein were detected in biopsy tissue from oral AIDS-Kaposi's sarcoma lesions.

"Taking into account the predominant function of vGPCR in Kaposi's sarcoma and the elevated expression of heme oxygenase-1 observed in Kaposi's sarcoma lesions, we decided to study whether vGPCR could increase heme oxygenase-1 expression and if so, to explore the putative role of the enzyme in vGPCR-dependent transformation," explains study author Maria Julia Marinissen of the Universidad Autonoma de Madrid.

Marinissen and her colleagues found that vGPCR does indeed increase production of the heme oxygenase-1 protein and the RNA that codes for it. They also discovered that mice with tumors that were given specific pharmacological inhibitors that blocked heme oxygenase-1 activity showed a significant reduction in vGPCR-induced tumor growth without apparent side effects.

"Considering that heme oxygenase-1 is overexpressed in human Kaposi's sarcoma lesions, the inhibition of intratumoral heme oxygenase-1 activity by currently available drugs can represent a new anticancer tactic in the treatment of Kaposi's sarcoma and may be of potential clinical interest after more extensive investigation," says Marinissen. "The inhibitor that we used in this study is a tin-protoporphyrin. A recent clinical trial showed that the inhibitor can be administered to newborns at any time point in the progression of postnatal hyperbilirubinemia to rapidly and predictably block heme degradation and prevent severe jaundice without significant short- or long-term side effects· This is very important because it shows that the inhibitor has been successfully used in human clinical trials to treat diseases in which heme oxygenase-1 is involved."

SPOTLIGHT ON ASBMB MEMBERS

Dale J. Benos Receives UAB Teaching Award

This past May, ASBMB member Dale J. Benos, Professor and Chair, Department of Physiology and Biophysics at the University of Alabama at Birmingham (UAB), was awarded the 2006 UAB Presidential Award for Excellence in Teaching.

The President's Awards for Excellence in Teaching were established by former UAB President Charles A. McCallum in 1990 to recognize exceptional accomplishments in teaching. Recipients are chosen from each of UAB's 12 schools and the Joint Health Sciences Departments. Each faculty member so honored receives a cash award and a Steuben apple that stands as a symbol of his or her achievement. Students, faculty and alumni make the nominations for the award.

According to the UAB Reporter, Benos has demonstrated a commitment to teaching that ranges from the elementary classroom to students pursuing doctoral degrees. He is credited with excellence in teaching, research and service, and also commended for his ability and willingness to help develop courses that address specific needs during his nearly 21 years at UAB.

Dr. Dale J. Benos

Benos, who has been an ASBMB member since 1998, is also currently an Associate Editor for the *Journal of Biological Chemistry.*

New Hybrid Virus Provides Targeted Molecular Imaging of Cancer

Researchers at The University of Texas M. D. Anderson Cancer Center have created a new class of hybrid virus and demonstrated its ability to find, highlight, and deliver genes to tumors in mice.

Researchers say the advance, reported in the April 21 issue of the journal *Cell*, is potentially an important step in making human cancer both more visible and accessible to treatment.

"In tumor-bearing mice, we show that this hybrid virus can target tumors systemically to deliver an imaging or therapeutic gene," says co-leader of the study, Renata Pasqualini, Ph.D., professor of Medicine and Cancer Biology. "The signal is specific only to tumors, so one can monitor drug effectiveness at the molecular level."

The team created and characterized the hybrid viruses by combining genetic elements and biological attributes of an animal virus (adeno-associated virus, or AAV) with those of a bacterial virus (phage). Unlike animal viruses that infect mammalian cells, bacterial viruses have evolved to infect only bacterial hosts. The paper shows how particles of the hybrid virus, called AAV phage or AAVP, can serve as a vehicle for targeted delivery of genes to experimental tumors in mice and to the tumors' blood vessel supply, providing a strategy for finding tumors and genetically marking them for imaging on a clinic-ready body scanner.

The AAVP hybrid combines the ability of the bacterial virus to target specific tissues with the capability of the mammalian virus to deliver genes to cells. The crucial vehicles, or vectors, in the AAVP hybrid retained the properties of their respective parental viruses, which the researchers called a surprising outcome.

"This is only a proof-of-concept, and although we have yet to translate these hybrid viruses for use in humans, we hope that this new system will have future clinical applications," says Wadih Arap, M.D., the co-leader of the study and professor of Medicine and Cancer Biology. "In addition to the obvious biological interest, when the vector is refined for patient use, it could perhaps help us diagnose, monitor and treat human tumors more accurately."

The finding is the latest in a series of studies by Pasqualini and Arap that are built around their discovery that the human vasculature system contains unique molecular addresses. Organs and specialized tissues also have specific "zip codes" on their blood vessels, as do tumor blood vessels. Knowing this, Pasqualini and Arap designed, constructed, evaluated, and validated the targeted AAVP system over the past several years. Amin Hajitou, Ph.D., a postdoctoral fellow in the Arap/Pasqualini laboratory and first author of the Cell study says, "we were pleased by the strong effects of gene transfer in mouse models of common diseases such as breast and prostate cancer."

Their next step was to work closely with the team of M. D. Anderson researcher Juri Gelovani, M.D., Ph.D., chair of the Department of Experimental Diagnostic Imaging, a pioneer in development of molecular-genetic imaging tools.

"We could see by using positron emission tomography that the reporter and therapeutic genes were being expressed throughout the tumors in the animals," Gelovani says. "This is an example of the so-called 'theragnostic' approach, a combination of the words therapeutic and diagnostic."

The international collaborative research team plans to evaluate the safety and efficacy of other hybrid vectors in animal models. The ultimate goal is to adapt and optimize the AAVP-based targeting prototype for use in patients.

ASBMB member Renata Pasqualini is professor of medicine at The University of Texas M. D. Anderson Cancer Center. Her research exploits the molecular diversity of blood vessels to develop targeted therapies and imaging agents for cancer and other debilitating diseases. Pasqualini received her doctorate in biochemistry from the Ludwig Institute for Cancer Research at the University of Sao Paulo, Brazil, in 1990. She completed postdoctoral research fellowships at Harvard Medical School, Dana Farber Cancer Institute and The Burnham Institute in La Jolla, Calif.

Dr. Renata Pasqualini

Pasqualini is principal investigator for a large interdisciplinary study titled "The Human Vascular Map Project," which focuses on developing a road map with "zip codes" of the molecular anatomy of blood vessels in human organs and tissues. Pasqualini and Wadih Arap, M.D., an attending physician and professor of medicine and cancer biology at M. D. Anderson, have pioneered the "zip code" technique to identify proteins specific to different tissues, including prostate and breast cancer. Their group also has identified homing ligands that target white fat vasculature in mice. Their ongoing work aims to develop ways to destroy blood vessels that support fat accumulation, a strategy that could eventually be applied to human obesity treatment.

Protein Folding Controls Cellular Routing

By Nicole Kresge, Staff Science Writer

Several years ago, P. Michael Conn, associate director of the Oregon National Primate Research Center (Oregon Health and Sciences University), showed that most disease causing mutants of the human gonadotropin-releasing receptor (GnRHR) were actually competent proteins that had become misrouted and retained by the cellular quality control system. These proteins could be rescued with pharmacological chaperones, or "pharmacoperones," and restored to function.

"Clearly, production of misfolded proteins is an important etiology of disease," says Conn. "This replaces the view that mutants are always functionally defective and leads to new therapeutic approaches."

Now Conn and his colleagues have shown that this approach is also exploited as a means of controlling routing in normal function of healthy cells. The study appears in the March 31 issue of the *Journal of Biological Chemistry* (281: 8417-8425) and shows a motif of four non-adjacent amino acids that allows this to occur in the human GnRHR.

The researchers discovered that the primate GnRHR contains a lysine residue at position 191 that restricts its expression at the plasma membrane, causing it to be retained in the endoplasmic reticulum and then degraded. Deletion of this amino acid dramatically increased its expression at the plasma membrane, and rodents that normally do not contain Lys191 expressed the synthesized receptor at much higher levels.

Conn and his colleagues showed that Lys191 in the human GnRH receptor destabilizes a specific Cys-Cys bridge that is essential for creation of a properly folded receptor. When they expressed the human plasmid in host cells, the bridge formed about half the time, resulting in about half the receptor being properly trafficked and the other half being retained in the endoplasmic reticulum and destroyed.

The view that Lys191 causes the receptors to be misrouted was also supported by the observation that a pharmacoperone that enabled mutants to pass through the cellular

Correct protein folding is required for accurate subcellular routing of receptors, ion channels and enzymes. GPRCs use engagement with the cellular quality control apparatus to regulate plasma membrane expression of receptors as a part of normal cellular function. Image by Joel Ito and P. Michael Conn, Oregon National Primate Research Center, OHSU.

quality control system also increased the plasma membrane expression of the human receptor but not the rat receptor.

"The creation of a receptor that is delicately balanced between the plasma membrane and the endoplasmic reticulum has resulted in a human receptor that is extremely sensitive to mutation," explains Conn. "Toleration, let alone strong and convergent evolutionary pressure for such mutational liability, along with the burden of inefficient function, suggests that this post-translational regulation is extremely important in advanced mammals. It provides a mechanism by which proteins, by interacting with chaperones, can rapidly respond to need without protein synthesis."

In the JBC paper, Conn's lab also provides evidence that other proteins employ this mechanism as well and that this is a more common mechanism than is currently recognized.

"These studies explain how very modest changes in DNA sequence make possible major differences in protein processing and cellular trafficking and present a mechanism by which cells can control functional protein levels without the requirement for transcription or translation," concludes Conn.

ASBMB member P. Michael Conn is the Associate Director and Senior Scientist of the Oregon National Primate Research Center, Special Assistant to the President, and Professor of Physiology and Pharmacology and of Cell Biology and Development at Oregon Health and Science University. After receiving a B.S. degree and teaching certification from the University of Michigan (1971), a M.S. from North Carolina State University (1973), and a Ph.D. degree from Baylor College of Medicine (1976), Conn did a fellowship at the National Institute of Child Health and Human Development, NIH, then joined the faculty in the Department of Pharmacology, Duke University Medical Center where he was promoted to Associate Professor in 1982. In 1984, he became Professor and Head of Pharmacology at the University of Iowa College of Medicine, a position he held for eleven years. Conn is the former Editor-in-Chief of *Endocrinology*, *Molecular and Cellular Neurosciences*, *Methods in Neuroscience* and *Recent Progress in Hormone Research* and prior editor of *J. Clinical Endocrinology and Metabolism*; he is presently is the Editor-in-Chief of *Endocrine*, *Methods*, *Contemporary Endocrinology*, and *Contemporary Drug Therapy*.

Conn, a vigorous supporter of the humane use of animals in research is best known for his research in the area of the cellular and molecular basis of action of gonadotropin releasing hormone action in the pituitary and CNS. He has authored or co-authored nearly 300 publications in this area. The work of his laboratory has been recognized with a MERIT award from the NIH, the J. J. Abel Award of the American Society for Pharmacology and Experimental Therapeutics, the Weitzman, Oppenheimer and Ingbar Awards of the Endocrine Society, the National Science Medal of Mexico (the Miguel Aleman Prize) and the Stevenson Award of Canada. Conn is a former President of the Endocrine Society, during which time he founded the Hormone Foundation.

New Patient-Oriented Research Section in JLR

In an effort to incorporate more clinical patient-oriented research, the Journal of Lipid Research is initiating a new category for submitted manuscripts called "Patient-Oriented Research Articles." This category will include research articles containing studies in which human subjects play an important role and at least one of the authors has had direct contact with the subjects. The criteria for review will be similar to regular research articles. To facilitate submission of these manuscripts, the online system for JLR manuscript submission now has a check box for papers in the Patient-Oriented Research category.

The new section will launch in the August 2006 issue of JLR. This issue will also contain a new Thematic Review Series devoted to patient-oriented research. The series will be edited by JLR Associate Editor Henry Ginsberg and will consist of several invited reviews covering a broad range of patient-oriented research involving lipids, lipoproteins, and arteriosclerosis.

For more information please visit the JLR website at www.jlr.org.

By Nicole Kresge, Staff Science Writer

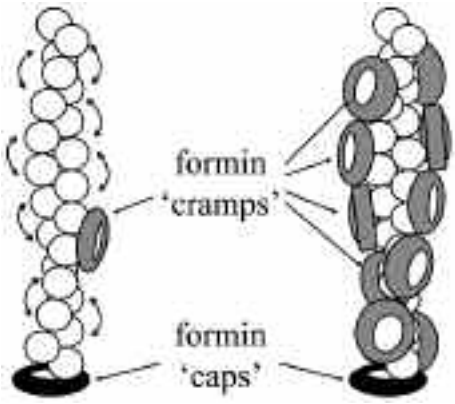
ASBMB Bio Bits

Formins Regulate Actin Filament Flexibility through Long Range Allosteric Interactions

Beáta Bugyi, Gábor Papp, Gábor Hild, Dénes Lôrinczy, Elisa M. Nevalainen, Pekka Lappalainen, Béla Somogyi, and Miklós Nyitrai

J. Biol. Chem. 2006 281: 10727-10736

Formins are proteins that nucleate actin filaments and play essential roles in the regulation of the actin cytoskeleton. In this paper, the authors describe the effects of a formin fragment from mice (mDia1-FH2) on the conformation of actin filaments using a temperature-dependent fluorescence resonance energy transfer method. Their results reveal that formin fragments increase the flexibility of actin filaments. The magnitude of this effect depends on the formin:actin concentration ratio. The characteristics of this concentration dependence indicate that more than one mechanism is involved in the formin effects. From their findings, the authors propose a model in which formin binding to barbed ends of actin makes the filaments more flexible through long range allosteric interactions while formin binding to the sides of the filaments stabilizes the protomer-protomer interactions.



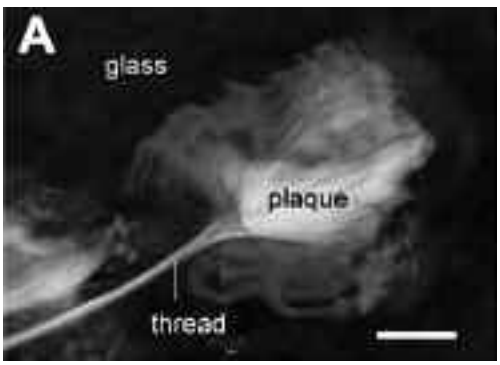
A model showing how formin affects the dynamic properties of actin filaments.

Probing the Adhesive Footprints of Mytilus californianus Byssus

Hua Zhao, Nicholas B. Robertson, Scott A. Jewhurst, and J. Herbert Waite

J. Biol. Chem. 2006 281: 11090-11096

Mussels are able to thrive despite persistent surf and tides in part due to their ability to adhere to surfaces via a holdfast structure called the byssus. This fibrous extracellular structure consists of a bundle of threads each of which is tipped distally by an adhesive plaque that bonds to mineral and metal surfaces. Using direct laser desorption ionization mass spectrometry, the authors of this manuscript analyze the "footprints" that were deposited onto glass coverslips by the California mussel Mytilus californianus. They find that the secretions contain variants of a family of proteins known as M. californianus foot protein 3 (mcfp3). Purification of these proteins from plaques and foot tissue reveals that mcfp3s are highly polar, dihydroxyphenylalanine-rich molecules. These findings suggest that adhesion in the California mussel may be governed by novel interactions in addition to the usual noncovalent ones.



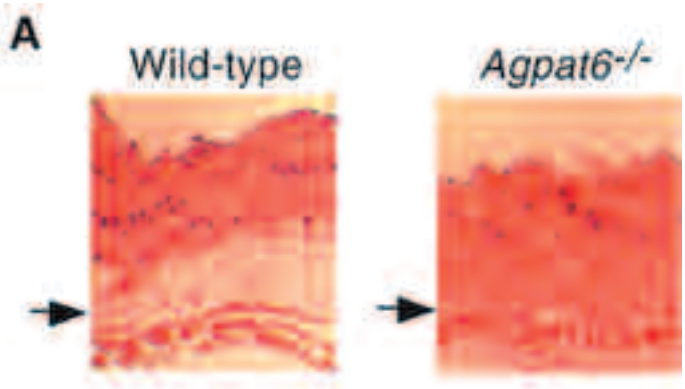
Mussel footprint plaques were analyzed by MALDI.

Agpat6 Deficiency Causes Subdermal Lipodystrophy and Resistance to Obesity

Laurent Vergnes, Anne P. Beigneux, Ryan Davis, Steven M. Watkins, Stephen G. Young, and Karen Reue

J. Lipid Res. 2006 47: 745-754.

In most mammalian tissues, the synthesis of triglycerides involves the sequential addition of fatty acids to a glycerol backbone, with unique enzymes catalyzing each step. The 1-acylglycerol-3-phosphate O-acyltransferase (AGPAT) is involved in an intermediate acylation step. To date, seven AGPAT genes have been identified, but their physiological functions remain elusive. In this study, the authors generate a mouse model deficient in AGPAT6 and find that the mice have a 25% reduction in body weight. The mice are also resistant to both diet-induced and genetically-induced obesity. Among other things, this reduction in body weight is associated with increased energy expenditure, reduced triglyceride accumulation in adipose tissue, and a lack of adipose tissue in the subdermal region.



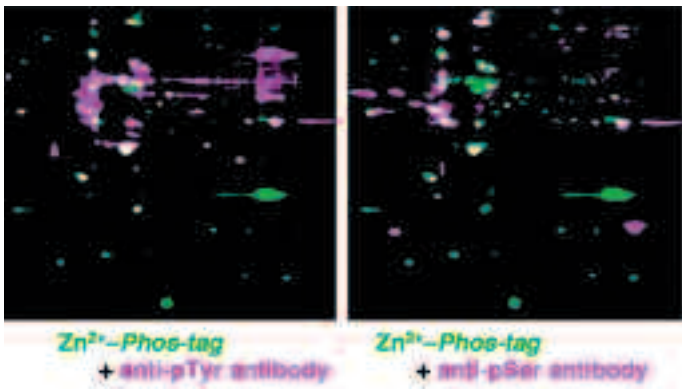
Mice without AGPAT6 lack a subdermal fat cell layer.

Phosphate-binding Tag, a New Tool to Visualize Phosphorylated Proteins

Eiji Kinoshita, Emiko Kinoshita-Kikuta, Kei Takiyama, and Tohru Koike

Mol. Cell. Proteomics 2006 5: 749-757

Phosphorylation is a fundamental post-translational modification that is used to regulate the function, localization, and binding specificity of proteins. However, abnormal protein phosphorylation can lead to carcinogenesis and neuropathogenesis. Thus, methods for determining the phosphorylation state of proteins are important with respect to evaluating biological and pathological processes. Here, the authors introduce two methods for the visualization of phosphorylated proteins using alkoxide-bridged dinuclear metal complexes as novel phosphate-binding tag (Phos-tag) molecules. The first method uses electroblotting to analyze protein phosphorylation status by phosphate-selective ECL signals. The second method is based on the mobility shift of phosphorylated proteins in SDS-PAGE with a polyacrylmide-bound Mn2+-Phos-tag.



Visualization of phosphorylated proteins on a blotting membrane.

by John D. Thompson, Editor

Making Space for a Biotech Center in the Chicago Area

The acquisition and subsequent redevelopment of a former Pfizer pharmaceutical research facility by Forest City, a Cleveland-based developer, promises to quadruple the amount of speculative office space available for biotechnology companies in the Chicago area.

Forest City bought the one-million-square-foot complex last spring for $43 million. Since then, the company has demolished 9 of the 13 buildings on the 23-acre campus, leaving about 700,000 square feet of offices and laboratories. The company plans to build an additional 1.3 million square feet of new space, also for offices and labs, over the next 10 years. Overall, the project is expected to cost more than $500 million.

The goal, according to Gayle Blakeley Farris, President of the Science and Technology Group of Forest City, is to create something that the company says is long overdue in Chicago: a biotech cluster similar to those in the high-tech bastions operating in the metropolitan areas around Boston and San Francisco.

"We think there's an opportunity for Chicago to become the third coast for the biotech industry," Farris said. "There are a number of major institutions here—such as the University of Chicago, the University of Illinois and Northwestern University—where important research is going on. But up until now, there hasn't been a critical mass of space available for companies seeking to capitalize on that research."

According to a recent study commissioned by Forest City, there is a shortage of speculative biotech office and laboratory space in Chicago. The city has about 500,000 square feet, much of it in facilities owned by public entities like the State of Illinois.

By comparison, the areas around Boston and San Francisco, the acknowledged leaders in the field, each have more than 11 million square feet. But even much smaller cities like Madison, Wisconsin and Raleigh-Durham, North Carolina outperform Chicago in this respect.

"The lack of space has been a stumbling block," said David Miller, President of the Illinois Biotech Industry Organization, "What has happened in the past is that a scientist could usually get a few thousand square feet of space from a university or a corporation to pursue a promising idea, but the minute he or she got bigger and needed 10,000 or 20,000 square feet it was a problem."

Scott Brandwein, Senior Managing Director of the life sciences group of CB Richard Ellis, the national real estate services firm, which is assisting Forest City in leasing the Skokie center, agrees. "There are very few options for companies coming into the market," he said. "There's never been a speculative market here."

What speculative space is available generally leases for $20 to $30 a square foot, but Forest City is hoping to do better than that — about $30 to $40 a square foot — for at least some of the newer space in the complex.

"We're targeting three types of tenants," said Michael Rosen, senior vice president for new business development for the center. "The first is international companies in need of space for a U.S. headquarters, the second is companies that provide services to the pharmaceutical industry on a contract basis, and the third is new companies that are ready to leave the incubators."

One company looking at the project is Midwest BioResearch, a biotech start-up based in an incubator facility owned by Northwestern University in nearby Evanston. "Our initial need is for 10,000 square feet but we could double that in a fairly short period of time," said Mark Weston, a business consultant who is advising the firm on real estate and financial issues.

Mr. Weston added that, "From the standpoint of networking, product innovation and creating new business, I think having a cluster of businesses in one location is important." The drawback, predictably, is rent. "I think," said Mr. Weston, "that both the lessee and the lessor will have to come together in the beginning to get tenants in."

'Florida Research Coast' Touted in Chicago

The goal of Florida development officials at the BIO 2006 conference in Chicago this April was to market Florida's Research Coast and form relationships that could bring biotechnology-related business or investments to the region. After spending several days promoting local assets at what is considered the world's largest biotech industry gathering, the Research Coast's three representatives said they think they achieved their goals.

The three said high-skill jobs associated with biotechnology are needed to offset the low-wage employment base and high home prices. Tammy Simoneau of the Economic Council of Martin County said agricultural biotechnology, for example, could be a great fit. "We can't continue to survive strictly as a service-based industry," she declared.

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by John D. Thompson, Editor

Biotech's Gains Again Outstrip Drug Giants

The American biotechnology industry has surpassed pharmaceutical companies for the third straight year as the primary source of new medicines, helping biotech revenue jump nearly 16 percent last year to a record $50.7 billion, according to an Ernst & Young report released last month.

Biotechnology companies still spend considerably less on research than their rivals in the traditional pharmaceutical industry, the report said, about $20 billion a year compared with more than $60 billion for the established drug giants. But the biotech companies get more results for their money, surpassing pharmaceutical companies for the first time in 2003 in getting novel types of medicines approved by the Food and Drug Administration.

"This is an independent, self-sustaining industry that is growing at more than twice the rate of the pharmaceutical industry," said Scott Morrison, a partner at Ernst & Young LLP.

Biotech firms have lately become the main source of innovation in U.S. medicine, including treatments for ailments such as cancer and heart disease. A prime example is Exubera, the inhaled insulin for which Pfizer Inc. won FDA approval early this year. Hailed as a milestone in the lives of diabetics, Exubera is the first new way to get insulin into the body since that hormone was discovered in 1921. However, it wasn't invented at Pfizer, it was developed by Nektar Therapeutics, a small biotech company in San Carlos, California, which may benefit handsomely if Exubra is a success in the marketplace.

"The pharmaceutical sector is clearly looking at biotech as the innovation leader," Morrison said, noting that the dependency runs both ways, with biotech companies needing cash, manufacturing skills, and marketing muscle from the big drug companies.

Despite big gains in revenue, the biotech industry overall still lost money last year as it spent heavily to develop new products, the report said. However, the industry's revenue has been growing at annual double-digit rates for nearly two decades, more than twice as fast as the traditional pharmaceutical industry, thanks to 58 biotech products at the FDA awaiting approval and hundreds more in late stages of testing.

Though losses continue, they are narrowing rapidly. The loss for publicly traded biotech companies in the United States shrank to $2.1 billion last year from the $4.9 billion of 2004, largely due to cost-cutting. The industry appears to be on track to achieve overall profitability by the end of the decade, Ernst & Young said.

China's Demand for Pharmaceuticals May Reach $46.29 Billion

Demand for pharmaceuticals in China is projected to increase 13.6 percent annually to 375 billion yuan (U.S. $46.29 billion) in 2010, according to a new study from The Freedonia Group, a U.S.-based industry research firm.

The report says that Western proprietary prescription drugs will generate the strongest growth based on new products, especially for cardiovascular, neurological, cancer and antiviral indications that are not treated effectively by currently available therapies. However, Western generic medicines will continue to hold the largest share of the Chinese pharmaceutical market. The best growth opportunities for these drugs will emerge in newly off-patent cholesterol-reducing, antipsychotic and second generation antihistamine preparations.

Based on proven efficacy over thousands of years and sustained profitability among older residents, traditional Chinese medicines will continue to form a large segment of China's pharmaceutical industry. However, due to the lack of proprietary products and sharper pricing competition, these preparations will see somewhat slower growth than Western drugs.

Regulatory and commercial reforms will open up distribution systems to a wider range of proprietary and generic drugs. The research report sees several therapeutic classes of medication, including antiviral, anticancer, psychotherapeutic, and neurological agents as underserved in China and the cause of major imbalances in the overall quality of health care.

Western over-the-counter (OTC) medication will increasingly penetrate the Chinese market as the government promotes the expansion of the retail drug sector to improve the accessibility of basic medicines to residents of rural and overcrowded urban areas.

Career Opportunities

IFOM-IEO CAMPUS For Molecular Oncology, MILAN, ITALY

HEAD OF PROTEOMICS

Two of the main Cancer research Institutions in Italy, the FIRC Institute of Molecular Oncology (IFOM) and the European Institute of Oncology (IEO) have expanded and integrated their research activities into a common campus. The IFOM-IEO Campus is also home to the PhD program of the European School of Molecular Medicine (www.semm.it). Central services include Animal Facilities (mouse, zebrafish and C. elegans), Imaging, Protein and Antibody Production, DNA and Tissue Microarrays, DNA Sequencing, Real-time PCR, Bioinformatics. Open structure laboratories foster communication between groups. We are now calling for applications for the position of Head of Proteomics.

The successful candidate will manage both his/her own research group and the Proteomics Facility in the Campus. Commensurate packages will be provided for each activity, including equipment and personnel. As a Group Leader in the Campus, the candidate will be expected to develop an internationally competitive line of research in any area pertinent to cancer biology, diagnostics or therapy. As a Facility Director, the candidate will guarantee access to proteomics-based solutions to other research groups in the Campus. The Facility is expected to maintain a state-of-the-art technological infrastructure over time. The Campus provides a vast potential for scientific interactions. Candidates should have extensive experience in proteomics and a proven track-record in running a competitive research group.

DEADLINE FOR APPLICATIONS: June 30, 2006.

Applications should be sent by e-mail only to search-proteomics@ifom-ieo-campus.it and should include: CV, publication list, statement of Achievements and Interests (max. 2 pages), names and e-mail addresses of 4-5 referees. Applicants should also ask their referees to directly send their letters to the same e-mail address.

The IFOM-IEO campus is an equal opportunity employer. We encourage applications from women and will implement measures required to place all scientists in a situation of equal competitiveness.

www.ifom-ieo-campus.it

Florida Int'l Univ.

Applied Research Center at Florida International University (Miami, FL)

MOLECULAR BIOLOGY RESEARCHER

The ARC at Florida International University (FIU) in Miami, FL has an immediate opening for a Molecular Biologist to lead a research program jointly performed by FIU-ARC and the National Renewable Energy Laboratory (NREL). The program constitutes cutting-edge biological hydrogen research consisting of gene cloning, regulation, and expression in E. coli, DNA and peptide sequencing, and hydrogenase enzyme purification and activity studies. A Ph.D. in Molecular Biology or related fields is required, along with strong experience in vector construction and analysis, bacterial expression systems, western blots, and sequencing. The ARC is a leading research institution in energy and the environment (www.arc.fiu.edu). NREL is the premier renewable energy laboratory of the Department of Energy (www.nrel.gov).

A competitive compensation package will be provided. FIU is an Equal Opportunity Employer. Submit your cv and three reference contacts to Dr. George Philippidis, Applied Research Center, Florida International University, 10555 W. Flagler St., EC 2100, Miami, FL 33174 or to George.Philippidis@arc.fiu.edu.

UMCP

ASSISTANT PROFESSOR IN APPLIED NUTRITION AND NUTRIGENOMICS

The Department of Nutrition and Food Science, University of Maryland, invites applications for a 9.5-month, tenure track research/teaching position. Applicants must have a PhD in Nutrition or related research discipline. Status as a Registered Dietitian with practical clinical experience is desirable.

Responsibilities include: 1.) teaching undergraduate and graduate courses in clinical nutrition, nutritional assessment, advanced nutrition, and other related areas as needed; 2.) establishing an independent, innovative, competitively funded research program in the area of applied nutrition/nutrigenomics.

A strong commitment to teaching at the undergraduate and graduate levels, the ability to obtain outside funding to support research and a demonstrated ability to publish in peer-reviewed journals are essential elements of this position.

The scientist in this position is encouraged to develop interdisciplinary research with other faculty in the department and other campus units.

Applicants should send a statement of teaching and research interests and how they will extend and complement that of the current faculty, a complete CV, representative publications, official transcripts, and the contact information (mail, email, phone, fax) for 3 references to:

Dr. Robert Jackson, Chair , Search and Screening Committee
Department of Nutrition and Food Science
0112 Skinner Building, College Park, MD 20742

Applications will be accepted until a successful candidate is selected. For best consideration, apply by June 15, '06. The University of Maryland is an EO, AA employer.

Calendar of Scientific Meetings

JUNE 2006

20th IUBMB International Congress of Biochemistry and Molecular Biology and 11th FAOBMB Congress in conjunction with 79th Annual Meeting of the Japanese Biochemical Society and 29th Annual Meeting of the Molecular Biology Society of Japan

June 16–23 • Kyoto, Japan
Deadline for On-line Registration: May 18, 2006
Website: www.congre.co.jp/iubmb/registration.html

Bacterial Cell Surfaces
A Gordon Research Conference

June 25–30 • Colby-Sawyer College
New London, New Hampshire
Chairs: Ry Young and Arnold J. Driessen
Vice Chairs: Anne H. Delcour and Jeff Errington

4th Annual Meeting of the International Society for Stem Cell Research

June 29–July 1 • Metro Toronto Convention Centre
Toronto, Ontario, Canada
For information on the ISSCR Annual Meeting, contact ISSCR Headquarters: Ph: 847-509-1944; E-mail: isscr@isscr.org
Conference Administrator: Deb Pederson dpederson@isscr.org
Press Inquiries: Heather Gagnon hgagnon@isscr.org
Conference Director: Liz Freyn lfreyn@isscr.org

JULY 2006

Third Annual World Congress on Industrial Biotechnology and Bioprocessing

July 11–14 • Toronto, Canada
Sponsored by the Biotechnology Industry Organization (BIO), American Chemical Society (ACS), National Agriculture Biotechnology Council (NABC), Agri-Food Innovation Forum, and BIOTECanada.
Email: worldcongress@bio.org; Ph: 202-962-9200

ASCB 2006 Summer Meeting: Stem Cell Niches

July 15-18 • Boston University
Organized by Sean Morrison, HHMI/University of Michigan
Keynote: Allan Spradling, Carnegie Institute of Washington/HHMI
Session 4: Hematopoietic Stem Cell Niches (Cont.)
Session Chair: Fiona Doetsch
Session 5: New Niches for Stem Cells
Session Chair: David Scadden

17th International Symposium on Plant Lipids

July 16–21 • Michigan State University Campus, East Lansing
Organizer: Christoph Benning
For registration information, preliminary program, instructions for submitting abstracts, and for information on financial aid available for young scientists to attend the meeting, go to: www.ispl2006.msu.edu/. Members of underrepresented groups are especially encouraged to apply for financial aid.

Bioscience 2006: Bioscience for the 21st Century

July 23–27 • Glasgow, Scotland
Abstract Submission Deadline: April 13, 2006
Early Registration Deadline: May 22, 2006
For information: www.BioScience2006.org

Biochemical Journal Symposium
Literature, Legacy, Life

July 24 • Glasgow, Scotland
Celebrating 100 Years of Biochemistry
For information: www.BioScience2006.org

AUGUST 2006

ISPMB 2006 – 8th International Congress of Plant Molecular Biology

August 20–25 • Adelaide Convention Centre, South Australia
Abstract and Early Registration Deadline: Friday, March 3.
Online registration and abstract submission pages:
www.sallyjayconferences.com.au/ispmb2006/registration.htm
www.sallyjayconferences.com.au/ispmb2006/abstract.htm
Abstracts cannot be accepted without registration and payment. All abstracts must be submitted online, abstracts sent as attachments will not be accepted.
www.sallyjayconferences.com.au/ispmb2006/program.htm

SEPTEMBER 2006

5th European Congress of Biogerontology

September 16-20 • Istanbul, Turkey
Tel: +90 216 347 35 35 Pbx; Fax: +90 216 347 78 50
Email: okarabel@symcon.com.tr; Website: www.symcon.com.tr
Congress President Prof. Serif Akman, Etlik, Ankara , Turkey
Tel: +90 312 304 3306; Fax: +90 312 304 3300
E-mail: sakman@gata.edu.tr

The 33rd Annual Conference of the Federation of Analytical Chemistry and Spectroscopy Societies (FACSS)

September 24–28 • Disney's Contemporary Resort, Lake Buena Vista, FL
Contact: FACSS, PO Box 24379, Santa Fe, NM 87502
Phone: 505-820-1648; Fax: 505-989-1073
Email: facss@facss.org; Web Page: www.facss.org

OCTOBER 2006

International Conference of Immunogenomics and Immunomics

October 8–12 • Budapest, Hungary
A joint meeting of 2nd Basic and Clinical Immunogenomics and 3rd Immunoinformatics (Immunomics) Conferences
Email: diamond@diamond-congress.hu
Website: www.bcii2006.org

4th International Conference on Structural Genomics

October 22 – 26 • Beijing, China
Website: http://www.sino-meetings.com/icsg2006/

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

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

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: Bio-active 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.
For Further information: www.biophysicsmap.com

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

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; Web Page: www.facss.org

cell signallingbiology

– an outstanding new online reference work from one of the world's leading experts

Professor Sir Michael J Berridge
(Cambridge)
Shaw Laureate in Life Science and Medicine 2005

This major new contribution to the field of cell signalling biology provides researchers, teachers and students alike with an essential online resource. Emerging information on cell signalling pathways is integrated and presented within the context of specific cell types and processes.

Contents

1 Introduction
2 Generation of second messengers
3 Ion channels
4 Sensors and effectors
5 Off reactions
6 Cellular processes
7 Spatial and temporal aspects of signalling
8 Development
9 Proliferation
10 Neuronal signalling
11 Cell stress, inflammatory response(s) and cell death
12 Signalling defects and disease

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