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ASBMB today

March 2007

Visualizing Lectin Oligomerization

American Society for Biochemistry and Molecular Biology

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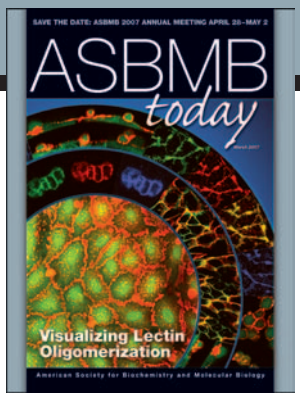
MARCH 2007



ON THE COVER: Confocal microscopic images illustrating that soluble galectin-3 molecules oligomerize upon binding to glycans on the cell surface. For more information, see ASBMB BioBits. 20

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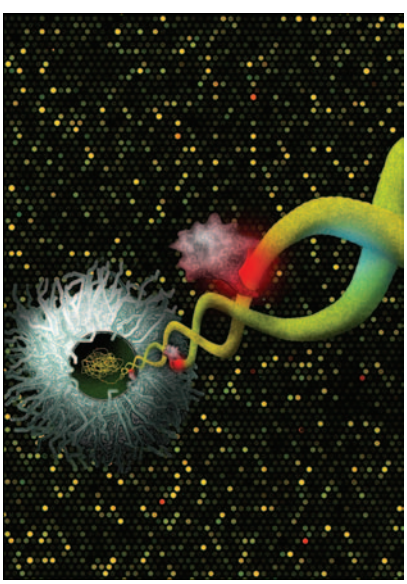

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president's message

Local Advocacy Activities—Here's How We Do It Here in Tennessee!

HEIDI HAMM, PRESIDENT

Research!America has shocking data indicating that upwards of 80% of Americans cannot name a living scientist. This means that most of our neighbors next door or across the street probably are not aware that they are living next to a scientist. One of the major projects I have taken on during my presidency of ASBMB is to get more of our members active in their local communities in spreading the word to the media, the general public, and to elected representatives about the importance of the work we do as scientists. One of the best ways to do this is to actually meet with these people regularly, and that is what a group of my students and I have begun to do here in Nashville.

We have been meeting since the fall with Newt Williams, a former CEO of Hoescht Marion Roussel Canada Inc. and the former vice president for government affairs with Celanese. Newt has retired to Nashville and has been a wonderful mentor and advisor to our group. His level of commitment to our cause is second to none. He has been able to give us a very solid basic course in local advocacy, having practiced it for so many years himself. Under his expert tutelage, we have been arranging a series of meetings with local members of Congress.

Jim Cooper and Jeff Conn with Dave Weaver, director of the High Throughput Screening Facility at Vanderbilt.

We had our first meeting, with Rep. Jim Cooper (D-TN) at the end of December. Cooper is serving his eighth term in Congress and serves on three committees: Budget, Oversight & Government Reform, and Armed Services. Our meeting with him was very successful. He had scheduled us for an hour, but he stayed for almost two hours, discussing our issues and clearly enjoying himself. For some perspective on how extraordinary this is, a typical meeting in Washington—if you meet with the member at all instead of a staffer—is usually 15–20 minutes. This is one of the major reasons why meeting with members when they are home—which is about half the time—is so valuable.

Three graduate students in the Department of Pharmacology at Vanderbilt, Ashley Torain, Tim Panosian, and Efrain Garcia (who recently defended his thesis), gave presentations on the economic benefits of biomedical research, discovering treatments for drug abuse, and NIH research's impact on the military. Then we took him on a tour of our high throughput screening facility at Vanderbilt to show him how biomedical research can lead to breakthroughs that lead to new drugs. Of course, as with all fundamental research, there is a long road to follow before it is put to actual use in patients, but these discoveries will someday have very direct effects on the lives of Americans.

During the tour, Jeff Conn, our director of Drug Discovery, showed Rep. Cooper a very dramatic video of a rat model of Parkinson disease in which the rat gets up and walks out of its cage when it is infused with a molecule that Jeff discovered silences an overactive inhibitory synapse in the basal ganglia involved in the immobility and rigidity seen in Parkinson patients.

Jim Cooper with (*left* to *right*) Ashley Torain; Efrain Garcia, Ph.D.; and Tim Panosian.

Cooper is very active and engaged on issues related to health care, and he responded very positively to the presentations and the tour. He was an adjunct professor of Management at Vanderbilt's Owen Graduate School of Management and thus is familiar with the academic environment and the importance of federal funding for research. A great entrée for future interactions is that he had not known the work of Murphy and Topol, two economists from the University of Chicago who did an economic analysis of the impact of biomedical research on the American economy.

Future meetings are planned with Rep. Bart Gordon (D-TN), chairman of the House Committee on Science and Technology, and with Rep. Marsha Blackburn (R-TN). Blackburn is a member of the prestigious Energy & Commerce Committee, one of the most powerful committees in the House of Representatives, with oversight responsibility for the National Institutes of Health. We also hope to meet with Sen. Bob Corker (R-TN), newly elected this past November to the seat held by former Sen. Bill Frist.

We will be writing a "tips for activists" page that will appear in this space in coming months. In the meantime, Pete Farnham, ASBMB's public affairs officer, will be happy to help you arrange meetings with your own members of Congress in your districts. You can visit our advocacy page for information on how to do this or contact Pete directly at pfarnham@asbmb.org for personalized advice.

ASBMB Expa

BY NICOLE KRESGE

As part of an effort to increase its international visibility, ASBMB has opened two offices in China. In doing so, the Society hopes to increase its international membership and boost submissions and subscriptions to its journals as well as encourage the exchange of different perspectives on topics of importance to the scientific community.

"In opening the office in China, the ASBMB is effectively recognizing the great contributions to science of the past that have come from China as well as their enormous potential for future scientific discovery," remarked Barbara Gordon, ASBMB executive director.

One of the major undertakings of ASBMB China is the publication of a selection of ASBMB journal articles that the Society feels will be of particular interest to scientists in Asia. Each article that appears in the compendium will be published with a commentary by a prominent Asian scientist. Several of these articles will be featured in an upcoming issue of *ASBMB Today*.

ASBMB's central Chinese office is located in Guangzhou, China, at the Guangzhou Institute of Biomedicine and Health at the Chinese Academy of Sciences. Guangzhou is the capital city of Guangdong Province and is located in the middle south of the province, north of the Pearl River Delta. The office is staffed by Jin Zhang and supervised by Duanqing Pei, and it handles the overall operations of ASBMB China.

Pei is a professor and the deputy director general at the Guangzhou Institute of Biomedicine and Health where he studies cancer. He earned his Ph.D. from the University of Pennsylvania and was a faculty member first at the University of Michigan, Ann Arbor, and then at the University of Minnesota before joining the Guangzhou Institute in 2002. Pei has been a *Journal of Biological Chemistry* Editorial Board member since July 2006. Zhang holds an M.B. degree from Tianjin Medical University and an M.S. from the University of Bristol in the UK. She has worked at the Guangzhou Institute as an assistant to the director since 2004 and continues to do so part-time.

GIBH: The Guangzhou Institute of Biomedicine and Health.

nds in China

The second office is at Tsinghua University Medical School in Beijing. This office is staffed by Xingru Zhou, whose duties include managing ASBMB's Asian membership, promoting the Society in Beijing and the northern part of China, and maintaining the Society's relationship with Chinese biological associations. Zhou has a B.S. degree in Biology from the Chinese University of Agriculture and has worked at Tsinghua University since 2003 as an executive assistant.

Both offices opened on December 1, 2006, and there will be an official dedication sometime this spring.


Jin Zhang


Duanqing Pei


Xingru Zhou

asbmb member spotlight

DuBois to Take Post at M. D. Anderson

Raymond DuBois, M.D., Ph.D., director of the Vanderbilt-Ingram Cancer Center, will join The University of Texas M. D. Anderson Cancer Center as provost and executive vice president for academic affairs by September 1, 2007.

"Dr. DuBois is a highly regarded laboratory scientist and clinical investigator, already well known to many at M. D. Anderson. He also is a skilled administrator who directs one of the nation's most respected comprehensive cancer centers," said M. D. Anderson President John Mendelsohn, in announcing the appointment. "He is a terrific choice for M. D. Anderson, which has ambitious plans for the future."

DuBois will have responsibility and authority for M. D. Anderson's research agenda, programs, resources, and space; educational programs at all levels; and all activities related to the appointment, resourcing, and mentoring of faculty.

DuBois is professor of medicine, cell/developmental biology, and cancer biology at Vanderbilt in Nashville. His research interests focus on studies of the molecular and genetic bases for colorectal cancer. He is internationally recognized for elucidating a key role of the prostaglandin biosynthetic pathway in producing inflammatory mediators that promote colorectal cancer. His research facilitated clinical trials targeting this pathway in humans, which demonstrated a reduction in colon polyps that are the precursors of cancer.

Katzenellenbogen Is E. B. Hershberg Awardee

John A. Katzenellenbogen, Swanlund Professor of Chemistry at the University of Illinois, Urbana-Champaign, has won the 2007 American Chemical Society's E. B. Hershberg Award for Important Discoveries in Medicinally-Active Products.

This award, which is given every other year, was established in 1988 by Schering-Plough Research Institute to honor the contributions of Emanuel B. Hershberg to the pharmaceutical industry, especially the application of organic chemistry for the discovery and development of novel drugs. Katzenellenbogen will present his award address at the Spring 2007 meeting of the American Chemical Society in Chicago.

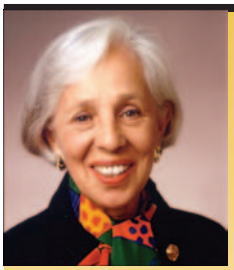
Katzenellenbogen is being honored for his pioneering research at the interfaces of chemistry, biology, physiology, and medicine that has illuminated the molecular aspects of estrogen action. Early in his career, Katzenellenbogen investigated the interaction of small molecule ligands with hormone receptors by developing a reactive analog of the antiestrogen tamoxifen. Later, he devised estrogens radiolabeled with gamma- or positron-emitting radionuclides, which could be used, along with imaging techniques, to provide information on estrogen receptor levels in breast tumors. He was the first to image breast tumors and breast tumor metastases on the basis of their estrogen receptor content. Katzenellenbogen also invented new receptor-based reagents that act as novel sensors for assessing the activity of new pharmaceuticals.

IN MEMORIAM

David K. Fukushima 1917–2006

David Kenzo Fukushima was born in Fresno, California, on August 24, 1917. He grew up in Montebello, California, and earned a B.A. in Chemistry at Whittier College in 1939. As a Japanese-American in California when World War II broke out, Fukushima was sent to a relocation camp. He completed his M.A. in Chemistry from the University of California, Los Angeles, in 1943, finishing his master's thesis in camp. He then left the camp to study organic chemistry at the University of Rochester where he earned his Ph.D. in 1946. In 1946 he joined the Sloan-Kettering Institute for Cancer Research, where he began his career as a leader in the study of steroid metabolism, becoming a full faculty member in 1960. He later became the Director of the Steroid Institute at Montefiore Hospital, affiliated with Albert Einstein Medical College.

Fukushima was well-known in the field of steroid biochemistry, particularly for his research into the metabolism of hydrocortisone and testosterone and later for his work on the activities of various polypeptide hormones. He authored hundreds of articles in prominent scientific journals on steroid and peptide hormone metabolism, was an associate editor of the *Journal of Biological Chemistry*, and mentored many postdoctoral students.



Singer to Receive NAS Public Welfare Medal

The National Academy of Sciences has selected Maxine F. Singer, president emeritus of the Carnegie Institution of Washington, to receive the Public Welfare Medal, its most prestigious award. The medal is presented annually in recognition of an individual's extraordinary commitment to the use of science for the public good. Singer will receive the award for providing inspired and effective leadership in matters of science and its relationship to education and public policy.

Singer is a pioneer in molecular biology and an accomplished spokesperson and leader in science policy who has dealt with many of today's key issues. She has championed the cause of women and minorities in science, fostering equal access to education and career opportunities, and has worked tirelessly to improve science education.

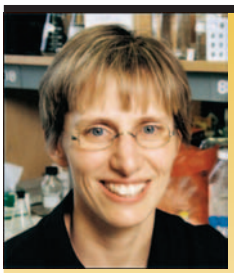
While at Carnegie, Singer implemented Project Magellan, a cooperative effort with a number of academic astronomy departments to build two world-class telescopes, developed Carnegie's department of global ecology, established the Carnegie Academy for Science Education (CASE), and introduced Carnegie's "First Light" project, in which young students attend an innovative Saturday science school. Singer has also spoken out authoritatively on the issue of genetic manipulation in research and biomedical applications and was among the first to publicly raise the issue of recombinant DNA's potential risks. As chair of the National Research Council's Committee on Science, Engineering, and Public Policy, Singer addressed graduate education, postdoctoral scholarship, the plight of women in science, and scientific conduct.

Liu Selected as Nakanishi Prize Recipient

Dr. Hung-Wen "Ben" Liu, a professor in the Department of Chemistry and Biochemistry at the University of Texas, Austin, has been selected as the 2007 recipient of the Nakanishi Prize from the American Chemical Society. The award is given "to recognize and stimulate significant work that extends chemical and spectroscopic methods to the study of important biological phenomena."

In 1995, the American Chemical Society and the Chemical Society of Japan created the award named for Koji Nakanishi, the renowned Columbia University chemist who transformed the field of natural products chemistry. The prize is given in odd-numbered years by the American Chemical Society and in even-numbered years by the Chemical Society of Japan. The award consists of $3,000 and a bronze medal inscribed with the insignia of both organizations.

Liu's work in natural products chemistry, biosynthesis, and mechanistic investigations has centered on the quest to elucidate and understand nature's strategies for making biologically important structures. Combining a background in both natural products chemistry and protein chemistry, Liu has been taking on what, to him, is one of biological chemistry's most intriguing traits: to start with simple chemical building blocks, such as sugar molecules, and end up with enormous molecular diversity and complexity.



Kiessling to Receive ACS Medal

Laura Kiessling of the Department of Chemistry at the University of Wisconsin-Madison has won the American Chemical Society's (ACS's) Francis P. Garvan-John M. Olin Medal for distinguished service to chemistry by women chemists.

The award consists of a cash prize (US $5,000) and a medal designed by Margaret Christian Grigor. The award was established by Francis Garvan and Mabel Brady Garvan in 1936 in honor of their daughter. It was initially an essay contest, which ran for seven years, as a memorial to their daughter. The Garvin-Olin Award is the ACS's third-oldest award and the first award established to honor women chemists.

Kiessling's insight and creativity have been recognized by many other honors and awards. Most notably, she received a coveted MacArthur Foundation Fellowship in 1999 as well as an Arthur P. Sloan Fellowship, an ACS Arthur C. Cope Scholar Award, and a National Science Foundation National Young Investigator Award.

Kiessling's research involves developing and implementing synthetic methods that provide access to biologically active compounds for hypothesis-driven and discovery-driven research. She has explored how multivalent interactions influence cell adhesion and cell signaling using small molecules and polymers and has also looked at the role of carbohydrates in mediating cell adhesion.

FASEB

FASEB Releases Annual Federal Funding Recommendations for FY 2008

BY CARRIE D. WOLINETZ

With the resolution of the appropriations process for FY 2007, which funded the National Institutes of Health (NIH), National Science Foundation (NSF), and U. S. Department of Energy's (DOE's) Office of Science at significantly higher levels than the previous fiscal year, FASEB was able to release our annual report, *Federal Funding for Biomedical & Related Life Sciences Research, FY 2008*, to Congress in a far more positive atmosphere than anticipated. "I was prepared to come here today to talk about the difficulty in looking ahead to FY 2008 when FY 2007 remains unresolved," said FASEB President Leo Furcht, M.D., talking to a group of reporters during a press conference releasing the report. "Instead, I am here today with you celebrating the good news that our voices were finally heard."

The FASEB report is developed each year by committees of scientists representing the 21 member societies, often in consultation with experts from the relevant federal agencies or other advocacy groups. "A major theme of this report and FASEB's advocacy efforts over the upcoming year will be sustainability of the scientific enterprise," said Furcht. "Only through predictable federal funding streams will we be able to maintain steady research progress to continue to improve the health of our nation and quality of our lives."

In line with this theme, for the first time ever, FASEB has released a pre-publication copy of its report lacking a specific funding recommendation for NIH. "We have calculated approximately where NIH funding would be had the agency received inflationary increases, just enough to sustain the enterprise since the end of the era of rapid budget increases in 2003," Furcht explained. "Our hope would be to bring NIH back to that sustainable level over a three-year period and to recoup the losses caused by flat funding and biomedical research cost inflation." The final recommendation, whose calculation is dependent upon finalized numbers for the previous fiscal year and new estimates for the biomedical inflation index, will be released in a final version of the report and will likely be at least 6%.

The annual report will be distributed to federal lawmakers, health research officials in the administration, and the research community. It will serve as the basis for FASEB's research funding advocacy efforts for the next fiscal year. The FASEB report may be read in its entirety at opa.faseb.org, and a summary of the recommendations for FY 2008 follows:

National Institutes of Health

FASEB recommends a FY 2008 appropriation level that would set the NIH on a three-year track to recoup the losses caused by biomedical research inflation.

National Science Foundation

FASEB recommends an appropriation of $6.5 billion for NSF in FY 2008.

U. S. Department of Energy

FASEB recommends an appropriation of $4.3666 billion for U. S. DOE's Office of Science in FY 2008.

U. S. Department of Veterans Affairs (VA)

FASEB recommends an appropriation of $480 million for the VA Medical and Prosthetic Research Programs and an increase in research infrastructure support to $45 million in FY 2008.

U. S. Department of Agriculture (USDA)

FASEB recommends an appropriation of $248 million for the USDA National Research Initiative and $1.377 billion for the Agricultural Research Service in FY 2008.

National Aeronautics and Space Agency (NASA)

FASEB requests that Congress provide NASA with an increase of at least $39.5 million for biological sciences research in FY 2008 to partially restore deep cuts made in the life sciences budget in the two previous fiscal years. Subsequent increases in funding should be contingent upon the implementation of the organizational changes outlined in this report.

Carrie D. Wolinetz, Ph.D., is with the FASEB Office of Public Affairs.

NIH Awards Nearly $11.5 Million to Support Science Education Programs

The National Center for Research Resources (NCRR), a part of the National Institutes of Health (NIH), announced in January that it will provide nearly $11.5 million to fund 11 Science Education Partnership Awards (SEPAs) across the nation. The SEPA projects are designed to inform the public about health issues, foster science literacy, and encourage students to consider careers in the health sciences.

Through mobile laboratories, portable science kits, planetarium films, and online activities, these SEPA projects will provide hands-on, inquiry-based instruction on topics such as cardiovascular risk factors, genetic testing, and diabetes treatment and prevention. Participants will study multiple research-related issues, learn about the clinical trials process, and examine their own health and lifestyle choices.

"These programs reach out to students and their families and target some of the most important issues in medicine today such as ethics, evidence-based medicine, and bioinformatics," said Barbara M. Alving, M.D., acting director of NCRR. "We also want to show students that they have the opportunity to envision careers in medicine, clinical research, drug discovery, and the basic sciences."

SEPA programs reach out to students in rural and underserved communities by funding K-12 classroom activities as well as science centers and museum exhibits across the country. The awards support professional development for science teachers, the development and distribution of hands-on science curricula, traveling exhibits, and Web sites for students, teachers, and the general public.

In the initial three-year phase, partnerships are formed among biomedical and clinical researchers, educators, community groups, and other interested organizations to create programs that provide a better understanding of scientific research. In a second two-year phase, these SEPA-generated curricula are broadly disseminated.

This round of 11 grants brings the SEPA portfolio to 72 active projects that span the country, from Maine to Florida and from Alaska to Texas. These SEPA projects address a wide range of subject matters from basic questions about biology to how clinical research is conducted.

NIH Leads Effort to Help Women in Science and Medicine Fulfill Potential

NIH Director Elias A. Zerhouni has created the Working Group on Women in Biomedical Careers to examine issues raised in the recent National Academies report, "Beyond Bias and Barriers, Fulfilling the Potential of Women in Academic Science and Engineering," and to respond to the challenges issued to government funding agencies to maximize the potential of women scientists and engineers.

"It is critical to address the barriers that women face in hiring and promotion at research universities in many fields of science," said Zerhouni. "I have appointed the NIH Working Group on Women in Biomedical Careers to help address this challenge and to develop innovative strategies and tangible actions that can be implemented to promote the advancement of women in research careers both within the NIH intramural community and throughout the extramural research community."

Zerhouni and Vivian Pinn, associate director for Research on Women's Health and director of the Office of Research on Women's Health, will co-chair the Working Group, which will carefully consider the recommendations in the National Academies report.

This report, which was initially funded by the NIH Office of Research on Women's Health as well as Eli Lilly and Co., the National Science Foundation, the Ford Foundation, and the National Academies, called for an urgent broad national effort to maximize the potential of women scientists and engineers in academia. The committee that produced the report was chaired by Donna Shalala, Ph.D., president of the University of Miami and former secretary of the U. S. Department of Health and Human Services.

NSF Provides $14 Million to Advance Research in Comparative Genomics of Economically Important Plants

Scientists will find improved ways of studying the structure, function and evolution of the genomes of economically important plants, thanks to $14 million in new awards from the National Science Foundation (NSF).

Resources to be developed include genomic sequences, genetic markers, maps, and expressed sequence collections. These are much-needed tools for researchers working in areas as diverse as genome evolution and plant breeding.

Awardees will address scientific questions including the role of polyploidy in genome evolution, the genomic basis of speciation, and the relationships between cultivated plants and their weedy relatives.

"If the Plant Genome Research Program has been making the bricks that build a conceptual framework for the genomes of economically important crop plants, these projects will provide the mortar," said James Collins, NSF assistant director for biological sciences. "The impact of genomics in evolutionary, ecological, and population studies of crop plants will be far-reaching."

Many crop plants have large, complex genomes that in some cases are "polyploid"–containing multiple genomes. Polyploidy is widespread in plants and animals and can lead to dramatic changes in gene content and genome organization that are only just beginning to be understood.

Biologists will investigate the red rice genome to find out whether it is a domesticated crop or was introduced as a weed from Asia. Credit: *Washington University, St. Louis*.

A project led by researchers at Iowa State University will develop sequence and map resources to study polyploidy in cotton, while researchers at the University of Missouri will look at the impact of polyploidy on plant morphology in *Brassica* species, which includes plants such as canola and brussels sprouts. Other projects at the University of Georgia and the University of Arizona will develop sequence resources to study genome organization in wheat and rice.

The outcomes from these projects will allow researchers to understand how extra copies of genes function in these plants and how genomes from different sources can work together in a single plant.

Projects based at the University of California at Davis and Cornell University will catalog variants in pine trees and in maize, respectively, to allow researchers to link genetic variation with changes in gene function. This information could have applications in plant breeding.

More than half of the world's most cultivated crops have relatives that are invasive weeds, competing with the crop for nutrients and water and leading to reduced yields. One example is red rice, a weedy form of rice that reduces the yields of cultivated rice by as much as 80% and contaminates harvests with its small, red-coated grains. A project led by researchers at Washington University in St. Louis will examine the regions of the red rice genome associated with weediness to find out whether it originated from the domesticated crop or if it was introduced as a weed from Asia.

A related project led by investigators at Michigan State University will investigate differences in gene expression in weedy and cultivated radishes to uncover which genes are associated with invasiveness. The outcomes of these projects could lead to a greater understanding of how plants become weedy and invasive and yield possible avenues for better selective control of weeds, scientists believe.

President's 2008 NIH Budget Lands with a Thud: $500 Million Below Likely 2007 Figure

BY PETER FARNHAM

"Other than roundly denouncing it, the best thing to do is ignore it." These words, spoken to *ASBMB Today* by an informed observer, were about the kindest we have heard concerning the President's 2008 budget proposal for the National Institutes of Health (NIH), released on February 5 as part of his $2.9-trillion proposal for federal spending for next year.

At first glance, it appears that the President's proposal for NIH would raise the budget by $232 million, from $28.3 billion to just over $28.6 billion. However, even if the President's budget were to become law at some point later this year, the proposal still falls almost $300 million short of the amount the House of Representatives has *already approved* for NIH for FY 2007, that is, over $28.9 billion. (As of this writing, the Senate has not approved the House figure but is very likely to do so soon.)

To make matters even worse, buried in the fine print of the budget proposal is a provision transferring about $200 million from NIH into a fund set up to fight global AIDS/HIV. In the past, much of this money has come from the State Department; however, this year, the White House proposes that it all come from NIH. This means that the proposed $232 million increase is in reality a $32 million increase; it also means that, in effect, the administration is proposing to fund NIH at $500 million below what the House of Representatives has already approved for the year before! This budget, it should also be kept in mind, comes against a backdrop of a presidential visit to NIH on January 17, during which President Bush said, "I truly believe the NIH is one of America's greatest assets, and it needs to be nourished."

Said Dave Moore, executive director of the Ad Hoc Group for Medical Research, "This budget diminishes our research efforts, delays scientific progress, and denies hope for millions of Americans suffering with disease and disability." He continued, "Enactment of the Administration's proposal would mean a 13% cut in inflation-adjusted dollars in the biomedical research capacity of our nation at a time when the health challenges confronting us have never been greater."

FASEB of course weighed in with equally negative comments. FASEB President Leo Furcht noted that "the President's proposal stands to cause grievous harm to our ability to combat debilitating diseases, from diabetes to Alzheimer's, as well as leaving us woefully unprepared to deal with emerging illnesses or pandemic influenza. Far from nourishing NIH, the FY 2008 budget represents further deprivation and attrition of this invaluable agency."

As might be expected, the democratic staff of the Senate Budget Committee denounced the budget proposal in the strongest terms: "President Bush's FY 2008 budget is filled with debt and deception. It is disconnected from reality and continues to move America in the wrong direction." Regarding NIH, the report blasts the proposal as short changing NIH by $743 million below the House-approved continuing resolution if the effects of inflation are factored in. (In early February, NIH released its numbers for inflation in 2005 and 2006 and projects that in 2007 and 2008 biomedical inflation will be 3.7%.)

Back to the Future—2007 Appropriations

As we noted above, on January 31 the House of Representatives approved a continuing resolution to fund the federal government for the remainder of 2007. The bill provides a $620 million increase for NIH, just over 2%. While this does not match inflation, it was far better than anyone had expected and is a triumph for our champions in Congress, Sens. Tom Harkin (D-IA) and Arlen Specter (R-PA), who worked tirelessly to bring this increase about. In the House, Reps. Dave Obey (D-WI) and Mike Castle (R-DE) were major players.

We also would be remiss without mentioning the outpouring of support for the increase generated by the biomedical community, including many readers of this magazine. Thousands of e-mails, faxes, and telephone calls deluged the House in late January, urging support for the proposed increase. ASBMB as an organization also put its money to work, paying for a full page ad in *Roll Call*, an influential newspaper widely read on Capitol Hill, calling for major funding increases at NIH. More than 128 ASBMB members signed the ad, including a dozen Nobel laureates, about 40 members of the National Academy of Sciences, and most of the society leadership, as well as representatives from over a dozen other scientific societies, several voluntary health organizations, and the CEOs from eight pharmaceutical and biotech companies.

The Senate is expected to pass the long-term continuing resolution by February 15; thus by the time you read this, it should already be law.

White House Calls for 8% Increase at NSF in FY 2008

BY PETER FARNHAM

The White House announced on February 5 that it would seek more than $6.4 billion—almost an 8% increase—for the National Science Foundation (NSF) in FY 2008. This is $466 million over the amount contained in the long-term continuing resolution the House passed in late January (and that will very likely have been approved by the Senate and signed into law by the time you read this article).

All but $100 million of the increase will go into NSF's core research programs, under Research and Related Activities. More than $50 million will go into the NSF's Education and Human Resources programs; most of the rest of the increase will go into Major Research Equipment.

NSF said that in addition to continuing ongoing projects, the budget proposal emphasizes new research on improved computing abilities to meet the challenges of 21st century inquiry, as well as polar research, ocean research, nanotechnology, education, and international collaborations.

This is the second year in a row that NSF has requested increases in the 7–8% range, in keeping with the American Competitiveness Initiative that President Bush announced during his State of the Union speech in early 2006.

While praising the NSF budget proposal in general, many analysts are worried about the long term. Sam Rankin, chair of the Coalition for National Science Funding, told *ASBMB Today* that, "Overall, I'm pleased, given the funding environment, although I think we need to look at science more broadly, because you never know where the next advance is going to come from. We need to fund all science at a level that will make us competitive in the future, and we're not doing that right now. These are only incremental changes, and in the long run we're short-changing science."

The NSF notes that while the bulk of the new money goes to support programs in mathematics, physical sciences, and engineering, it is often hard to differentiate between disciplines because of the nature of modern science. According to an NSF press release, "Natural systems, for example, provide stunning examples of effective communications, complex computation, efficient signaling, adaptive self-organization, and multimodal sensing using small but complex chemical and physical networks. Studies of such biophysical systems will engage physical and computer scientists, engineers, biologists, and social scientists."

The House gave NIH some instructions on how the additional $620 million is to be spent. The resolution appropriates $483 million specifically for the Common Fund. This is a change from previous practice; institutes and centers (ICs) used to contribute a uniform percentage of their appropriations to the Fund; Congress didn't specify the Fund amount. From now on, it will. This is an important point, because although most of the House-approved increase will go to the Common Fund, the money that would have come from the ICs will now remain in the ICs, thus providing the ICs with an effective increase.

Of the $483 million for the Common Fund, $40 million is for a new Junior Pioneer awards program. Like regular Pioneer awards, they will fund high-risk research with potentially high-impact returns. The Junior version will be for smaller amounts of money, for shorter periods, and will be fully funded.

The resolution also includes $69 million, an increase of $58 million, for the National Children's Study. There's also a new $91 million fund within the Office of the Director (OD) to support new investigators. This will take some of the pressure off the ICs, which have been asked to maintain the level of awards to first-time investigators.

NIH estimates that the additional funding is sufficient to support 500 additional research project grants. Under a straight Continuing Resolution (CR) the number would have risen by 470, partly because of NIH's December 2006 guidance stating that there will be no inflationary adjustments for noncompeting renewal awards. So, there will be a total of 970 more research project grants than in FY 2006.

Peter Farnham, CAE, is ASBMB's public affairs officer.

ASBMB 2007/2008 SCIENCE POLICY FELLOWSHIP

ASBMB is pleased to announce that it is accepting applications from newly graduated Ph.D.s for the ASBMB Science Policy Fellowship. The fellowship affords the opportunity to gain experience and insight into the workings of the policy process and the role that science plays in government decision-making in a wide range of issues. The Fellow will work closely with policy professionals both inside and outside the government. The experience will provide exposure to the federal research budget process, regulatory issues, and the interplay between science and decision-making.

The application deadline for the 2007/2008 fellowship is May 15, 2007.

TERMS

The Society will sponsor one Fellow who will spend one year as a staff member in the ASBMB Office of Public Affairs. The fellowship will begin September 1. The Fellow will receive an annual stipend of $40,000 and health care coverage.

QUALIFICATIONS

Fellows will be selected on a competitive basis from ASBMB members who have:

- a recently awarded doctorate (i.e. the applicant is not beyond the post-doctoral stage);
- interest in the relationship between science, technology, and public policy;
- flexibility in tackling a variety of tasks; and,
- excellent interpersonal and communication skills.

HOW TO APPLY

Individuals interested in applying for the ASBMB Science Policy Fellowship should submit the following:

1. A resume/CV;
2. A letter of intent (2-3 pages) that outlines:
   - why you have applied
   - what policy issues and situations interest you
   - what you hope to accomplish as a Fellow
   - how you feel this experience will enhance your career
   - your participation in civic activities and/or public affairs
3. Two letters of reference sent directly to ASBMB. Please include the addresses and telephone numbers for your references in your application.

Send all application materials to:
Peter Farnham, CAE, Public Affairs Officer, ASBMB, 9650 Rockville Pike, Bethesda, MD 20814.
Tel.: 301-634-7384; Fax: 301-634-7126; E-mail: pfarnham@asbmb.org

FDA Proposes Drug Safety "Report Cards"

Public outcry over the withdrawal of the painkiller Vioxx is leading to closer government scrutiny of new drugs to identify and disclose late-developing safety problems.

As part of a pilot program, the Food and Drug Administration (FDA) will issue drug "report cards" that will detail unexpected side effects that emerge within 18 months of a drug's approval. The reports also would include follow-up studies and details about how the drugs are being used.

The proposal is among more than a dozen initiatives the FDA unveiled in January in response to a recent report by a committee of experts at the Institute of Medicine that criticized the agency's handling of drug safety in the wake of the Vioxx case. Nor will it be the last word, said FDA Commissioner Dr. Andrew von Eschenbach.

"It will be a continuous process of improvement. The initiatives we are announcing today are not the full story, nor are they the final chapter in that story," von Eschenbach told reporters.

The Institute of Medicine report was prompted in part by the 2004 withdrawal of Vioxx after research showed it increased risk of heart attacks and strokes. The report said the FDA needs more funding, people, and authority to ensure it focuses on the safety of drugs while the drugs remain on the market.

The FDA also hopes to step up its mining of large public and private health care databases to detect emerging safety problems, said agency drug chief Dr. Steven Galson. The Department of Veterans Affairs recently signed an agreement to share such information with the FDA.

The agency also said it would regularly publish newsletters to summarize its safety reviews of older drugs and disclose emerging issues. However, the newsletters would be scrubbed of whatever the FDA deemed confidential commercial and predecisional information, the agency said.

The Institute of Medicine, part of the federally chartered National Academies, had pushed for even more public disclosure of that underlying information, said Alta Charo, a member of the committee that wrote the report who is also a University of Wisconsin professor of law and bioethics.

"Crucial to our recommendation was that these post-marketing data be shared with an advisory committee made up of independent safety experts. Their proposal would make the data remain internal," Charo said.

Both the FDA and the drug industry are concerned that broad disclosure of preliminary information about apparent safety problems could do more harm than good.

"One thing we don't want to see happen is if patients get concerned and they decide to stop therapy and don't talk to their doctors," said Alan Goldhammer of the Pharmaceutical Research and Manufacturers of America, a drug industry group.

Genentech, Seattle Genetics in Deal

Biotechnology companies Genentech, Inc. and Seattle Genetics, Inc. announced that they have entered an exclusive worldwide license agreement for the development and commercialization of a humanized monoclonal antibody, SGN-40. SGN-40 is in Phase I and Phase II clinical trials to treat multiple myeloma, chronic lymphocytic leukemia, and non-Hodgkin's lymphoma.

Under terms of the deal, Seattle Genetics will receive an upfront payment of $60 million, potential milestone payments exceeding $800 million, and escalating double-digit royalties on annual sales of SGN-40. The milestone payments include $20 million in committed payments during the first two years of the agreement. The payments are dependent on clinical and regulatory events across multiple disease indications worldwide and the attainment of certain annual sales levels.

Seattle Genetics will continue some Phase I and Phase II clinical trials and development activities, with Genentech reimbursing the costs. Genentech also will fund future research, development, manufacturing and commercialization costs.

California Proposes Labeling for Cloned Food Products

In January, legislation was introduced in California to require the clear labeling of all products derived from cloned animals if these are approved for human consumption. Introduced by State Senator Carole Migden (D-San Francisco), the proposed bill aims to provide California residents with the option to choose what they consume.

The move came just weeks after the U.S. Food and Drug Administration (FDA) issued draft guidance on allowing meat and milk from cloned cows into the food chain. The regulator has opened a 90-day consultation period to gather feedback before deciding whether its proposals–including allowing cloned food to be sold with no special labeling–should become policy.

There is currently no regulation preventing cloned food from entering the nation's food supply. But the FDA has asked clone producers and livestock breeders to voluntarily refrain from introducing food products from clones or their offspring into the food supply until the agency endorses the findings of a National Academies report it commissioned in 2002 that declared cloned products safe for human consumption.

In its recent draft proposal, the agency said its assessment of the available scientific evidence shows no additional safety risks are posed by the technology. In the risk assessment section, however, FDA recommended that cloned sheep are not to be used for human food because of the limited data available.

Last month, the Center for Food Safety urged the American public to campaign against the regulator's draft proposal. In October 2006, the group filed a legal petition with the FDA seeking a moratorium on foods produced from cloned animals. It was joined in its efforts by a coalition of consumer, environmental, and animal welfare organizations.

Biomed Firm Commercializes Stem Cell Sales

This past January, Aruna Biomedical, Inc. announced that it reached an agreement to commercialize the distribution of neural stem cells by the billions.

"We're going to be able to distribute a cell that has the ability to produce all the different cell types in the nervous system," Aruna Biomedical, Inc. Chief Executive Steven Stice told the *Atlanta Journal-Constitution*.

Stice developed neural progenitor cell technology at the University of Georgia, where he is also a professor and director of its Regenerative Bioscience Center.

His arrangement with the university's research foundation calls for Aruna to market the cells, with the university getting a cut.

"We are offering a product that may accelerate the pace of neurological research for tens of thousands of scientists," he said, "and thereby may provide patients with possible therapies and treatments for debilitating neurological diseases and spinal cord injuries much sooner than imagined."

Venture Industry Likes Health, Web, Energy

According to a survey released in January by market research firm VentureOne in San Francisco, California, U.S. venture capital investments rose 8% to $25.75 billion in 2006. This is the highest level in five years. The industry's growth was fueled by funding for biotechnology and medical devices, Internet services, and alternative energy start-ups.

Health care venture investments attracted 628 deals as financing levels grew 12% to $8.25 billion.

"Health care, really for the past couple of years, has been generating most of the growth that we are seeing," said Josh Grove, an analyst with VentureOne.

Investment in medical devices saw a record year, both in the number of deals and dollars invested, which grew 20% to $2.63 billion. Biotech and pharmaceutical firms lured nearly twice as much capital, growing 12.5% to $4.72 billion.

"Medical devices in the past was the poor stepchild to biotech," said Mike Carusi, a health care investor and partner at Advanced Technology Ventures in Palo Alto, California. "No longer, judged in terms of exits through IPOs and mergers over the past 12–24 months," he said.

ALAN SACHS: *Moving from Academics to Industry*

Alan Sachs

Alan Sachs, vice president, RNA Therapeutics and Molecular Profiling, Merck Research Laboratories (MRL), assumed scientific leadership at Sirna Therapeutics and shared scientific leadership at Rosetta Inpharmatics, LLC in January 2007. Prior to that, he had responsibility for the scientific leadership at Rosetta Inpharmatics LLC beginning in July 2002 and responsibility for the Department of Molecular Profiling in 2006. Sachs joined Merck & Co., Inc. as director of Clinical Genomics for MRL in July 2001. He is one of the leading figures in the field of mRNA translation and regulation and has made landmark contributions, particularly to the understanding of the role of the poly(A) tail in translation and mRNA stability.

I left a position in the Molecular and Cell Biology Department at the University of California, Berkeley in 2001 to join a new genomics group at Merck Research Laboratories. The discussion below tries to capture the events and emotions that led up to this decision, and, I hope, will help others as they consider career changes.

My career path until 2001 was pretty much on a straight line for a lifetime in academics. I had undergraduate training in biochemistry at Cornell University, medical school and graduate training in cell biology at Stanford University, a Whitehead Fellow's position at the Whitehead Institute for Biomedical Research, and then a junior faculty appointment and subsequent promotion to tenure in the Department of Molecular and Cell Biology at the University of California, Berkeley. My research path was equally straight. I studied post-transcriptional control of gene expression in yeast and was involved in research involving biochemistry, genetics, and biophysics.

Being a "yeast person" in the 1990s prepared me scientifically for the changes to come in research on more complex organisms. I can now see that this contributed to my decision to make a career change. For example, yeast research tools included targeted gene disruptions, a complete genome sequence, utilization of gene expression microarrays, simple—and sometimes not so simple—genetics, and affinity purification of tagged macromolecular complexes. As more and more biologists working in complex model systems—such as worms and flies—reduced to practice the use of these tools for their needs, it became abundantly clear that some or all of them would ultimately have utility in human studies. I also realized that knowing how to apply these tools to new studies with humans would be an asset for me.

In addition to seeing how my skills could be applied to human studies, there were other positive events during this period that contributed to my decision. The draft sequence of the human genome was announced, and it was very exciting to consider being part of its exploration. I had just successfully completed a major set of studies in my laboratory and a new research direction in human genomics seemed very timely. Finally, many of my close colleagues were enjoying their recent career changes, so the jump to something totally different didn't seem as hard.

Negative events also helped to shape my decision. In the mid-1990s, a visiting professor and I had worked on a method to identify human DNA variation rapidly at low cost because we were excited about human genetics and wanted to see whether our efforts could make a difference. They didn't. What I learned was that I could not afford to do this type of work as an independent academic investigator. Gene expression profiling on microarrays also became an experimental tool during this period. What I learned was that I did not have and could not access a robust infrastructure to help me run the

experiments and analyze the large amount of data coming from them. Finally, in considering how to rectify the lack of resources available to me, I quickly realized that it was going to be a multiyear process involving an enormous effort just to get started, while others in industry or large consortiums were not facing this lag time. In summary, I appreciated that genomics was a complex and costly science and that, as an independent investigator, it wasn't at all clear that I was positioned for success.

In 2001 Peter Kim, the president of Merck Research Laboratories (MRL), asked me to consider working for Merck and Co., Inc. Peter had been a close friend and scientific colleague for many years, and we had discussed on several occasions my interest in applying my skills more directly to medical research. Among the various options I was able to consider at MRL, the one that was most appealing was to be part of an effort to build a leading pharmacogenomics group. This opportunity addressed all of my past concerns–it would allow me to apply my scientific skills to studies involving humans, and, given the scale of MRL research, it would position me for success in this area.

Of course making the decision to join MRL was not that simple. For starters, I still had a laboratory with postdocs, students, and staff. Was I really going to just stop what I was doing? I had a permanent position in a world-class department that asked nothing more of me than to be creative. Could I really leave that behind? Finally,

It is one thing to think you want to change your career; it is another to go ahead and change it

and most importantly, my family situation made it nearly impossible to relocate. Could I really make the change with this constraint?

It is one thing to think you want to change your career; it is another to go ahead and change it. It would be dishonest to say that I never wavered while making my decision to join MRL.

It would be dishonest to say my family wholly embraced the idea. Nonetheless, everything, thankfully, fell into place. My laboratory funding was transferred to colleagues who agreed to host those students/staff in need of continuing support, and the remainder of my staff found satisfying positions elsewhere. The university generously offered me an extended period of unpaid leave, thereby giving me the opportunity to return if things didn't work out. My family begrudgingly agreed to allow me to commute out of state.

That was June 2001. It is now five years later, and looking back I am sure that the decision to join MRL was the correct one for me. In addition to all the reasons cited above, I now realize that there are many other positive attributes of a large research-driven organization that should have been given more weight while making the decision. For example, working within a pharmaceutical company has allowed me to make better use of my medical degree. It has allowed me to take advantage of my interpersonal skills, something that was of value in the university setting but definitely not a required skill set. Finally, it has given me the opportunity to work every day in a team setting where everyone has, as the primary goal, the creation of medicines that will help others.

What are some take-away lessons from all of this for those of you considering a career change? First, a career change should not be out of the question just because you are successful in your current job. Second, try to choose a new career that allows you to apply some part of your past experience so that you can hit the ground running. Third, be aware of the changing environment, and evaluate whether you will be able to succeed in it if you don't change with it. Fourth, defining moments for your decision can come from unexpected places, so keep an open mind to all opportunities. Fifth, perceived barriers to change are not insurmountable. Finally, although the grass always looks greener on the other side, be ready for some of your expectations after the change to be exceeded and others to be left unfulfilled.

I hope that this essay has given you insight into the situation surrounding my career change and, as a result, can help you in considering yours.

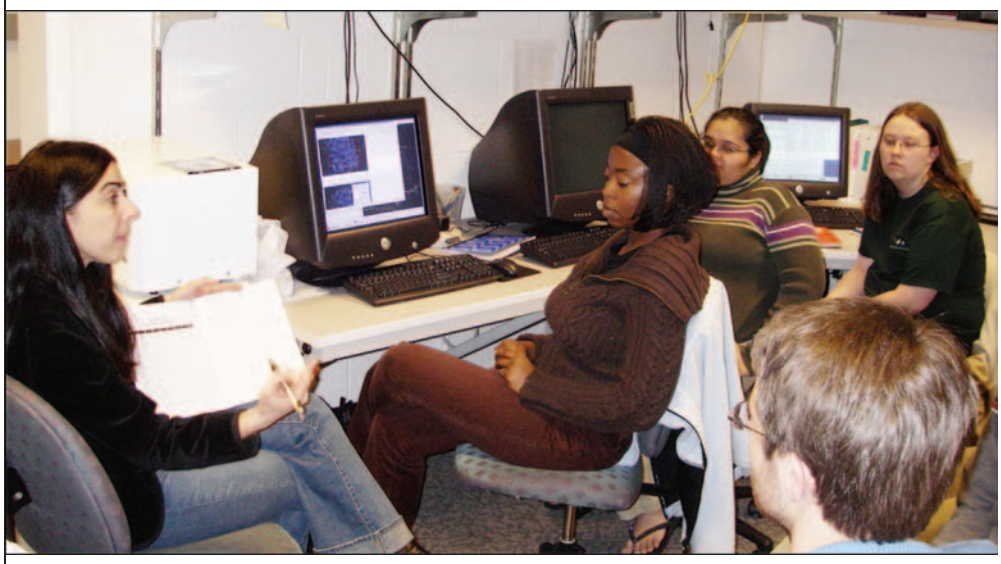
Creating Networks to Enhance Research and Education in Undergraduate Institutions

BY J. ELLIS BELL

Several years ago ASBMB created the Undergraduate Affiliates Network (UAN) to promote and support both research and education at the undergraduate level. The continued growth of interest in biochemistry and molecular biology at the undergraduate level is evidenced by both the continued growth of the network and the increased numbers of undergraduates that come to the national meeting each year.

At this year's ASBMB annual meeting in Washington, D.C., about 10% of the submitted abstracts have undergraduate students as first authors. These abstracts span virtually all of the "meetings" within the meeting, and undergraduates will be presenting their research alongside graduate students, postdocs and faculty. Although most attendees at the meeting usually can't distinguish undergraduate students from the other attendees, these students' contributions to the Society and biochemistry and molecular biology overall will be significant. Most of the undergraduates will also present their research as part of the Undergraduate Poster Competition, and the winners will receive travel awards for next year's annual meeting through the UAN program–a major benefit offered to schools or programs involved with the UAN. (See the Education section of the ASBMB Web site for more details.)

The UAN was built on the idea that connecting students at the undergraduate level with the discipline at a professional level and showing them the range of career possibilities and the richness of the science in the discipline would encourage them to pursue graduate work in one of the molecular life sciences. To achieve this most effectively it is important that students get involved with the Society, the profession, and research as early as possible. Studies have shown that undergraduate students who are also exposed to research and who work with senior researchers and advanced student investigators–such as advanced undergraduate students, graduate students, and postdocs–are more interested in pursuing graduate studies. Therefore we should encourage more undergraduate students to pursue research activities during their first and second year in college and not wait for them to become seniors. This would allow them not only to be involved in a research project but also to understand how to write a grant proposal, make presentations, write manuscripts, and plan follow-up experiments. All of these aspects play critical roles in their education, providing many of the skills detailed in the ASBMB recommended undergraduate curriculum.



Carla Mattos (*left*) with students at North Carolina State University.

The payoff of such an investment in undergraduate research should be

evident–not only will these students be more motivated to go on to graduate school, they also will be far better prepared and are far more likely to succeed in graduate school.

A focus on research in the education of undergraduates need not be and should not be focused solely on "investigator" driven research in a research laboratory. Increasingly, innovative faculty at all types of institutions are incorporating true research activities into classroom teaching, focusing on unanswered questions and approaches to solutions in the "lecture" portion of a course and real experimental research in the "laboratory" portion of a course. What better way to teach biochemistry and molecular biology? Carla Mattos, an associate professor of biochemistry at North Carolina State University in Raleigh, is a prime example of such an innovative approach to teaching. She has been so successful in combining her research activities with her teaching that she received the Presidential Early Career Award for Scientists and Engineers during a ceremony in September 2005 at the White House. She will be featured in the "Classroom of the Future" symposium at this year's annual meeting in Washington, D.C.

What are the benefits of the concept of the UAN? Whereas larger schools often have an infrastructure to support undergraduate research, smaller schools usually don't. One of the long term goals of the UAN is to increase the research experience of undergraduate students in the molecular life sciences by enhancing interactions between various academic institutions. By connecting institutions of various sizes, it is hoped that the research paradigm of teaching can be extended to a larger number of institutions. This requires more "teaching" faculty to be actively involved in research activities. Too often colleges separate these two activities, and credit is given for teaching but not for research.

So how can faculty at smaller institutions bootstrap themselves and their programs into productive research activities? Here again the network concept comes into play. At smaller institutions one faculty member usually has responsibilities in the area of biochemistry and molecular biology and little infrastructure support for research. Networking with colleagues in similar situations in other institutions is an important way to create a community that can lead to productive research and collaboration. In this day and age of high speed communication and videoconferencing such interactions do not necessarily have to involve institutions geographically close to one another. All that is needed is a few faculty members with related interests who have one or two students, and before you know it, students and faculty can be conducting research to the benefit of all. Such groups can easily focus on a single project area and can also tie into ongoing research at other institutions. In the coming months, the UAN site will include details of how academic institutions can join collaborative research efforts in biochemistry and molecular biology, especially when those efforts involve undergraduate students.

Although the number of UAN schools is increasing, many more schools could be involved. If your school or program has an undergraduate major in biochemistry or molecular biology and is interested in participating in the UAN, please contact ASBMB and find out more about the UAN. Details of the application process can be found on the ASBMB Web site. If you attend this year's annual meeting in Washington, D.C., please feel free to stop by the ASBMB booths (#201 and #203) in the Exhibit Hall and arrange to meet with the appropriate regional director of the program or to sign up your institution for UAN membership.

There are many direct benefits of being affiliated with the UAN. Each school or program affiliated with the UAN is eligible for one or more travel awards, allowing undergraduate students to compete in the Undergraduate Poster Competition at ASBMB's annual meeting. If you are interested in the UAN and its regional activities, please contact the appropriate regional director. You don't have to have a biochemistry or molecular biology major to participate and benefit from the UAN. Also, as a member of the UAN, you can have access to consulting services of ASBMB regarding how to start a biochemistry or molecular biology degree program.

The UAN is based upon regional networks, and each region organizes and sponsors local activities including regional meetings where undergraduates can present their research (and win travel awards to the next annual meeting). At the regional level there is also coordination of outreach activities, particularly those focusing on outreach to K-12 education as well as the type of undergraduate research coordination discussed above.

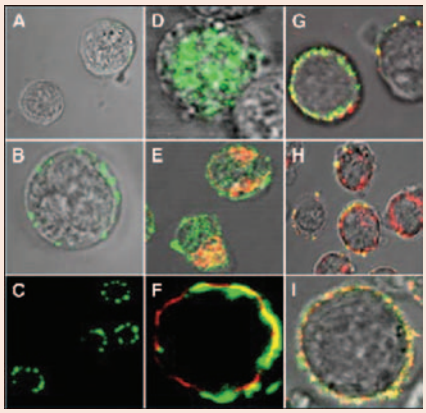
biobits asbmb journal science

Mol. Cell. Proteomics 2007 6: 29–42

Morphine Administration Alters the Profile of Hippocampal Postsynaptic Density-associated Proteins: A Proteomics Study Focusing on Endocytic Proteins

José A. Morón, Noura S. Abul-Husn, Raphael Rozenfeld, Georgia Dolios, Rong Wang, and Lakshmi A. Devi

Numerous studies have shown that opiates modulate synaptic transmission and plasticity in the hippocampus. Within the synapse, the postsynaptic density (PSD) receives and transduces synaptic information. As a result, the PSD contains a high concentration of proteins. In this study, the authors used quantitative proteomics to examine the changes in PSD protein levels in the mouse hippocampus upon repeated administration of morphine. They found that clathrin experienced an increase in concentration, and this increase was localized to the PSD. They also found that levels of adaptor protein-2 α1 (AP-2) and dynamin were increased locally at the PSD after morphine treatment. Because the clathrin-dynamin-AP-2 complex modulates the level of cell surface receptors including α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA), the authors investigated the effect of morphine on the association of the AMPA receptor subunit, GluR1, with clathrin. They found a substantial decrease in the levels of GluR1 associated with clathrin, suggesting that morphine modulates synaptic plasticity at hippocampal glutamatergic synapses by causing a redistribution of endocytic proteins at the synapse.



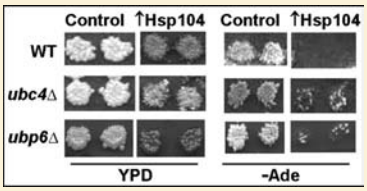
Morphine alters clathrin levels in the PSD.

J. Biol. Chem. 2007 282: 3004–3013

Effects of Ubiquitin System Alterations on the Formation and Loss of a Yeast Prion

Kim D. Allen, Tatiana A. Chernova, E. Paula Tennant, Keith D. Wilkinson, and Yury O. Chernoff

In cells, degradation of abnormal or damaged proteins occurs in part via the ubiquitinproteasome system (UPS), in which proteins are targeted for destruction by the attachment of ubiquitin tags. Mutations in different UPS components have been found to be associated with disorders linked to amyloid-like protein aggregation, such as Alzheimer and Parkinson diseases. In this paper, the authors investigated the involvement of the UPS in amyloid diseases caused by prions, using the yeast prion [*PSI*$^+$]. Previously, they found that overproduction of the chaperone protein Hsp104 results in loss of [*PSI*$^+$]. Here they show that this effect is decreased by deletion of either the gene coding for one of the major yeast ubiquitin-conjugating enzymes, Ubc4, or the gene coding for the ubiquitin-recycling enzyme, Ubp6. Moreover, deletion of Ubc4 significantly increases spontaneous formation of [*PSI*$^+$]. These results imply that the UPS is one of the major modulators of prion formation and clearance in the yeast cells.



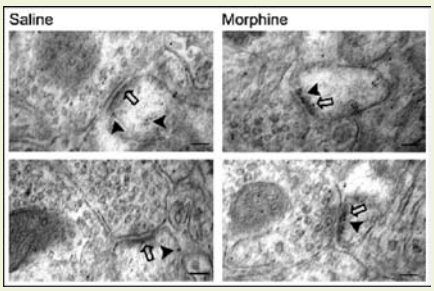
Deletion of UBC4 or UBP6 decreases *[PSI$^+$]* curing by excess Hsp104.

J. Lipid Res. 2007 48: 19–29

Novel Anti-Cholesterol Monoclonal Immunoglobulin G Antibodies as Probes and Potential Modulators of Membrane Raft-Dependent Immune Functions

Adrienn Bíró, László Cervenak, Andrea Balogh, András Lorincz, Katalin Uray, Anna Horváth, László Romics, János Matkó, George Füst, and Glória László

Because of its widespread distribution and important biological roles, cholesterol has long been assumed to be a nonimmunogenic or poorly immunogenic molecule. However, several laboratories have reported that cholesterol activates the classical and alternative pathways of the complement system. In this paper, the authors produced two IgG isotype monoclonal antibodies against cholesterol (AC1 and AC8) by immunizing mice with cholesterol-rich liposomes. They found that the antibodies specifically detected cholesterol and several structurally related sterols in cell-free assays, and they reacted with human lipoproteins. The IgG-type anti-cholesterol antibodies also bound to various cellular compartments, both extracellularly and intracellularly. These data suggest that these IgG anti-cholesterol antibodies may serve as probes of clustered cholesterol in live cells and thus may also have immunomodulatory potential.



Anti-cholesterol antibody AC8 binds to various compartments in cells.

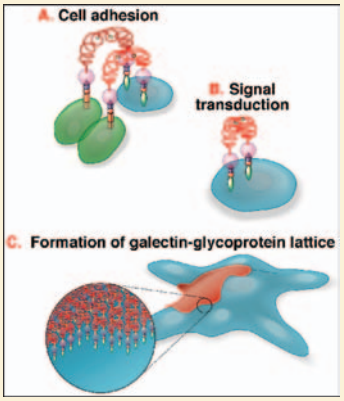
JLR

J. Biol. Chem. 2007 282: 1374–1383

Visualization of Galectin-3 Oligomerization on the Surface of Neutrophils and Endothelial Cells Using Fluorescence Resonance Energy Transfer

Julie Nieminen, Atsushi Kuno, Jun Hirabayashi, and Sachiko Sato

Galectin-3 is a member of the galectin family of soluble host carbohydrate binding proteins. The lectin is widely expressed, particularly in cells involved in the immune response, and has been implicated in various biological activities ranging from cell repression to cell activation and adhesion. Galectin-3 most probably performs these activities by ligand cross-linking, which is thought to be accomplished by oligomerization of its N-terminal domain after ligand binding by its C-terminal domain. This *JBC* report presents data confirming that galectin-3 oligomerization does in fact occur. By labeling the C terminus of the lectin, the authors were able to detect oligomerization between galectin-3 molecules using fluorescence resonance energy transfer (FRET). They detected FRET signals during galectin-3 lattice formation on neutrophils and endothelial cells, galectin-3-mediated signal transduction in neutrophils, and during galectin-3-mediated adhesion of neutrophils to an endothelial cell layer. These data suggest that galectin-3 indeed oligomerizes on cell surfaces in those three cross-linking modes.



Oligomerization of galectin-3 through three different cross-linking modes.

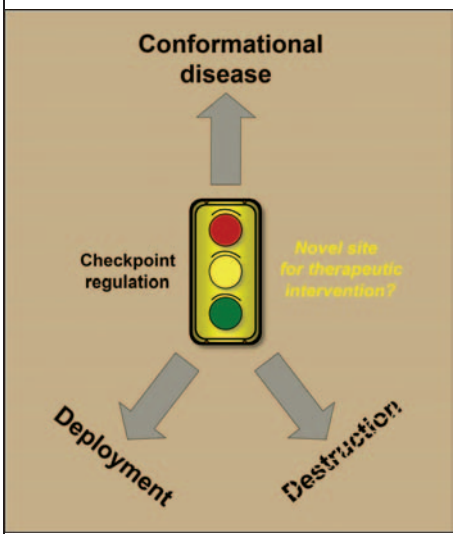
jbc

The Underlying Clockwork Mechanics of a Biosynthetic Checkpoint

BY RICHARD N. SIFERS

Proteins are often needed at sites far away from the cells in which they are manufactured. To prepare these molecules for their journeys, newly synthesized proteins destined for secretion are segregated into the endoplasmic reticulum (ER). Therein, dedicated machinery helps to facilitate the protein's adoption of correct protein structure. As a rule, newly synthesized proteins unable to achieve this structural milestone are confined to the ER, where they are eventually sorted into the cytosol for degradation by proteasomes.

Realizing that protein folding and deployment play critical roles in many genetic diseases, my colleagues and I asked whether selective protein destruction might directly function as an underlying etiologic agent of some genetic diseases. This notion was supported by the fact that not every individual harboring a specific mutation will develop a demonstrable clinical manifestation (*i.e.* a phenomenon coined incomplete penetrance). Furthermore, it is now generally accepted that impaired protein transport through the secretory pathway contributes to several diseases including cystic fibrosis, familial osteogenesis imperfecta, and heritable forms of chronic emphysema and childhood liver disease.



An illustration depicting how the decentralized surveillance of gene expression might be used as a site for the therapeutic intervention of diseases caused by the misfolding of secretory proteins.

The capacity of cells to distinguish between normal and misfolded proteins in the secretory pathway such that only the latter class is eliminated is paramount to the success of eukaryotic genome expression. In the early secretory pathway, opportunistic cleavage of asparagine-linked oligosaccharides by ER mannosidase I (ERManI) targets misfolded glycoproteins back out into the cytosol where they are degraded by proteasomes.

Cleavage by ERManI is thought to govern the intersection between the glycoprotein folding and degradation pathways. Although central to the substrate discrimination process, the manner by which the concentration of ERManI is regulated has long remained unknown. Importantly, the yeast homolog of the enzyme is subject to transcriptional elevation, but no such mechanism had been reported for any detectable mammalian ortholog, implying an alternative mechanism operated under basal conditions.

Our report published in the February 16 issue of the *Journal of Biological Chemistry* demonstrates that both the human and mouse orthologs of ERManI are subject to rapid proteolysis-driven down-regulation. The

Richard N. Sifers

Richard N. Sifers, Ph.D., is an associate professor at the Baylor College of Medicine in Houston, Texas, where he initiated a mechanistic analysis of conformational diseases as a National Institutes of Health (NIH) postdoctoral fellow. He is the recipient of many honors and awards, including a Research Career Investigator Award from the American Lung Association for his pioneering work on heritable α1-antitrypsin deficiency. Sifers has authored numerous primary research articles, review articles, and book chapters and organized the first international conference on the subject of conformational diseases. His lab continues to characterize the glycoprotein degradation process as it pertains to the etiology of human genetic diseases.

regulatory mechanism is reminiscent of many nuclear checkpoint proteins that delay progression through the cell cycle in response to damaged DNA. The findings are consistent with the central role played by ERManI in the regulation of a biosynthetic checkpoint that decentralizes the surveillance of eukaryotic gene expression.

Lysosomes, rather than proteasomes, are used for ERManI down-regulation, possibly to avoid complications that might arise from controlling the mannosidase's concentration by the same proteolytic system for which it functions. Moreover, the amino-terminal tail appears to play a major role in the down-regulation process, consistent with the observation that the domain is much longer in the human and mouse orthologs than in yeast.

This novel discovery underscores the importance of employing biochemical techniques to identify sites for the possible therapeutic intervention of human genetic diseases caused by protein misfolding in the secretory pathway. Drugs capable of speeding up the glycoprotein degradation rate might be used to prevent the toxicity often associated with the inappropriate accumulation of misfolded proteins. In contrast, the drug-delayed onset of glycoprotein degradation might lead to the correct folding and deployment of molecules that would otherwise fall subject to degradation under basal conditions. At the very least, the new findings are expected to provide a novel biomarker by which one's susceptibility to certain diseases might be predicted.

Sulfated Polysaccharides in Liver Clear Fats from the Bloodstream

Maybe you ate a big, juicy steak for dinner last night, adding a large amount of triglycerides to your system. For one in ten of us, that could be a big problem. Although we try to reduce fat in our diet, our bodies use it for energy. But patients with elevated levels of fat in their bloodstream–nearly 10% of Americans–are more likely to develop atherosclerosis, or build-up of plaques in the arteries, which can lead to a heart attack or stroke.

Now, researchers at the University of California, San Diego (UCSD) School of Medicine have discovered a factor that could be responsible for many unexplained cases of elevated triglyceride levels.

In humans, high levels of triglycerides can be diabetes-related, diet-induced, or caused by drug interactions or chronic alcohol consumption. The problem can also be genetic. But it turns out that another important factor is a sulfated polysaccharide called heparan sulfate.

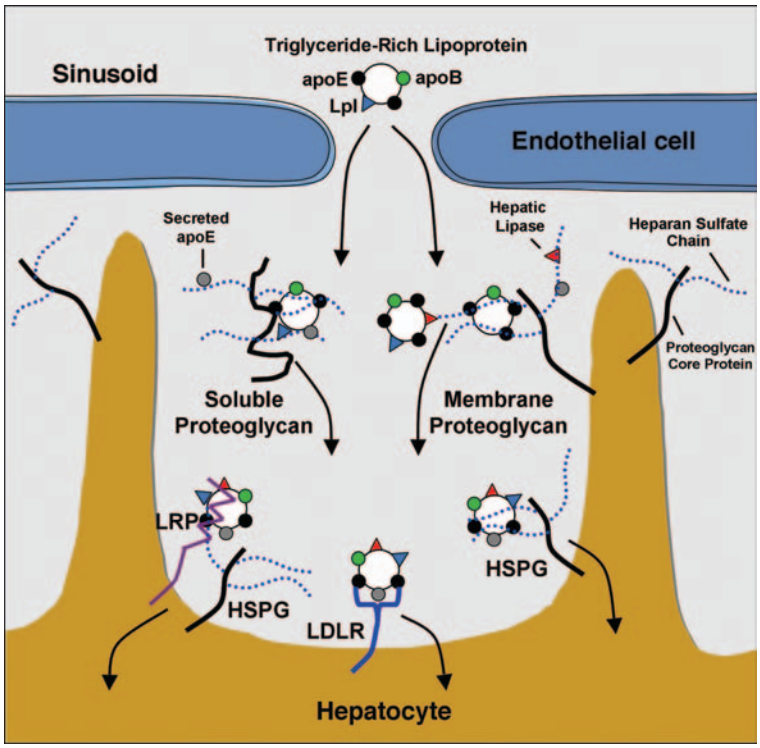
The UCSD team found that heparan sulfate in the liver helps the body clear triglycerides from the blood. Their study, published in the January 2 issue of the *Journal of Clinical Investigation*, suggests that some patients with elevated triglyceride levels could have changes in heparan sulfate in the liver. The discovery could pave the way for new therapies for a major and growing medical problem.

"The work confirms that heparan sulfate in the liver plays a crucial role in clearing fat," said Jeffrey D. Esko, professor of Cellular and Molecular Medicine at UCSD's School of Medicine. "These molecules clear tri-

Jeffrey D. Esko

Jeffrey D. Esko is professor of Cellular and Molecular Medicine and co-director of the Glycobiology Research and Training Center at UCSD. He received his B.Sc. from the University of Illinois at Urbana-Champaign (1976) and his Ph.D. from the University of Wisconsin, Madison (1980). After working as a Fellow at the Molecular Biology Institute at the University of California, Los Angeles, Esko joined the faculty of the University of Alabama at Birmingham in 1983. He remained there until 1996 when he joined UCSD.

Esko is the recipient of several fellowships, a March of Dimes Basil O'Connor Award, and a National Institute of General Medical Sciences MERIT award, and is a past president of the Society for Glycobiology. His laboratory currently studies the structure, biosynthesis, and genetics of glycoproteins and proteoglycans.



Heparan sulfate proteoglycans play multiple roles in hepatic lipoprotein clearance.

glycerides and cholesterol from the blood, working alongside the better-known low-density lipoprotein [LDL] receptors."

The UCSD researchers created a mouse model with a mutant form of heparan sulfate in their livers. They did this by inactivating the biosynthetic gene *GlcNAc N-deacetylase/N-sulfotransferase 1* (*Ndst1*) in hepatocytes using the Cre-loxP system, which resulted in an approximately 50% reduction in sulfation of liver heparan sulfate. This caused elevated triglyceride levels very much like those seen in many patients with diabetes.

The researchers then compounded the mutation with LDL receptor deficiency. This caused enhanced accumulation of both cholesterol- and triglyceride-rich particles compared with mice lacking only LDL receptors, suggesting that heparan sulfate participates in the clearance of cholesterol-rich lipoproteins as well.

Hepatocytes and endothelial cells produce membrane-bound heparan sulfate proteoglycans (HSPGs) and secrete proteoglycans into the space of Disse. After lipolytic processing of lipoproteins, apolipoprotein E (apoE)-enriched remnant lipoproteins enter the space of Disse where they are sequestered near the hepatocyte cell surface via apoE-heparin sulfate binding or lipase-heparin sulfate bridging on soluble and secreted HSPGs. Lipoproteins are further processed in the space of Disse by transfer of soluble apoE and by hepatic lipase bound via heparin sulfate.

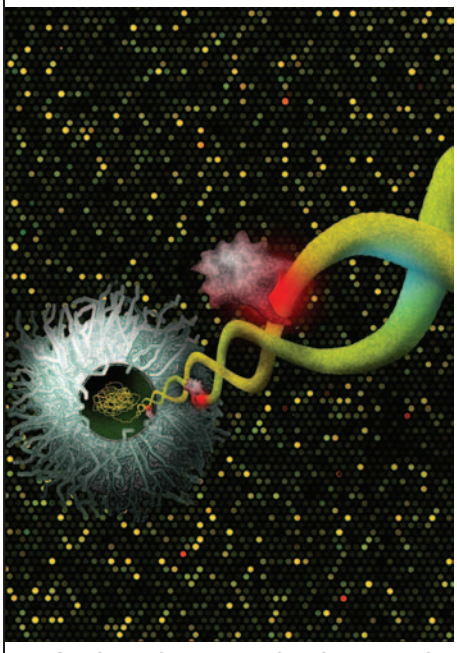
"The finding, that the LDL receptor plus heparan sulfate work together to clear triglyceride and cholesterol-rich particles from the blood in a healthy person is very exciting," Esko said. The study suggests the possibility that mutations in one of 40 or so genes involved in production of heparan sulfate in the liver could result in high blood-fat levels and lead to complications such as atherosclerosis, according to Esko.

In animal models with induced diabetes, changes in liver heparan sulfate–consistent with the UCSD researchers' findings–often appear. One of the team's next steps will be to induce diabetes in animals and examine the role of heparan sulfate in more detail. "Such studies could lead to new drugs that change heparan sulfate in order to lower fat levels in patients," said Esko.

Cracking Open the Black Box of Autoimmune Diseases

Autoimmune diseases such as type 1 diabetes, lupus, and rheumatoid arthritis occur when the immune system fails to regulate itself. But researchers have not known precisely where the molecular breakdowns responsible for such failures occur. Now, a team of scientists from the Whitehead Institute and the Dana-Farber Cancer Institute has identified a key set of genes that lies at the core of autoimmune diseases, findings that may help scientists develop new methods for manipulating immune system activity.

"This may shorten the path to new therapies for autoimmune diseases," says Whitehead member and Massachusetts Institute of Technology (MIT) Professor of Biology Richard Young, senior author on an article that was published online on January 21 in *Nature*. "With this new list of genes, we can now look for possible therapies with far greater precision."



A schematic representing the researchers' strategy to identify where Foxp3 physically interacts with the genome in T cells. The background is a microarray where the red probes reveal regions of DNA where Foxp3 is bound. *Image by Tom DiCesare.*

The immune system is often described as a kind of military unit, a defense network that guards the body from invaders. Seen in this way, T cells are the frontline soldiers of immune defense, engaging invading pathogens head on. The T cells are commanded by a second group of cells called regulatory T cells. Regulatory T cells prevent biological "friendly fire" by ensuring that the T cells do not attack the body's own tissues. Failure of the regulatory T cells to control the frontline fighters leads to autoimmune diseases.

Scientists previously discovered that regulatory T cells are themselves controlled by a master gene regulator called Foxp3. Master gene regulators bind to specific genes and control their level of activity, which in turn affects the behavior of cells. In fact, when Foxp3 stops functioning, the body can no longer produce working regulatory T cells. When this happens, the frontline T cells damage multiple organs and cause symptoms of type 1 diabetes and Crohn's disease. However, until now, scientists have barely understood how Foxp3 controls regulatory T cells because they knew almost nothing about the actual genes under Foxp3's purview.

Researchers in Young's Whitehead lab, working closely with immunologist Harald von Boehmer of the Dana-Farber Cancer Institute, used DNA microarray technology developed by Young to scan the entire genome of T cells and locate

Richard Young

Richard Allen Young is a member of the Whitehead Institute for Biomedical Research, professor in the Department of Biology at MIT, and associate member of the Broad Institute. He earned his B.S. from Indiana University (1975) and his Ph.D. from Yale University (1979). After postdoctoral fellowships at the Swiss Institute for Experimental Cancer Research (1979–1980) and Stanford University (1981–1984), he joined the Whitehead Institute.

Young has received numerous awards, including the 2006 Wilbur Cross Medal from Yale University, a National Institutes of Health (NIH) Merit Award, a Burroughs Wellcome Scholar Award, and an NIH Public Service Award.

Photo credit, Sam Ogden.

the genes controlled by Foxp3. Roughly 30 genes were found to be directly controlled by Foxp3 and one, called Ptpn22, showed a particularly strong affinity.

"This relation was striking because Ptpn22 is strongly associated with type 1 diabetes, rheumatoid arthritis, lupus, and Graves' disease, but the gene had not been previously linked to regulatory T cell function," says Alexander Marson, an M.D./Ph.D. student in Young's lab and lead author of the paper. "Discovering this correlation was a big moment for us. It verified that we were on the right track for identifying autoimmune-related genes."

Discovering this correlation was a big moment for us. It verified that we were on the right track for identifying autoimmune-related genes

The researchers still don't know exactly how Foxp3 enables regulatory T cells to prevent autoimmunity. But the list of genes that Foxp3 targets, many of which are key modulators of T cell activation and function, provides an initial map of the circuitry of these cells, which is important for understanding how they control a healthy immune response.

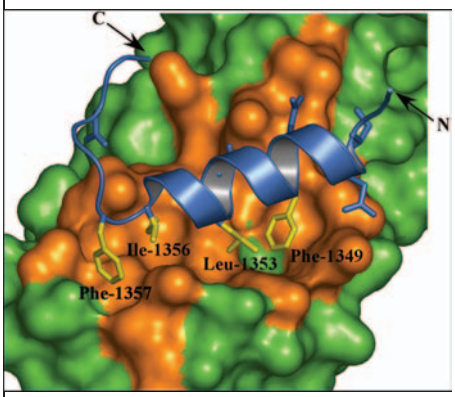
"Autoimmune diseases take a tremendous toll on human health, but on a strictly molecular level, autoimmunity is a black box," says Young. "When we discover the molecular mechanisms that drive these conditions, we can migrate from treating symptoms to developing treatments for the disease itself."

Synthetic Peptide Targets Latent Papillomavirus Infections

ROBERT SANDERS, UNIVERSITY OF CALIFORNIA, BERKELEY-OFFICE OF MEDIA RELATIONS

While a newly marketed vaccine promises to drastically reduce human papillomavirus (HPV) infections, the major cause of cervical cancer, a new discovery by University of California (UC), Berkeley researchers could some day help the millions of people already infected and at constant risk of genital warts and cancer.

Many of the 90-plus known genetic variants or strains of HPV cause epithelial lesions, but three variants, HPV-16, -18, and -31, are notorious as primary causes of cervical cancer. The viral genome is in the form of a circular DNA plasmid that resides in the nucleus of the infected cell. Each cell can house hundreds of plasmids. When the cell divides, the plasmids attach to the chromosomes via a viral protein called E2 that attaches to the cellular protein Brd4. The plasmids are then copied and delivered along with the duplicate chromosomes into the daughter cells, where they again take up residence in the nucleus as latent viral DNA. The viral plasmids turn into infectious viruses only in the top, differentiated layers of tissue.



Surface rendering of E2 in green, with residues contributing to the E2:Brd4 interaction in orange. Brd4 is depicted in blue as a ribbon diagram, and side chains making contact with E2 are shown. *Figure courtesy of the Botchan lab/UC Berkeley.*

Michael Botchan, of UC Berkeley, led a group that solved the crystal structure of the carboxy-terminal domain of Brd4 in complex with HPV-16 E2. Using this information, they developed a Brd4-Tat fusion protein that was taken up by different transformed cells harboring HPV plasmids. By tracking the plasmid DNA in dividing cultured cells, the researchers showed that the synthetic peptide prevented HPV from hitching a ride on a cell's chromosomes as the cell divides.

Because they built the peptide to enter cells easily, it has potential as a topical treatment for the viral infection. The researchers hope to partner with a biotechnology company to improve the peptide or develop better drug candidates and ideally to find a formulation that can be taken orally rather than applied topically.

Botchan, who has studied DNA replication in viruses for 30 years to understand similar processes in higher organisms, says that such a drug might work against all strains of HPV because the E2 tethering protein is similar in all the viruses. And because the E2 protein is found only in papillomaviruses, a drug that blocks it shouldn't have side effects in humans.

The study appears in the December 28 issue of the journal *Molecular Cell*.

Michael Botchan

Michael Botchan is head of the Biochemistry and Molecular Biology Division in the Department of Molecular and Cell Biology at the University of California, Berkeley and a member of the Howard Hughes Medical Institute scientific advisory board. He received his Ph.D. in Biophysics from UC Berkeley (1972) after which he was a postdoctoral fellow at Cold Spring Harbor Laboratory (CSHL). Promoted to a senior staff scientist at CSHL in 1975, Botchan was recruited to UC Berkeley in 1980. He is a fellow and member of the American Academy of Arts and Sciences.

Among other things, Botchan demonstrated that DNA tumor viruses integrate randomly into host chromosomes, developed the first methods for "single copy" detection of DNA via blotting methods, and was the first to define a metazoan origin recognition complex.

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Dr. Kenneth G. Mann, Biochemistry Department, University of Vermont, College of Medicine, 208 South Park Dr., Suite 2, Room T227, Colchester, VT 05446 or e-mail to kenneth.mann@uvm.edu.

meeting calendar

MARCH 2007

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www.scripps.edu/imm/curtiss/deuel/index.html

Cell Signaling and Proteomics
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www.keystonesymposia.org/Meetings/

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233rd American Chemical Society National Meeting
March 25–29, 2007
CHICAGO, IL
chemistry.org/meetings/chicago2007

Keystone Symposia–Metabolic Syndrome and Cardiovascular Risk
March 27–April 1, 2007
STEAMBOAT SPRINGS, CO
www.keystonesymposia.org

RNAi2007: The Expanding Roles of Small RNAs
March 29–30, 2007
ST. ANNE'S COLLEGE, WOODSTOCK ROAD, OXFORD, UK
www.libpubmedia.co.uk/Conferences/RNAi2007/Home.htm
Organizer: Dr. Muhammad Sohail
E-mail: Muhammad.sohail@bioch.ox.ac.uk
Tel: 44(0)1865-275231

APRIL 2007

3rd European Symposium on Plant Lipids
April 1–4, 2007
YORK, UK
www.eurofedlipid.org/meetings/index.htm

Second Workshop on Biophysics of Membrane Active Peptides
April 1–4, 2007
LISBON, PORTUGAL
www.biophysicsmap.com

Arteriosclerosis, Thrombosis and Vascular Biology Annual Conference 2007
April 19–21, 2007
CHICAGO, IL
www.americanheart.org/presenter.jhtml?identifier=3039918

2nd International Congress on Prediabetes and the Metabolic Syndrome
April 25–28, 2007
BARCELONA, SPAIN
www.kenes.com/prediabetes2007
E-mail: prediabetes2007@kenes.com

ASBMB Annual Meeting in Conjunction with EB2007
April 28–May 2, 2007
WASHINGTON, DC
www.asbmb.org/meetings
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Tel.: 301-634-7145

MAY 2007

7th International Symposium of the Protein Society
May 12–16, 2007
STOCKHOLM-UPPSALA, SWEDEN
www.proteinsociety.org/pages/page02b.htm
E-mail: cyablonski@proteinsociety.org
Tel.: 301-634-7277

94th Annual Meeting the American Association of Immunologists
May 18–22, 2007
MIAMI BEACH, FL
www.immunology2007.org/

National Lipid Association Annual Scientific Sessions
May 31–June 3, 2007
SCOTTSDALE, AZ
www.lipid.org/chapters/swla

Epistasis: Predicting Phenotypes and Evolutionary Trajectories
May 31-June 3, 2007
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JUNE 2007

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www.lifesciences2007.org/

Gordon Research Conference-Molecular and Cellular Biology of Lipids
July 22–27, 2007
WATERVILLE VALLEY, NH
www.grc.org

4th British Society for Proteome Research/European Bioinformatics Institute Proteomics Meeting
Integrative Proteomics: Maximizing the Value of Proteomics
July 25–27, 2007
WELLCOME TRUST CONFERENCE CENTRE, HINXTON, CAMBRIDGE, UK
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E-mail: meetings@bspr.org

Senescence, Aging and Cancer Symposium
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IOWA STATE UNIVERSITY, AMES, IA
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August 1–4, 2007
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www.smp-2007.com/

FASEB Summer Research Conference-Lipid Signaling Pathways in Cancer
August 11–16, 2007
INDIAN WELLS, CA
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8th International Symposium on Mass Spectrometry in the Health & Life Sciences
August 19–23, 2007
FAIRMONT HOTEL, SAN FRANCISCO, CA
www.donatello.ucsf.edu/symposium/
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234th American Chemical Society National Meeting
August 19–23, 2007
BOSTON, MA
chemistry.org/meetings/boston2007

13th Nordic Mass Spectrometry Conference
August 28–31, 2007
SAVONLINNA, FINLAND
www.nsms.no/moter.html

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

XVI International Symposium on Drugs Affecting Lipid Metabolism
October 4–7, 2007
NEW YORK, NY
www.lorenzinifoundation.org/download/dalm2007.pdf

HUPO 6th Annual World Congress
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ART-0108A	Galactose, D-[1-^{3}H(N)]	1 mCi	$499
ART-0109A	Galactose, D-[6-^{3}H(N)]	1 mCi	$499
ARC-0118A	Glucosamine hydrochloride, D-[1-^{14}C]	250 µCi	$799
ARC-0120A	Glucose, D-[1-^{14}C]	1 mCi	$1699
ARC-0147	Glucose, D-[2-^{14}C]	50 µCi	$699
ARC-0121B	Glucose, D-[6-^{14}C]	250 µCi	$1049
ARC-0122B	Glucose, D-[^{14}C(U)]	250 µCi	$499
ART-0111	Glucose, D-[1-^{3}H(N)]	5 mCi	$799
ART-0112	Glucose, D-[2-^{3}H(N)]	5 mCi	$849
ART-0115	Glucose, D-[5-^{3}H(N)]	1 mCi	$699
ART-0113	Glucose, D-[6-^{3}H(N)]	5 mCi	$599
ART-1299	Glucosylphosphoryldolichol, [glucose-1-^{3}H]	10 µCi	$1199
ART-0440	Guanosine Diphosphate, GDP [mannose-2-^{3}H]	50 µCi	$1099
ARC-1582	Guanosine Diphosphate Glucose, [α-D-glucose-^{14}C(U)]	10 µCi	$1849
ARC-0440	Guanosine Diphosphate Mannose [mannose 2-^{3}H]	50 µCi	$1099
ARC-0116	Inositol [2-^{3}H(N)]	1 mCi	$729
ARC-0127	Mannitol, D-[1-^{14}C]	250 µCi	$1049
ART-0118	Mannitol, D-[1-^{3}H(N)]	5 mCi	$899
ARC-0129	Mannose, D-[1-^{14}C]	250 µC	$1399
ART-0120	Mannose, D-[2-^{3}H(N)]	5 µCi	$899
ARC-0322	Methyl-β-D-thiogalactopyranoside, [methyl-^{14}C]	50 µCi	$849
ARC-0221	Octyl-β-D-glucopyranoside, [glucose-^{14}C(U)]	50 µCi	$1099
ARC-0157	Ribitol, D-[1-^{14}C]	50 µCi	$899
ARC-0137	Ribose, D-[1-^{14}C]	50 µCi	$659
ART-0152	Ribose, D-[1-^{3}H]	1 mCi	$799
ARC-0143A	Sucrose, [^{14}C(U)]	1 mCi	$1099
ART-0156	UDP N-acetyl-D-galactosamine, [6-^{3}H(N)]	50 µCi	$649
ARC-1379	UDP N-acetyl-D-galactosamine, [1-^{14}C]	10 µCi	$1199
ART-0131	UDP galactose, [galactose-6-^{3}H]	250 µCi	$749
ARC-0151	UDP Glucose [glucose ^{14}C(U)]	5 µCi	$749
ART-0525	UDP Glucose [glucose-1-^{3}H]	250 µCi	$749
ARC-1744	Xylitol, D-[1-^{14}C]	250 µCi	$799
ARC-0525	Xylose,D-[1-^{14}C]	50 µCi	$699

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