

SEPTEMBER 2006

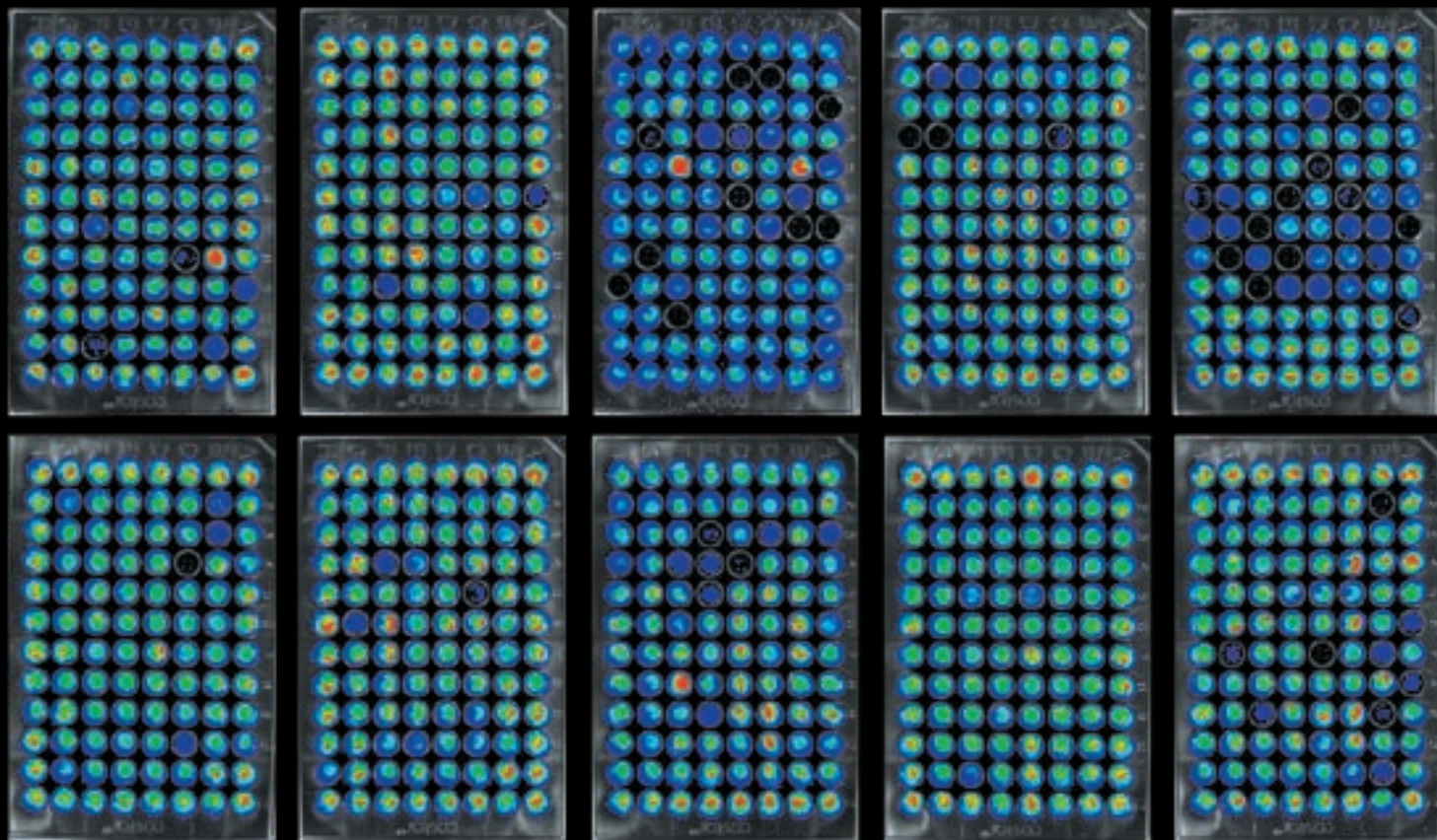
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ASBMB *Today*

Constituent Society of FASEB

AMERICAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY

ASBMB President:
Whither the R01 and the
Individual Investigator?



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

Biomarker Discovery and Clinical Proteomics

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

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

This special issue will include articles from presentations at the 2005 Asilomar Conference on "Biomarker Discovery and Clinical Proteomics" organized by Steven Carr and Leigh Anderson,

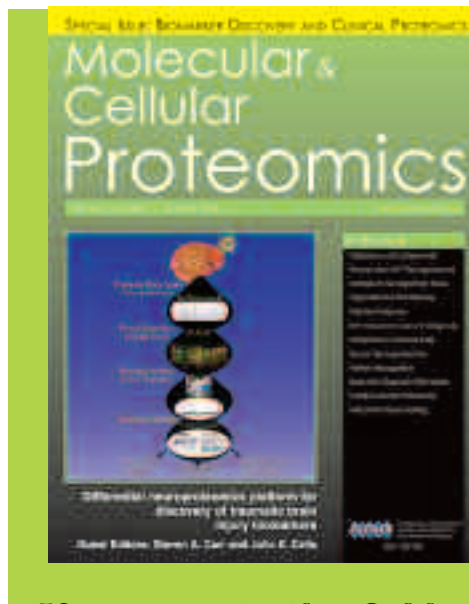
several invited contributions, as well as four research reports selected from direct submissions to the journal. The issue is organized in three sections covering the following topics: 1) biomarkers of disease and conditions, 2) proteomic data analysis, and 3) methodologies.

**Issue
Date:
October
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snapshot of accepted papers

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



If you are in the field of Proteomics or a Clinician interested in biomarkers

you cannot afford to miss this issue!



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special clinical issue

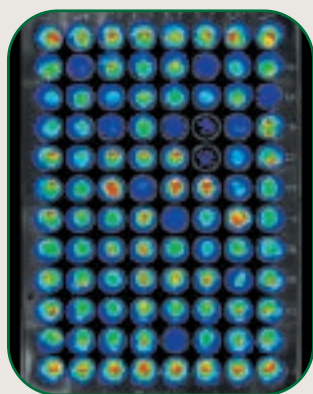
ASBMB Today

AMERICAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY

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Volume 5, Issue 6

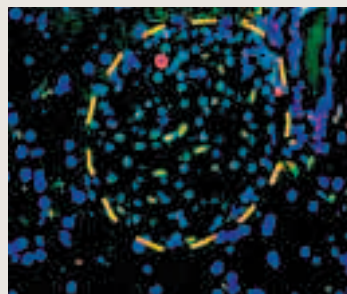
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AWARDS OF EXCELLENCE:

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Please direct any comments or questions
concerning *ASBMB Today* to:

John D. Thompson
Editor, *ASBMB Today*
9650 Rockville Pike
Bethesda, MD 20814-3996
Phone: 301-634-7145; Fax: 301-634-7126
E-mail: jthompson@asbmb.org

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From the Desk of the President:

Whither the R01 and

Many scientists are asking, why success rates for grant proposals are declining (from 32% during the doubling to 21% in 2005)? A common misperception is the so-called “big science” approach of the NIH Roadmap. But the Roadmap received slightly over \$300 million in 2006, hardly a crippling amount of money in a near \$30 billion budget. In this editorial, we will attempt to answer this question with evidence for a subtle, but nevertheless important trend towards NIH-solicited research.

Many biomedical scientists agree that the most vital contributor to the American preeminence in biomedical research is investigator-initiated, rather than NIH-solicited, research. The R01 mechanism has been hugely successful in advancing biomedical research and has been the predominant funding mechanism at the NIH. However, in recent years there appears to have been a shift in emphasis.

Last month *ASBMB Today* featured a lengthy interview with NIH Director Elias Zerhouni. We are very grateful to Dr. Zerhouni for providing us with so much thoughtful commentary on issues involving NIH that affect ASBMB members and biomedical research. This interview was an outgrowth of a face-to-face meeting we had with Dr. Zerhouni in early June. During that meeting, it became clear that the major area where we had differing perceptions regarding was in the role and treatment by NIH of grants submitted under the Program Announcement (PA) mechanism.

From data we have seen, it appears that grants funded under the PA mechanism account for approximately 20% of all R01s, while grants funded under Requests for Applications (RFA) make up about 10% of R01s. While funding for grants originating from the RFA mechanism is still somewhat stable, it appears that the percentage of grants

funded under the PA mechanism is steadily increasing. Here are some numbers addressing this point:

Competing R01s as a proportion of Research Project Grants (RPG) are down from a high of 75% in 1995 (\$1.2B R01s of \$1.6B RPG) to 65% in 2005 (\$2.2B of \$3.4B) (Fig. 1). Add to that the increased grants funded in response to PAs in 2005 (Fig. 2), and it is clear that there has been significant erosion in investigator-initiated grants. At a point when R01s were down from their high (7,255 competing awards in 2003 to 6,275 in 2005), the number of R01s funded through PAs increased dramatically AFTER the doubling (from 654 in 2003, to 888 in 2005), raising questions regarding the recent emphasis on this mechanism of soliciting grant applications. See charts on next page.

There are a number of reasons why we consider this to be an important issue. The NIH leadership considers R01s originating from PAs to be investigator-initiated and counts them as such in statistical data. The NIH reasoning is that 1) PAs are usually very broadly worded which allow for much flexibility from investigators responding to them; and 2) there is no money set aside to fund grants originating under PAs, unlike the case for those that fall under RFAs. Furthermore, PA grant applications have to compete with all other grants since there is not a set-aside to fund these applications alone.

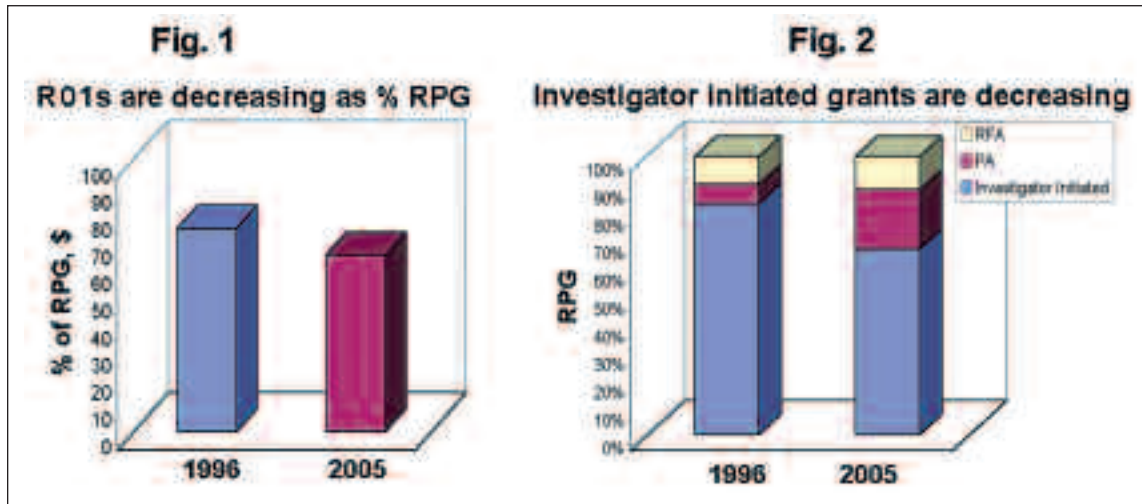
We have considered this rationale and find it debatable on several points. First, NIH itself defines PAs as “requested” by the agency in a glossary on the NIH website. The glossary defines a program announcement as “An announcement by an NIH Institute or Center requesting applications in the stated scientific areas.” The definition goes on to note that usually money is not set aside to pay for applications received in response to PAs.

the Individual Investigator?



Dr. Heidi E. Hamm

ing. Given this circumstance, investigators have no other choice than to write more applications. This is not intended as a criticism of the NIH leadership, who have done the best



However, in 1996 the NIAID "instituted a policy through which some applications responding to a program announcement with percentiles beyond the pay line will be funded." Thus, in at least one institute, applications responding to PAs will be favored and have an easier time being funded than standard R01s.

Second, the fact that money is not set aside to fund a PA is largely irrelevant to the issue of whether or not the research is investigator-initiated. The fact that a PA "requests" applications in a specific scientific area has the clear effect of driving science in specific directions at the instigation of NIH staff, thus competing with funds for unsolicited applications funded through the standard R01 investigator initiated mechanism.

Third, after an informal review of the titles of PAs over the past several months that appear in the weekly NIH Guide to Grants and Contracts we conclude that while some of the PA titles are broadly worded, the fact is that many—a majority, in fact—are quite specific on concerning the type of research being requested. Thus, we are seriously concerned that a consistently growing percentage of NIH research is being directed by NIH program directors, rather than by the historical norm of unsolicited grants emanating from

the extramural community. Clearly, given the current funding climate, extramural researchers will respond to PAs, encouraged to change their science to conform to the scientific directions requested. These practices will clearly have an influence on the type of science undertaken and the direction of science as a whole. When only about 50% of the NIH budget goes to fund extramural research, it is vital that every effort be made to maintain funding for truly unsolicited grants, since we all agree that historically the unsolicited grant has a magnificent track record of discovery and has been chiefly responsible for NIH's remarkable success.

A related problem with PAs is that the NIH appears to have increased the number of calls for applications. This aggravates the fact that a vastly increasing number of grant applications are already being submitted due to the increased number of researchers applying. In a climate when more researchers are applying, and more of these are writing more grants in response to PAs, the problem can only be compounded. We thus find ourselves in a vicious cycle with undifferentiated study sections lacking the proper expertise (discussed in a previous editorial) triaging many applications from successful investigators, and with success rates plummet-

they can under very difficult circumstances given the flat funding over the past several years. Importantly, we recognize that individual institutes have very different percentages of investigator-initiated versus solicited grants in their portfolios. For example, the NIGMS is the champion of investigator-initiated research with more than 90% of competing R01s (and 80% of RPGs) in 2005 going to unsolicited applications. This is likely the reason that the NIGMS is so successful in producing such an impressive stable of award winning scientists over the years.

All of these data lead to the conclusion that there is a serious imbalance in the funding of individual scientists and investigator-driven science. Thus, we believe that we should work closely with the NIH and institute directors to examine the allocation of resources given to investigator-initiated R01s, and boost this category significantly over the coming years.

The fundamental problem is that NIH is trying to cope with many competing demands on its limited resources in an environment of flat funding where the purchasing power has declined by more than 10 % since the end of 2003. Unfortunately, no one—at either NIH or in the extramural community—seems to

Continued on page 5

by Peter Farnham, CAE, ASBMB Public Affairs Officer

Funding at NIH Still Flat as Congress

Congress recessed in early August for the traditional summer work period, leaving a spate of unfinished business behind, and the prognosis for science funding is, at best, mixed now that House and Senate members have returned for the expected brief session between now and the election (although a post-election “lame duck” session is almost a certainty). First, the good news.

Before leaving Washington at the beginning of August, Congress moved a step closer to enacting large increases for physical sciences funding agencies under the Administration’s American Competitiveness Initiative (ACI), proposed last February during the State of the Union message. Both the House and the Senate propose to fully fund requested increases for three key physical sciences agencies. The National Science Foundation would receive a 7.9 % increase to \$4.5 billion; the Department of Energy’s Office of Science would increase 18% to \$3.9 billion; and the Commerce Department’s National Institute of Standards and Technology laboratory research would increase 21% to \$382 million.

But Little Good News for NIH

Congress also plans to add money to some basic and applied research programs the administration had targeted for cuts this year, but the federal investment in basic and applied research would still decline in FY 2007 under separate House and Senate plans that must be reconciled in the fall. Congress is planning to slash homeland security R&D funding for the first time, and to make steep cuts in other federal research portfolios. Unfortu-

nately, they apparently plan to keep the National Institutes of Health (NIH) budget flat or declining for the second year in a row when inflation is factored into the picture.

After the large increases that came during the 5-year doubling of NIH’s budget that ended in 2003, NIH research funding would fall short of inflation for the third year in a row under both the House and Senate plans. The House Appropriations Committee proposes to fund NIH at the Administration’s request level, which is the same as the funding it received last year.

The Senate would add \$201 million to the Administration’s request, enough to keep NIH institute and center budgets flat instead of declining for the second year in a row (NIH received an actual cut of \$70 million last year, the first time the budget had been cut since 1970).

According to an analysis prepared by the American Association for the Advancement of Science, “The 2007 Senate appropriation would be a 0.7 percent increase for a total NIH budget of \$28.8 billion. The modest Senate increase would keep NIH well behind its own calculations of biomedical research inflation, estimated at 4.1 percent this year and 3.8 percent in 2007. The NIH budget would fall 10 percent from 2004 to 2007 based on these calculations, and nearly 5 percent based on economy-wide inflation.”

In the House, a great deal of contentious debate occurred in July over a provision to raise the minimum wage, which prevented the Labor/HHS bill from reaching the floor before the August recess. However, it is still

unlikely that the LHHS bill will reach the House floor before the election, and maybe not even then. The House leadership is unwilling to bring the bill to the floor without an additional \$3 billion for discretionary programs promised to a group of Republican moderates led by Rep. Mike Castle (R-DE) in order to gain their support for the draft bill. Without the promised additional funding, the bill is very likely to be defeated, as the two dozen or so moderate Republicans Castle leads have effective veto power on the floor by simply siding with democrats.

Nor is the Senate expected to bring its bill to the floor without an additional \$2 billion. Thus, there is an increasing likelihood that Congress will fund the NIH through a continuing resolution in the fall, or possibly through an omnibus appropriation bill during the likely “lame duck” session, expected to begin on November 14.

On a slightly more optimistic note, ASBMB staff met with House Majority Leader John Boehner (R-OH) in July under the auspices of the Campaign for Medical Research (of which ASBMB is a member). Boehner, the second most powerful Republican in the House, seemed to understand the situation in which NIH is currently languishing. He noted that the leadership had made a promise to the Castle moderates to find the needed money and indicated they intended to meet that commitment even though it was not obvious where the money to do so was going to come from. He also observed in response to a question that it “made sense” to keep the NIH whole after all the effort Congress had made to double the agency’s budget.

Returns from August Break

Earmarks up this year

As might be expected in an election year, Congress is well on its way to matching last year's record-breaking total of R&D earmarks. So far, both House and Senate would designate \$2.4 billion for congressionally designated performer-specific R&D projects in FY 2007. This equals the combined total of all of last year in both houses.

However, not all legislators are going along with this congressional tradition. Senator Tom Coburn (R-GA) has sent a letter to over 100 major research universities asking for information on all appropriations they have received since 2000. The letter, sent on July 27, asks a number of questions related to earmarks the universities have received, including a summary of the "specific objectives or goals set to be achieved" by each earmark and "a list of accomplishments that can be attributed" to the earmark, such as published peer-reviewed research.

The letter also asks each institution to describe its "stated policy regarding Congressional earmarks or appropriations" and whether the institution has ever "considered hiring a lobbyist to assist your institution in attaining familiarity with the opportunities that may exist to obtain Federal funds for research—such as the earmarking process?"

While the letter is a non-binding request (it is not a subpoena), a number of the letter's recipients have expressed considerable dismay at receiving it.

FASEB in general opposes specific earmarks in the funding of scientific research, instead supporting allocation based on peer review. In a recent story in the publication, *Inside Higher Ed*,


FASEB Public Affairs Director Howard Garrison said that FASEB opposes earmarks, except in some specific capacity-building instances. The National Science Foundation's Experimental Program to Stimulate Competitive Research, for example, supports competitive grants in states that haven't historically received big federal research dollars. Generally, to build research capacity, Garrison said, "you don't need earmarks, what you need is committed funding and insightful leadership from the states."

Tensions Growing in Extramural Community

As funding becomes more problematic for many biomedical researchers, efforts are increasing to generate more activism in the community. FASEB President Leo Furcht said on August 9 that "It is time to reeducate Congress and the public about the critical value of NIH. There's overwhelming support for medical research—everyone looks forward to the next breakthrough, the next new treatment. We just need to make the connection between lifesaving advances and funding of the National Institutes of Health."


FASEB has announced a renewed grass roots campaign to convince Congress through the local community of the value of NIH. To kick-off the campaign, FASEB has produced a customizable slide presentation that scientists, department heads and deans can use locally to demonstrate NIH's impact on human health (this presentation is available for viewing on ASBMB's homepage). FASEB is creating versions of the presentation for every state.

"Nothing is more important than the health and well-being of the American people. We are all only one diagnosis away from needing the hope that NIH embodies," Furcht continued. "It is our obligation, as a scientific community, to explain how science is done—to explain how continued improvements in human health are dependent on a sustained commitment to NIH. Supporting medical research in concept is no longer enough."

ASBMB has joined this effort by creating a page on its website called "Advocating for Science—Resources You Can Use." The initial FASEB advocacy presentation is posted there, as well as a number of other resources that ASBMB members can use and freely download as they engage in advocacy efforts with their Members of Congress and Senators. For more information on ASBMB's efforts to boost NIH funding, please see ASBMB President Heidi Hamm's article in this issue of *ASBMB Today* called "Whither the R01 and the Individual Investigator." 

Continued from page 3

have anticipated such a draconian shutdown. It is incumbent on us, the community that recognizes the significance of the NIH in keeping Americans healthy, to make the case to those who are fundamentally responsible for NIH's current plight—our elected officials in the United States House of Representatives and the Senate.

We are currently working with ASBMB members to make this case in each and every congressional district. Next month's editorial will give a progress report on this grass-roots campaign. 

Heidi E. Hamm
ASBMB President



FASEB Receives Grant to Explore Conflict-of-Interest in Research

FASEB has received a grant of more than \$112,000 from the Office of Research Integrity (ORI) to proactively address the issue of conflict-of-interest management in biomedical research. The grant, funded through a collaborative program between ORI and the Association of American Medical Colleges, is a continuation of the work detailed in a recently released report by FASEB, *Shared Responsibility, Individual Integrity: Scientists Addressing Conflicts of Interest in Biomedical Research*. "There is a clear need for voluntary standards for the conduct of academia-industry interactions from the scientists' perspective," according to Laura Brockway, Ph.D., senior science policy analyst for FASEB and principal on the award.

Leo Furcht, M.D., who took office as the 91st FASEB President on July 1, 2006, chaired the committee that developed the report. "We have clearly entered a new era in which interactions between academia and industry are being accompanied by public concern and scrutiny," said Furcht. "FASEB has generated a set of guiding principles for investigators to address challenges as a result of financial relationships with industry."

The ORI grant funding will be used to expand this work by convening a coalition of major academic stakeholders to manage the next phase. This group will include representatives from the institutional leadership community and other scientific societies that have interest in this issue. Over the course of one year, FASEB will develop and implement activities to achieve two major goals: 1) raise awareness of conflict-of-interest issues on the part of investigators and 2) develop more standard practices for conflicts of interest management in biomedical research. Proposed activities include developing tools for investigators and laboratories and developing a model conflict of interest disclosure form. A

conference will be held to unveil these products to the community in order to achieve national buy-in and begin the process of implementation. The report can be accessed on FASEB's website at: opa.faseb.org/pdf/FASEB_COI_paper.pdf

Academic-industry interactions will be only one set of issues among many that Furcht will be addressing during his year-long tenure as FASEB President. "The current federal funding situation and its effect on researchers, stem cell research policies, reauthorization of NIH, impact of regulatory burden – these are all topics on which FASEB will remain engaged," he said. "FASEB plays a critical role in bringing the perspective of the working scientist to Congress and the public, and I look forward to continuing to be a part of that." The report was released at an event at the National Press Club on July 14, resulting in coverage by *Science*, *Nature*, *C&E News*, *The Scientist*, and other media organizations

throughout the country.

Furcht is currently Allen-Pardee Professor of Cancer Biology at the University of Minnesota and head of the Department of Laboratory Medicine and Pathology. Dr. Furcht's research interests include cell adhesion molecules and tumor metastasis. Previously, he served as Vice Provost/Vice President for Research, Academic Health Center, University of Minnesota (Health Science Schools). Dr. Furcht received his B.S. from Columbia University and his M.D. from SUNY Upstate Medical Center, followed by a residency at the University of Minnesota. In addition, he is chairman of the Board of Directors, University of Minnesota Physicians.

FASEB also congratulates Robert E. Palazzo, Ph.D., who was voted FASEB President-Elect. Dr. Palazzo is director of the Center for Biotechnology and Interdisciplinary Studies and Acting Provost at Rensselaer Polytechnic Institute.

Department Heads Take Note:

ASBMB Offers Free Membership to New Ph.D.s

ASBMB is now offering a free one-year Associate membership to all students who have, within the past year, earned a Ph.D. degree in the molecular life sciences or related areas.



Membership in ASBMB brings with it a free subscription to the online versions of the *Journal of Biological Chemistry* and *Molecular and Cellular Proteomics*, and *ASBMB Today*.

In addition, we are asking department chairs to provide ASBMB with the names and addresses of each new Ph.D. recipient from their institutions, so that we can congratulate them on their accomplishment and offer them the free one-year membership in ASBMB.

Please email to: membership@asbmb.org or visit www.asbmb.org for more information.

August JLR Includes Patient Oriented Research Reviews

The *Journal of Lipid Research* (JLR) recently initiated a new category for submitted manuscripts called “Patient-Oriented Research Articles.” To celebrate this initiative, the Journal published a series of articles that review major areas of patient-oriented lipid and lipoprotein, nutrition, and atherosclerosis research. The eight thematic reviews were coordinated by Associate Editor Henry N. Ginsberg and appeared in the August issue of the journal. The reviews are available for free on the JLR website.

The new JLR category covers research articles containing studies in which human subjects play an important role and at least one of the authors has had direct contact with the subjects. Ginsberg explains, “In recent years, fewer patient research articles have been submitted and, I believe, there was a growing feeling among patient-oriented investigators that the JLR was not as receptive to such work as it once had been. The editors and editorial board members felt, therefore, that it was important to dispel such feelings and open our journal, in a formal way, to patient-oriented research. I am extremely pleased to have been able to participate in this effort and to organize this ‘special’ issue.”

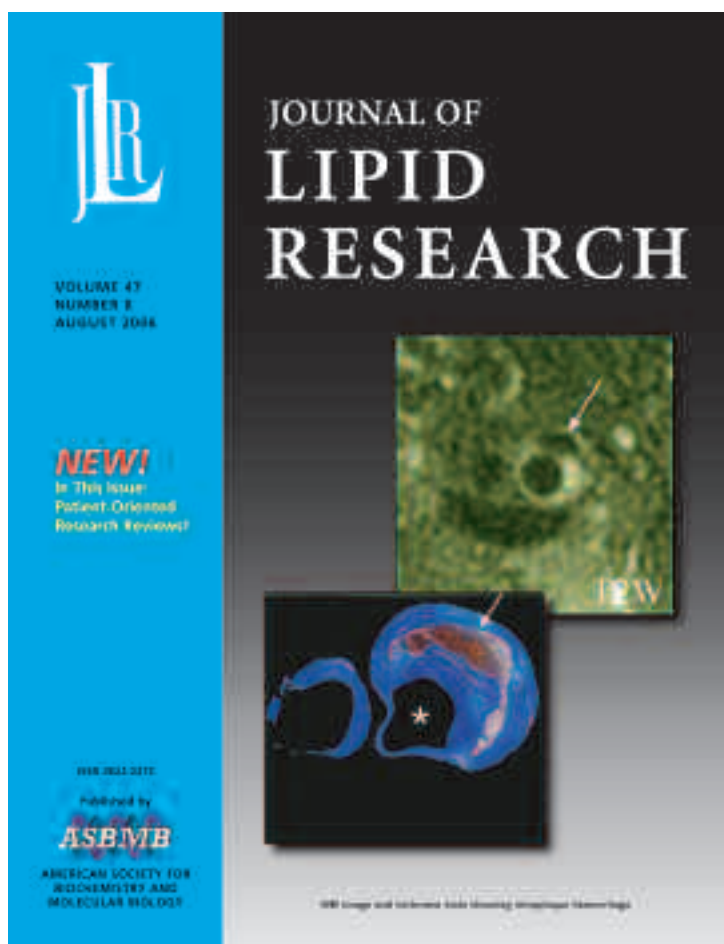
The authors of each JLR thematic review were asked not only to provide an overview of the published work in their respective areas but to offer

1961. In reviews by Klaus Parhofer and Gary Lewis and their colleagues, we hear about recent studies of VLDL, LDL, and HDL metabolism. Next,

Michael Jensen discusses free fatty acid metabolism in human obesity. Elizabeth Parks and Marc Hellerstein take us inside the human liver to provide insights regarding the sources of VLDL and hepatic triglycerides, combining multiple tracers and mathematics to move science forward. A review of the effects of dietary nutrients on lipid metabolism by Alice Lichtenstein and a review of dietary nutrient effects on insulin resistance by Jim Mann are also included. Finally, John Crouse provides an update on the non- or minimally-invasive imaging of atherosclerosis.

“I believe that this collection of reviews, which the JLR plans to publish in a separate format, is a treasure chest of information,”

says Ginsberg. “The authors have given us most of the relevant data in each area but, more importantly, have surrounded their literature reviews with honest and understandable presentations of the strengths and weaknesses of what they and their colleagues do as they search for patient-relevant knowledge.”



insights regarding the difficulties inherent in each area of investigation. The areas chosen for review ranged from metabolism of lipids and lipoproteins to imaging of atherosclerosis.

In his review, Hugh Barrett introduces the theory and practice of lipoprotein kinetics, a field that made its appearance in the JLR in January,

Careers at the Interface of Biology

By Brad Smith

Biology will have a bigger impact on the events of the 21st century than physics and chemistry did on the 20th century. While this statement is provocative and perhaps impossible to prove, it cannot be denied that the most significant challenges of the 21st century—security, health care, over-population, under-nourishment, infectious disease, sustainable energy, and economic development, among others—will be significantly impacted in the coming decades by biological research performed in laboratories around the world.

What does this mean for the politicians and civil servants that craft and implement public policy? It means that individuals with biological expertise must have a seat at the political table as policies are debated and implemented. However, apart from narrowly defined issues, such as health care, biology has not been historically viewed as central to public policy or the activities of government. As a result, there too few people with biological expertise in government and in the broader policy community given the impact that biology will have in the coming years. What does this mean for biologists? It means that the community must be highly proactive in its efforts to educate policy makers about science and to inform scientists about the policy process. Biologists must actively participate in these broad policy debates so that the stage can be set for society to benefit fully from 21st century biology.

There are many ways to engage in the public policy process as a scientist. After earning a Ph.D. in molecular biology, I chose to step away from the lab bench and begin a full-time career working at the interface of “science policy,” health policy, and security policy. I now work at a “think tank” affili-

ated with an academic medical center, where my colleagues and I focus on developing effective medical, public health, and scientific policies to improve the preparedness for, and response to, emergent epidemics of infectious disease, be they natural pandemics or bioterrorist attacks.

However, my career path is just one example of a career in science policy—there are many outlets, such as think tanks, academia, advocacy groups, philanthropic foundations, scientific societies, the Congress, and the executive branch (e.g. the White House and other departments and agencies). Each of these have distinct characteristics that need to be weighed when considering a career. In a think tank or other non-governmental organization, you have considerable flexibility and can look years into the future, but generally don't have the authority to actually implement any of the solutions you develop. Implementation is the role of government—where you can actually “do” things. However, in government, you generally are so busy that you can barely look ahead to next week and your activities can be constrained by the policies and philosophies of your superiors—ultimately the President if you work in the executive branch, or a Member of Congress if you work in the legislative branch. While my experience is primarily with U.S. federal policy-making, state and local governments can also be a good place to participate in the science policy process, especially as states look to biotechnology as an engine of economic development (e.g. California's recent efforts to fund stem cell research).

In spite of the variety of work environments, there are some generaliza-

tions that can be drawn about working in science policy and the transition from the lab bench. The world of public policy is a marketplace of ideas; basic concepts, core strategies, and even full-fledged policies circulate incessantly throughout the community. New ideas are added; old ones fade away, but sometimes return years later. This mix is continually buffeted by current events, the media, scientific discoveries, and politics. Occasionally, an idea emerges, surpasses all its competitors, and becomes real. What does this mean for a biologist contemplating a move to science policy? Primarily, you have to be able to communicate effectively (and repeatedly) in writing, at the lecture podium, and in one-on-one conversations. You may develop brilliant new policies (and the analytical skills that are at the core of a scientific education are extremely helpful in this regard), but if you can't communicate your ideas to audiences large and small, you will have a limited impact on the real world. In the realm of public policy, real world results are what matter.

One aspect of work in policy that differs from scientific research is that success is generally much less direct than it is at the lab bench. In the lab, when you do an experiment, you will, in a defined amount of time, get results that support your hypothesis—or not. You can then plan your next experiment in a deliberative fashion. In science policy, rarely do your activities lead to a clear result. In fact, you usually have to say the same thing over and over for months or years—it takes a long time to turn the ship of state. Then one day someone in authority (e.g. the President, a Member of Congress, or other opinion leader) will say exactly what you've been say-



Dr. Brad Smith


and Public Policy

ing, but they will likely not reference you or acknowledge your contribution as one would expect in the scientific community. Nevertheless, this is a huge success. And you have to be poised to rapidly take advantage of that success by building on the new-found acceptance of your ideas.

One of the greatest benefits of a career in science policy is the interdisciplinary nature of the public policy enterprise. Bench science is certainly interdisciplinary, but even if you are working with geneticists, biochemists, and structural biologists, everyone has a very similar core background and set of experiences. Work in science policy routinely means collaborating not only with scientists, but also politicians, public health experts, economists, public advocates, business

executives, legal experts, social scientists, reporters and physicians, among others—a group with a far more diverse set of agendas, worldviews, and personal histories than you would likely find in any scientific research building. Working in such a diverse environment has its challenges, but leads to a much broader view of the world and of science's place in society. After all, effective science policy can only be formulated by honest conversation among researchers, policy makers and the public.

The key to successfully building a public policy environment that will allow the nation—and the world—to safely and effectively harness the power of biology will be a partnership between scientists working at the bench and scientists working in the

policy community. One cannot succeed without the other, and together they can constructively engage with the entire public policy landscape, with all its diverse constituencies, and work collaboratively to change the world for the better. Insuring security, improving health, and raising economic standards won't be easy, but it is definitely worth the struggle. 

Brad Smith, PhD—a molecular biologist and policy analyst—is an Associate at the Center for Biosecurity of UPMC, and an Assistant Professor at the University of Pittsburgh School of Medicine. Dr. Smith is also an Associate Editor of the peer-reviewed journal “Biosecurity and Bioterrorism: Biodefense Strategy, Practice, and Science.” He can be reached at bsmith@upmc-biosecurity.org.



Massachusetts Institute of Technology

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BIOLOGICAL ENGINEERING DIVISION

Assistant Professor

The MIT Biological Engineering Division invites applications for a tenure-track faculty position at the assistant professor level, to begin July 2007 or thereafter. Applicants should hold a Ph.D. in a science or engineering discipline related to biological engineering. In special cases, a more senior faculty appointment might be possible. The candidate is expected to integrate strong expertise in molecular/cellular bioscience with an engineering design perspective; example areas of application might include stem cell technologies, therapeutics development, biomolecular materials, tissue engineering, or synthetic biology. We especially encourage minorities and women to apply, because of MIT's strong commitment to diversity in engineering education, research and practice.

Interested candidates should send application materials to: be-fac-search@mit.edu. Each application should include: a curriculum vitae; the names and addresses of three or more references; a strategic statement of research interests; and a statement of teaching interests specifically in the context of the Biological Engineering graduate and undergraduate educational programs at MIT. (<http://web.mit.edu/be/education/> and <http://web.mit.edu/be/education/ugrad.htm>)

We request that each candidate arrange for the reference letters to be sent directly to: be-fac-search@mit.edu with a copy mailed or faxed to the following address: Professor Paul Matsudaira, Chair, Faculty Search Committee, Biological Engineering Division Bldg. NE47, Room 223, Massachusetts Institute of Technology, Cambridge, MA 02139; Fax: 617-258-7226

Responses by 1 November 2006 will be given priority.

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Small Molecules Fight Treatment-Resistant Tumors

Using a newly developed drug screen, researchers at the University of Pennsylvania School of Medicine have discovered several small molecule compounds that are able to restore function to cells with defective tumor suppressor p53. By combining molecular imaging techniques with human cancer cell culture and animal model approaches, the researchers were able to reveal the ability of the compounds to kill human tumor cells.

The tumor suppressor p53 is widely mutated across all types of cancer. In addition to causing aggressive tumor growth, a mutation in the p53 gene contributes to chemotherapy- and radiotherapy-resistance. In search of methods to combat treatment-resistant tumors, Wafik S. El-Deiry, Professor in the Departments of Medicine, Genetics, and Pharmacology, and colleagues employed molecular imaging techniques to evaluate the ability of small molecules to produce normal p53 function in p53-deficient and p53-mutant cancer cells. They report their

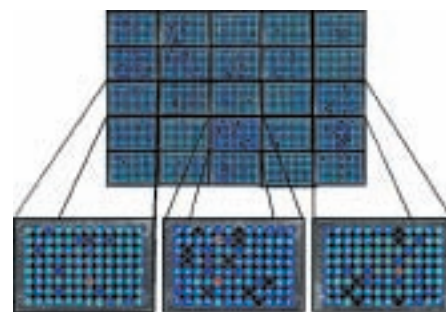
findings in July 18 issue of the *Proceedings of the National Academy of Sciences*.

In an attempt to defend the body, a normal p53 protein will bind to DNA during periods of cellular stress or damage. This binding initiates downstream reactions that keep the stressed cells from multiplying. Under normal conditions, p53 will activate the p21 gene, causing the cell cycle to freeze and thereby halting cell proliferation. p53 will also activate KILLER/DR5, which causes apoptosis. Chemotherapy and radiotherapy set out to deliberately stress tumor cells in hopes of promoting their self-destruction via this pathway. Unfortunately, mutations to the p53 gene disrupt the intracellular defense system.

“Mutants of p53 that occur in human cancer fail to bind to DNA or to activate target genes, such as p21 and KILLER/DR5,” explains El-Deiry. “Therefore, when cells are stressed or damaged, p53-mutant cells fail to shutdown and continue to divide uncontrollably.”

institutes, including the Comprehensive Cancer Center, Graduate Group in Biochemistry and Molecular Biophysics, Graduate Group in Cell and Molecular Biology, and Graduate Group in Pharmacological Sciences.

El-Deiry is among the top 40 most-cited researchers of the 1990s. His research has been awarded many honors, including the Elizabeth and John Cox Award for Molecular Advances in GI Diseases and Cancer and the ISI Highly Cited Researcher in the category of Molecular Biology and Genetics, both in 2005. He currently studies tumor suppressor genes, especially p53, and the contribution of its downstream target genes to cellular growth control. Recently he has been developing and applying non-invasive in vivo imaging technologies for cancer research.



Many classes of compounds were screened for activity in p53 mutant cancer cells. Image Credit: Wafik El-Deiry, M.D., Ph.D., University of Pennsylvania School of Medicine; *Proceedings of the National Academy of Sciences*.

The development of a drug screen by El-Deiry's lab allowed the researchers to trace the activity of small molecules in p53-mutant cancer cells. The small molecule drug screen was created by inserting firefly luciferase into human tumor cells carrying the p53 mutation, and observing the subsequent response. “The cancer cells were engineered to emit light if a p53-like response was triggered by any of the small molecules that we examined,” explains El-Deiry.

The researchers were able to isolate a number of small molecules that activate p53 reporter activity, increase expression of p53 target genes such as p21 or KILLER/DR5, and induce apoptosis in p53-deficient cells. Some of the compounds activated a p53 response by increasing expression of the p53 homolog p73, while others induced a high p53-responsive transcriptional activity in the absence of p53. Further testing exposed the ability of high doses of several groups of the small molecules to kill human cancer cells in cell culture and in mouse models implanted with human tumors.

“Our work provides a blueprint for how molecularly targeted therapy can be discovered using new optical imaging technology,” states El-Deiry. “This is a very important advance in the era of molecular medicine and individualized therapy for cancer patients.”

ASBMB member Wafik S. El-Deiry is Professor of Medicine, Genetics, and Pharmacology at the University of Pennsylvania, Adjunct Professor at the Wistar Institute, and Co-Program Leader of the Radiation Biology Program at the Abramson Cancer Center.

He received his B.S. in Chemistry in 1981 and both his M.D. and Ph.D. in Biochemistry in 1987 from the University of Miami. He



Dr. Wafik S. El-Deiry

became Director of the Laboratory of Molecular Oncology and Cell Cycle Regulation in 1994 and Director of the Bioluminescence Molecular Imaging Core facility in 2002. El-Deiry also serves as a member of several research

Mutation Causes Pancreatic Islet Cells to Reproduce

Researchers at the University of Pennsylvania School of Medicine have found that the acute loss of a protein called menin can cause the proliferation of pancreatic islet cells, which secrete insulin and thereby regulate blood sugar. The menin gene (Men1) mutation in humans causes an inherited disease called Multiple Endocrine Neoplasia type 1 (MEN1). Not only could this discovery inform basic cancer biology, it also has implications for treating Type 1 diabetes. The researchers report their findings in the June 1 issue of *Cancer Research*.

MEN1 patients develop mostly benign tumors or hyperplasia in several endocrine organs, such as parathyroids and pancreatic islet cells. Normally, the menin protein has a tumor-suppressing or cell-proliferation-suppressing function. Loss of menin can cause proliferation of pancreatic islet cells, but not the adjacent exocrine cells that secrete proteins other than insulin.

The researchers developed an animal model that allowed for precise timing in removing the Men1 gene from the genome of knock-out mice. They showed that within seven days of excising Men1, pancreatic islet cells proliferated in the mice. Previously, other labs could only see proliferating islet cells after months of Men1 excision because they could not precisely time the

process. “Our results show an acute effect of Men1 excision and directly link Men1 to repression of pancreatic islet cell proliferation,” says senior author Xianxin Hua, Assistant Professor of Cancer Biology at Penn’s Abramson Family Cancer Research Institute.

The researchers excised Men1 from both islet cells and adjacent exocrine cells in the pancreas, but only in islet cells did they observe cells proliferating. This is important because Men1 mutations largely cause endocrine hyperplasia or tumors, but not exocrine tumors. “Our results showing preferential effects on islet-cell proliferation could at least in part explain that the loss of menin only leads to endocrine tumors,” explains Hua.

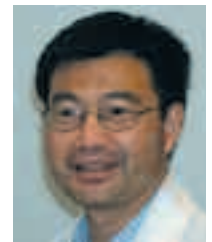
In type I diabetes, the loss of islet beta cells is the leading reason why a sufficient amount of insulin cannot be produced. “If we could eventually repress menin function to specifically stimulate beta-cell proliferation, this may facilitate devising new strategies to increase insulin-secreting beta cells and treating diabetes,” notes Hua.

“We did not expect the connection between a study about a tumor suppressor and a potential new avenue for treating diabetes,” he adds. “By taking advantage of studying a genetically well-characterized tumor syndrome, MEN1, we set out to understand how the first step of benign tumor development is precisely controlled. The more we dis-

covered about menin function, the better we understood the precise role of menin in regulating islet cell proliferation. This latest finding about the acute and specific role of menin on repressing islet cells, but not adjacent exocrine cells, led to the realization that manipulating the menin pathway might be a powerful way to stimulate islet cell proliferation to fight type I diabetes, although we are just beginning toward that goal.”

ASBMB member Xianxin Hua is Assistant Professor in the Department of Cancer Biology at the University of Pennsylvania School of Medicine and Assistant Investigator in the Abramson Family Cancer Research Institute at the University of Pennsylvania.

He received his M.D. and M.S. in Medical Sciences at the Hubei Medical College in China in 1983

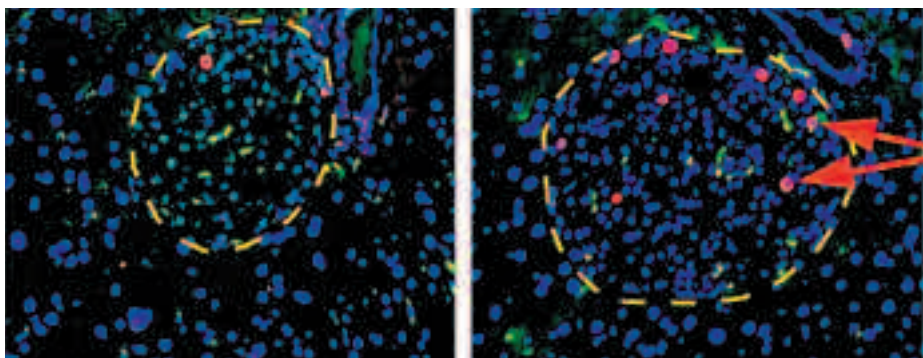


Dr. Xianxin Hua

and 1986, respectively. He then was a fellow at the Akita University School of Medicine in Japan, before he enrolled in the University of Texas Southwestern Medical Center at Dallas where he earned a Ph.D. in Cell Regulation in 1995. In 1996, he became a postdoctoral fellow and clinical scientist at the Whitehead Institute at the Massachusetts Institute of Technology. He joined the University of Pennsylvania in 2000.

Among his many honors, Hua has received a Howard Temin Award from NIH, a Burroughs Wellcome Career Award in Biomedical Sciences, and a Rita Allen Scholar Award. His research focuses on elucidating how tumor suppressor Menin regulates proliferation of insulin-secreting beta cells, hematopoietic cells, and leukemia cells.

Comparison of islet cell proliferation in pancreatic islets with (left panel) and without (right panel) menin.



An Antidote to Those Stifling Textbooks: How to Help Support Science in Your Local Schools

by Bruce Alberts, immediate Past President of the National Academy of Sciences

Now, more than ever, we need to excite all young people about science and to encourage the flow of talented students, including girls and minorities, into scientific and engineering careers.


To help meet this need, the National Academies Press has just launched a new “Women’s Adventures in Science” paperback series and the accompanying iwasondering.org Web site. Written in narrative style and heavily illustrated, the 10 books tell the life stories of 10 differ-

ent contemporary woman scientists. Aimed at the middle school years, each book begins with the scientist’s childhood and continues through her schooling and scientific career. Young people are encouraged to envision themselves in the role of a scientist, as they imagine what it would be like to build the first robot that could interact with people — or to study human remains in search of criminal evidence.

Immensely successful in inspiring both boys and girls, one of these books



recently won the National Science Teachers Association/Children’s Book Council Outstanding Science Trade Book award prize from the National Science Teachers Association.

Scientists have been donating the books to the science teachers in their local middle schools, and giving them as presents to their young relatives. Individual books can be purchased from www.nap.edu/catalog/was for \$9.95 each; entire sets are \$89.50 plus shipping. Excerpts of each book can also be read at this website. 

Passion. Drive. Persistence.

- The Story of Planetary Astronomer Heidi Hammel**
By Fred Bert
Heidi was on the team that first spotted Neptune's Great Dark Spot, a raging storm as big as Earth.
- The Story of Biochemist Maria Thérèse**
By Steve O'Connell
Maria, the daughter of an illegal American immigrant, uses her expertise to help government leaders create equal opportunities for millions of people.
- The Story of Climate Scientist Inez Fung**
By Bruce Alberts
Inez foresees the future—a future when Earth could look and feel a lot different than it does now.
- The Story of Robot Designer Cynthia Brezina**
By Jordan R. Brown
Cynthia uses electronic gadgets, software programs, and her endless imagination to create lifelike machines that can respond to the world around them.
- The Story of Neuropsychologist Nancy Wexler**
By John Glaser
Nancy hunts for the gene responsible for a fatal, inherited disease so that a prevention or cure might one day be found.
- The Story of Planetary Geologist Adriana Ocango**
By Corinne Jean Mopping
Adriana helped find the Crater of Doom, caused by a "killer" space rock hitting the Earth 66 million years ago, when the dinosaurs died out.
- The Story of Wildlife Biologist Amy Vetter**
By Peter Chernin
Amy saves animals whose lives and habitats are threatened. She helped rescue Florida's mountain gorillas from extinction.
- The Story of Physicist Shirley Ann Jackson**
By Diane O'Connell
Shirley Ann uses her knowledge of the subatomic world to better the lives of others—for instance, by making nuclear power plants safer.

McGill Professor Told No Intelligent Design, No \$\$

The last thing McGill University Professor Brian Alters expected upon opening a letter last Spring was to see his latest \$40,000 Canadian (\$36,400 U.S.) grant rejected for not providing enough evidence to support a theory he'd made a career of defending: evolution.

Alters had applied for funds from Canada's Social Sciences and Humanities Research Council (SSHRC) to study the effect of intelligent design debates in the United States on Canadian students, teachers, administrators, and policymakers. In the rejection letter, the SSHRC said Alters, who is a vocal advocate for education about evolution and an expert witness in the recent Dover trial, did not provide "adequate justification for the assumption in the proposal that the theory of evolution, and not intelligent design theory, was correct."

Alters said he was completely blown away to read that one of Canada's largest funding bodies seemed to consider intelligent design an alternative scientific theory to evolution. Coincidentally, he received the letter a few days before giving a Canadian Royal Society lecture on "Intelligent Design, God, and Evolution." Alters read the six-sentence rejection aloud to the 650 people attending, and "there was an audible gasp in the audience," he says.

SSHRC called the letter an unfortunate "miswording," but one with consequences that haven't yet gone away, the agency admits. "To my knowledge, a controversy being generated by the wording of a rejection letter hasn't

happened to us before," says Eva Schacerl, a spokesperson for SSHRC. The agency has received "a lot of emails from scientists," she adds.

Hundreds of letters have also shown up at Alters' door. "People from all over the world have been writing to me," says Alters. Indeed, at a recent talk at the U.S. National Institutes of Health, scheduled well before the controversy, Alters was asked to change his topic from the Pennsylvania Dover case to the SSHRC controversy.

Scientists say the rejection letter aroused such interest because a four-person peer-review committee composed it and the SSHRC reviewed it, yet it appears to doubt the theory of evolution. "When I saw the comments [of the SSHRC rejection letter] it was clear that the evolution community should be concerned," says Douglas Morris, an evolutionary biologist at Lakehead University in Canada. "I'm not disputing the decision on the grant, but [I am disputing] the message that evolution needs to be justified on an equal footing with intelligent design."

Morris wrote a letter to SSHRC asking that it provide detailed reasons for rejecting the grant and clarify its position on intelligent design. The Canadian Society for Ecology and Evolution, the American Sociological Association, and the American Institute for Biological Sciences have all requested the same thing in open letters to SSHRC. In response, SSHRC has released a statement on its website saying "the theory of evolution is

not in doubt" and that it "regrets that the summary of the committee's comments sent to Dr. Alters was poorly formulated."

Morris and Alters wanted SSHRC to take a stand on intelligent design, and they are less than satisfied with SSHRC's silence on the issue. However, "it's not our role as an organization to enter into this debate," says Schacerl, adding that SSHRC is nevertheless looking into its peer-review process because of the controversy.

Meanwhile, Alters, who continues to study the debate over intelligent design, could have a lot more grant writing ahead of him. "I think Alters has enough [material from this experience] to write a few research papers already," says Morris. ☺

Important Deadlines for the Upcoming ASBMB Annual Meeting

❖ **Abstract Submission:**
November 8, 2006

❖ **Early Registration:** March 2, 2007

❖ **Housing:** March 23, 2007

April 28 - May 2, 2007
Washington D.C.,
Convention Center

For more information please
visit www.asbmb.org

The Extracellular Matrix at Multiple Biological Scales

Organizer: Vito Quaranta, Vanderbilt University Medical Center

The Extracellular Matrix (ECM) Theme at ASBMB 2007 in Washington is organized in four sessions, divided by biological scales: molecular, cellular, tissue and organism scale. This unusual organization is designed to produce new, integrative views in a field that has enjoyed explosive growth in the past two decades. An enormous amount of data has been produced especially at the molecular and cellular scale, and it must now be integrated with our burgeoning understanding of the ECM roles at tissue and organism scale. ECM functions in organisms are rooted in its molecular structure and, conversely, ECM structure informs functions at higher scales. It is unlikely, save few exceptions, that unveiling the detail of a particular ECM macromolecule will directly provide mechanistic information on its role at the organism level. Rather, intervening biological scales must be understood and bridged in an integrative approach. This is perhaps one of the most important challenges facing the ECM field in the immediate future. The ECM Theme presents an impressive line-up of speakers, who will provide a comprehensive view of the elements of this challenge.

In the Molecular Scale session, Tim Springer of Harvard University will provide an update on the structure of integrins, the premiere cell surface receptors for ECM that provide mechanical links of cells to the ECM, and initiate downstream signaling as well. Billy Hudson, from the Division of Nephrology at Vanderbilt University

Medical Center, will describe the structure of Collagen IV, an ancient ECM macromolecule whose structure is critical for assembly of basement membranes and has been elegantly linked to human disease. Vito Quaranta will report on the molecular basis of integrin binding to laminins, which surprisingly is still undefined and may hold some surprises, since it is established that laminin-binding integrins do not bind to the classic RGD peptide motif that explains binding by most other integrins.

The Cellular Scale session will provide examples of the latest insight into mechanistic interactions between cells and ECM. Viola Vogel, from ETH in Zurich, has produced quantitative perspectives on mechanotransduction. This is a hot subject at the moment, but experimental approaches are still being developed and data are scarce. Alissa Weaver, of Vanderbilt University, will report on cellular organelles, named podosomes or invadopodia, recognized for decades, but only recently receiving increased attention as the main organelle that cells use to remodel ECM. Peter Yurchenco, R.W. Johnson University in New Jersey, has for years been studying the mechanism of assembly of laminins, and has recently identified some critical steps that are carried out by cells at their surfaces.

The Tissue Scale session is dedicated to the role the ECM plays in the organization of cells into tissue. Elaine Fuchs of Rockefeller University is a world-class epithelial cell biologist who has made key discoveries on epidermal morpho-



Dr. Vito Quaranta

genesis. Her talk will focus on the interplays that occur in epithelial stem cell niches. Jeff Miner, from Washington University in Saint Louis, has made great strides in characterizing the role of laminins in the organization of kidney and intestinal tissue. He will report the latest findings from laminin knock-out mice. Raghu Kalluri, Harvard University, has a distinguished record in the characterization of ECM fragments that regulate angiogenesis, and will report on the mechanistic effects of collagen fragments in physiological and pathological processes such as cancer progression.

In the Organism Scale session, the effects of deleting ECM macromolecules by genetic means will be discussed. Uli Mueller, The Scripps Research Institute, has conducted systematic analyses by gene knock-out in mice, in order to define the role of several ECM molecules and their receptors in the development of the nervous system. Mary Zutter, Vanderbilt University, has studied collagen-binding integrins for many years, and will speak about cancer progression and immune system defects in mice that lack the gene for a specific collagen receptor. Nick Brown, Cambridge Uni-

Continued on next page

Theme Meeting to Focus on Wide Scope of RNA Function and Regulation in Biology

Organizer: Krysten Lynch

The past few years have brought a resurgence in RNA research and an increased appreciation for the many critical and complex roles that RNA plays within a cell. The discovery that mammals have many fewer genes than anticipated has emphasized the importance of RNA processing and regulation as a means of generating genetic diversity and controlling protein expression; the discovery of RNAi has led to the uncovering of a plethora of small, non-coding RNAs that are involved in almost all aspects of cellular function; and our appreciation of how RNAs can control their own expression has been deepened by the characterization of riboswitches and ribozymes.

The RNA Theme at the 2007 ASBMB meeting in Washington, DC will bring together many internation-

ality, is investigating ECM in *Drosophila*, and has made some crucial discoveries as to how integrin-ECM mechanotransduction can direct and maintain wing assembly.

Together, these four sessions will provide an integrated overview of the state-of-the-art in the ECM field. Such multiscale perspective will be of interest to the specialist, since it is rarely offered in ECM meetings, as well as to all meeting participants, since multiple scale concepts apply to all branches of biochemistry and molecular biology. Graduate students and post-doctoral fellows are particularly encouraged to attend, since they will carry out these new concepts in the next generations. See you in Washington! 🍷

ally recognized scientists who are engaged at the forefront of RNA research. Specifically, the sessions will focus on molecular recognition and enzymology of RNA, RNA-based gene regulation, small RNAs and RNA modification. However, an important and recurrent discussion point will be how these sub-topics naturally converge with one another, as well as with other aspects of Biochemistry, such as Protein Translation (which will be represented by a separate, but coordinated Theme at the ASBMB meeting). Together, these sessions will emphasize the breadth and importance of RNA in Biology, and will highlight recent advances in our understanding of RNA function.

The session entitled Molecular Recognition and Enzymology of RNA will focus on structural insights into the activities that RNA can adopt. Dr. Anna Pyle (Yale University) will chair the session and will discuss her work on RNA helicases and conformational changes within Group II self-splicing ribozymes. Additional insight into how RNA structure impacts enzymatic function will come from Dr. Scott Strobel (Yale University). Dr. Robert Batey (University of Colorado-Boulder) will describe how the binding of RNA structures known as "riboswitches" by ligand causes alterations in the expression of downstream genes.

The session entitled RNA-based Gene Regulation will track the matu-

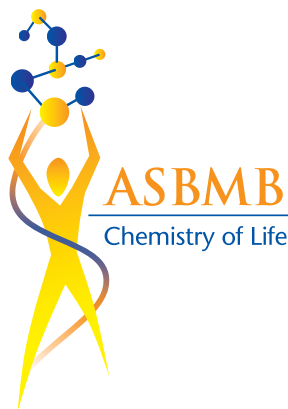


Dr. Krysten Lynch

ration of a nascent pre-mRNA from splicing to localization to translation, to describe recent findings regarding how such processing steps are regulated to control gene expression. This session will be chaired by Dr. Kristen Lynch (UT Southwestern Medical Center) who will discuss mechanisms by which alternative splicing patterns are influenced by extracellular stimuli. Dr. David Spector (Cold Spring Harbor Labs) will describe how gene expression can be controlled by regulation of the nucleo-cytoplasmic localization of mRNA. Dr. Joel Richter (U. Mass Worcester) will discuss mechanisms of translation regulation by the protein CPEB.

Dr. Witold Filipowicz (Friedrich Miescher Institute) will chair the session on Small RNAs and will present his work on regulation and function of microRNAs. Dr. Thomas Tuschl (Rockefeller University) will also describe recent advances from his group regarding small-RNA regulated gene expression. Dr. E. Gerhart Wagner (Uppsala University) will discuss the diverse functions of bacterial sRNAs.

The session on RNA modification will encompass the mechanisms and consequences of editing of mRNAs, as well as other forms of RNA modification. This session will be chaired by Dr. Robert Reenan (Brown University) who will discuss ADAR function. Dr. Kazuko Nishikura (Wistar Institute) will discuss editing of non-coding RNAs, and Dr. Ronald Emeson (Vanderbilt University) will describe the function of editing of the serotonin receptor. 🍷



2007 ASBMB

April 28 – May 2, 2007 • Washington, DC

Organized by: Benjamin F. Cravatt, The Scripps Research Institute, Michael K. Rosen, University of California, San Diego

Abstract Submission Deadlines

Award Lectures



Herbert Tabor/Journal of Biological Chemistry Lectureship:

Anthony Hunter, The Salk Institute:
Tyrosine phosphorylation: from discovery to the kinome and beyond



Anthony Pawson, Samuel Lunenfeld Research Institute:
Phosphotyrosine signaling: A prototype for modular protein-protein interactions



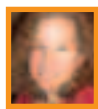
ASBMB Amgen Award

Angelika Amon
HHMI/Massachusetts Institute of Technology



ASBMB Award for Exemplary Contributions to Education

Sarah C. Elgin
Washington University



ASBMB Merck Award

Judith P. Klinman
University of California, Berkeley



Avanti Award in Lipids

Scott D. Emr
HHMI/University of California, San Diego, School of Medicine



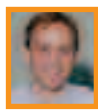
Fritz Lipmann Lectureship

Ronald M. Evans
The Salk Institute



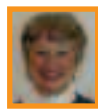
Howard K. Schachman Public Service Award

To Be Determined



Schering-Plough Research Institute Award

Christopher B. Burge
Massachusetts Institute of Technology



William C. Rose Award

Susan S. Taylor
HHMI/University of California, San Diego

Preliminary Program

Genome Dynamics

From Genome to Epigenome – Modification and Repair
Gregory L. Verdine**, Harvard University

Methylating and De-methylating DNA
Timothy Bestor*, Robert Fisher, Tomas Lindahl

Recombining and Modifying DNA
William S. Reznikoff, David G. Schatz*, Gregory Van Duyne

Making and Re-making DNA
Lorena Beese*, Gregory L. Verdine, Graham C. Walker

Telomeres and Telomerase
Elizabeth Blackburn, Kathleen Collins*, Carol Grieder

The Chromosome Cycle
Hongtao Yu**, University of Texas Southwestern Medical Center

Centromeres and Kinetochores
Donald W. Cleveland*, Gary H. Karpen, Huntington F. Willard

Chromatin Structure and Remodeling
Jeffrey J. Hayes, Geeta Narlikar*, Song Tan

Chromosome Duplication and Cohesion
Jan-Michael Peters*, Camilla Sjögren, Johannes Walter

Chromosome Segregation and Aneuploidy
Jan van Deursen, Gary J. Gorbsky, Hongtao Yu*

RNA
Kristen W. Lynch**, University of Texas Southwestern Medical School

Molecular Recognition and Enzymology of RNA
Robert T. Batey, Anna Marie Pyle*, Scott Strobel

RNA-Based Gene Regulation
Kristen W. Lynch*, Joel D. Richter, David Spector

Small RNAs
Witold Filipowicz*, Thomas Tuschl, E. Gerhart H. Wagner

RNA Modification: Mechanism and Function
Ronald Emeson, Kazuko Nishikura, Robert Reenan*

Protein Synthesis, Folding and Turnover
James R. Williamson**, The Scripps Research Institute

Molecular Mechanisms of Protein Biosynthesis
Rachel Green*, Daniel Herschlag, Timothy W. Nilsen

Co- and Post-Translational Folding
Elizabeth Craig, F. Ulrich Hartl, Jonathan S. Weissman*

Protein Modification and Turnover
Christopher P. Hill*, Christopher D. Lima, Tom Rapoport

Ribosome and Translation
Joseph D. Puglisi, James R. Williamson*, Nahum Sonenberg

Special Events

- 11th Annual Undergraduate Student Poster Competition
- ASBMB Business Meeting
- ASBMB Social Event

Structure and Design

Macromolecular Structure and Dynamics
Joseph P. Noel**, The Salk Institute for Biological Studies

Conformational Transitions and Protein Aggregation
Jeffery W. Kelly, Beat Meier, Roland Riek*

Experimental and Computational Dynamics
Lewis E. Kay*, J. Andrew McCammon, Sunney Xie

Protein-Lipid Interface
Susan Buchanan, John Bushweller, Charles R. Sanders*

Structural and Mechanistic Evolution
Antony M. Dean, Joseph P. Noel*, Rama Ranganathan

Enzymes – Mechanism and Design
Dorothee Kern**, Brandeis University

Structural Enzymology
Karen Allen, Perry A. Frey, Gregory A. Petsko*, Dagmar Ringe

The Role of Dynamics in Enzyme Catalysis
Gordon G. Hammes, Dorothee Kern, Judith P. Klinman, Peter E. Wright*

Computational Studies of Mechanistic and Dynamical Aspects of Enzymes Reactions

Jiali Gao, Sharon Hammes-Schiffer*, Kenneth M. Merz, Jr.

Enzyme Design
Homme W. Hellinga, Kenneth N. Houk, Stephen L. Mayo*

Extracellular Matrix at Multiple Biological Scales
Vito Quaranta**, Vanderbilt University

Extracellular Matrix at the Cellular Scale
Viola Vogel*, Alissa Weaver, Peter D. Yurchenco

Extracellular Matrix at the Molecular Scale
Billy G. Hudson, Vito Quaranta*, Timothy A. Springer

Extracellular Matrix at the Organism Scale
Nick Brown, Uli Mueller*, Mary Zutter

Extracellular Matrix at the Tissue Scale
Elaine Fuchs*, Raghu Kalluri, Jeffrey H. Miner

Chemical Biology
Jack Taunton**, University of California, San Francisco

Chemical Biology of Cell Death
Craig Crews, Paul Hergenrother*, Junying Yuan

Fragment Based Drug Discovery
Jon Ellman, Daniel A. Erlanson*, Harren Jhoti

Chemistry and Cell Biology of Natural Products
Jun Liu, Jack Taunton*, Christopher Walsh

Antibiotics for the 21st Century
Heike Brotz-Oesterhelt, Deborah T. Hung, Floyd Romesberg*

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Annual Meeting

DC • Held in conjunction with EB 2007

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Online: November 8, 2006

Cell Systems

Metabolism

Jared Rutter**, University of Utah School of Medicine

Metabolic Sensing and Signaling

David Carling, Michael Hall, Jared Rutter*

Molecular and Cellular Aspects of Metabolic Disease

Morris Birnbaum, Marc Montminy*, Craig B. Thompson

Mitochondria in Health and Disease

E. Dale Abel*, Nika N. Daniai, Antonio Vidal-Puig

Aging and Metabolism

Andrew Dillin, Stephen L. Helfand, Pere Puigserver*, Richard Weindruch

Organelle Dynamics

Matthew Shair**, Harvard University

Golgi Structure and Biogenesis

Matthew Shair*, Jennifer Lippincott-Schwartz, Graham Warren

Membrane Biogenesis

Daniel E. Kahne*, Natividad Ruiz, Hajime Tokuda

Mitochondrial Dynamic

David Chan, Jodi Nunnari*, Richard Youle

Nuclear Dynamics

Ueli Aebi, Katherine S. Ullman, Yixian Zheng*

Systems Biology

Tobias Meyer**, Stanford University School of Medicine

Modeling of Cell Systems

James Ferrell*, Rustem Ismagilov, Wendell Lim

Molecular Profiling of Cell Systems

Tobias Meyer*, Elizabeth Winzeler

Proteomics of Cell Systems

Reudi H. Aebersold*, Anne-Claude Gavin, Michael Snyder

Mathematical Biology

Mark Chaplain, Ravi Iyengar, Edwin Munro, Vito Quaranta*

Minority Affairs Committee Sponsored Symposia

George Hill**, Vanderbilt University

Best Practices in Program Assessment

Taketa Felder*, A. James Hicks, John Matsui, J. Lynn Zimmerman

Infectious Diseases in Minority Populations – Hepatitis C

Craig E. Cameron*, Antonio Estrada, Kouacou Donan, Gerond Lake-Bakaar

Genetic Diseases in Minority Populations - Sickle Cell Anemia

Jane Hankins, Phillip A. Ortiz*, William P. Winter, Steven N. Wolff

Infectious Diseases in Minority Populations - Tuberculosis

Bavesh Kana, Ujjini Manjunatha, Marcos Milla*, Harvey Rubin

- Minority Scientists' Mixer
- Opening Reception
- Research Funding by the American Cancer Society

**denotes thematic meeting chair / *denotes session chair

Signaling

Biochemistry and Signaling of Lipids

Hugh Rosen**, The Scripps Research Institute

Biogenesis, Transport and Compartmentalization of Lipids

Christoph Benning, Joost C.M. Holthuis, Dennis R. Voelker*

Chemical Probes of Lipid Systems

Doreen Cantrell, Benjamin F. Cravatt, Hugh Rosen*

Lipids as Transcriptional Regulators

Joseph L. Goldstein, Steven Kliewer*, Peter Tontonoz

Specific Protein-Lipid Interactions

Michael H. Gelb*, Tamir Gonen, Stephen White

Signaling Pathways Controlling Cell Structure and Fate

Michael B. Yaffe**, Massachusetts Institute of Technology

Cytokine and Growth Factor Signaling

Carl-Henrik Heldin, Mark Lemmon, Joseph Schlessinger*

DNA Damage Signaling

Wade Harper, Michele Pagano, Michael B. Yaffe*

Cell Cycle

Susan Biggins, Rebecca Heald*, Tim Stearns

Signaling to the Cytoskeleton

Sandrine Etienne-Manneville, Dyche Mullins*, Michael K. Rosen

Public Affairs Advisory Committee Sponsored Symposium

Sponsored by EB participating societies
NIH at the Crossroads: How Diminished Funds Will Impact
Biomedical Research and what Scientists Can Do About it

Education and Professional Development Committee Sponsored Symposia

J. Ellis Bell**, University of Richmond

Classroom of the Future II

J. Ellis Bell*, Catherine L. Drennan, Carla Mattos

Science at Undergraduate Institutions

Teaster Baird, Lisa Gentile, Joseph J. Provost*, Mark A. Wallert*

Graduate Student/Postdoctoral Starting Faculty Transitions

Jessica Bell, Parag Chitnis*, Ann L. Miller

Preparing for a Successful Career in Industry

Gregory Bertenshaw*, Robert A. Copeland*, Manuek Navia

Travel Awards

Application Deadline: November 30, 2006

- Graduate Students / Postdoctoral Fellows
- Minority Graduate Students
- Systems Biology Workshop Teams

- Women Scientists' Networking Reception
- Meet the Speakers
- Thematic Receptions

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Angelika Amon Selected for

Angelika Amon, of the Department of Biology and Center for Cancer Research at the Massachusetts Institute of Technology, has been selected to receive the ASBMB-Amgen Amgen Award. This award is made to a new investigator (defined as an individual with no more than 15 years experience since receipt of a doctorate) for significant achievements in the application of biochemistry and molecular biology to the understanding of disease. Nominations must be originated by Society members, but the nominees need not be ASBMB members. The Award consists of a silver and crystal commemorative sculpture, an honorarium to the recipient, an unrestricted research grant, and transportation and expenses to present a lecture at the 2007 ASBMB Annual Meeting, April 28 – May 2 in Washington, DC. Past recipients of this award include: in 2006 Ali Shilatifard, in 2005 Barry Forman, in 2004 Steven C. Almo, in 2003 - Wesley Sundquist, and in 2002 - Joseph Heitman.

Dr. Amon has made unparalleled contributions to our understanding of the cell cycle in particular to the metaphase/anaphase and M/G1 transitions. These include four seminal observations: The elucidation of the regulatory circuitry that controls the transcription of M and G1 cyclins; the discovery that the cyclin degradation machinery is active from M to G1, and that its inactivation in G1 is critical to the G1/S transition; the identification of the specificity factors for the mitotic degradation machinery that regulate the metaphase/anaphase transition and exit from mitosis; the discovery that the exit from mitosis is controlled by subnuclear sequestration and subsequent release of cell cycle regulators.



Dr. Angelika Amon

In the last six years, Amon has discovered two regulatory pathways that control exit from mitosis. She has also become one of the world leaders in studying the regulation of meiotic progression, and her lab has just discovered a novel regulatory step in meiosis I in which recombination during prophase is a prerequisite for the dissolution of cohesion along chromosome arms during anaphase I.

The duplications of cells, the building blocks of all organisms, requires the duplication of the genetic material followed by its segregation to the future daughter cells. For the building of an organism, it is essential that this cycle of events, the mitotic cell division cycle, occurs in a precise and orderly manner. Deciphering the regulatory networks that ensure accurate segregation of the genetic material is thus vital to understanding both normal cell division and abnormal cell division that leads to cancer and birth defects. Owing to the high degree of conservation of this process among eukaryotes, she has used the budding yeast *Saccharomyces cerevisiae* as a model system to uncover the regulatory networks that govern the segregation of the genetic material.

A key transition in the segregation of the genetic material (chromosomes) to

the two daughter cells is exit from mitosis. During this transition, cells complete the chromosome segregation phase and get ready for the next duplication phase. The Amon lab has shown that the protein phosphatase Cdc14 is a key trigger of this transition and that its activation during chromosome segregation is essential for exit from mitosis to occur.

Due to the central importance of Cdc14 in exit from mitosis, Amon next focused on determining how the phosphatase is regulated. Cdc14 is regulated by an inhibitor Cfi1/Net1 that binds to and sequesters Cdc14 in the nucleolus during G₁, S phase, G₂ and metaphase. During anaphase, Cdc14 is released from its inhibitor and spreads throughout the nucleus and cytoplasm, where it dephosphorylates its targets. Subsequently, the Amon lab identified two regulatory networks that control the association of Cdc14 with Cfi1/Net1. The Cdc14 early anaphase release network (FEAR network) promotes Cdc14 release from the nucleolus during early stages of chromosome segregation, whereas the Mitotic Exit Network (MEN) maintains Cdc14 in a released state during late stages of the chromosome segregation phase. Finally, Dr. Amon uncovered functions for the two pathways that regulate Cdc14 function. The FEAR network couples the onset of chromosome segregation with Cdc14 activation and thus, exit from mitosis. The MEN senses nuclear position and ensures that cells only exit from mitosis when the genetic material has been partitioned between the two future daughter cells.

In addition to studying how Cdc14 is regulated, the Amon lab investigated the function of the phosphatase during mitosis. Cdc14's main role is to


ASBMB-AMGEN Award

reverse mitotic phosphorylation events, thereby triggering exit from mitosis. However the phosphatase has other functions. The lab found that Cdc14 regulates the segregation of repetitive DNA, which segregate late during mitosis and determined the mechanism whereby Cdc14 accomplishes this task. These studies also revealed a solution for a long-standing question: How cells ensure that chromosome separation is completed before cells exit from mitosis. The fact that Cdc14 promotes the partitioning of late-segregating DNA, regions as well as exit from mitosis, provides a mechanism for ensuring that the two events are coupled.

The meiotic cell division cycle is a specialized cell division cycle that leads

to the formation of gametes. Defects in meiotic chromosome segregation are the leading cause of miscarriages and one of the leading causes of birth defects in humans. Understanding how meiotic chromosome segregation is regulated and how the mitotic chromosome segregation cycle is modulated to bring about the specialized meiotic chromosome segregation program is crucial to uncovering the molecular causes of chromosome mis-segregation during meiosis.

Dr. Amon has made significant contributions towards determining how regulators of mitotic chromosome segregation are employed to bring about the specialized meiotic chromosome segregation program and discovered meiosis-specific proteins that bring

about these meiotic functions. First, the lab identified factors that are involved in regulating cohesins, which hold sister chromatids together. Using a functional genomic approach they identified three genes, IML3, CHL4 and SGO1 as being required for regulating cohesin maintenance on chromosomes, as well as characterizing their function in the process. Furthermore, their studies showed that a highly conserved Polo kinase is a critical coordinator of the meiotic chromosome segregation program. In addition they have shown that the FEAR network, which plays a critical role in the regulation of mitotic chromosome segregation, is essential for establishing the order of events of meiotic chromosome segregation. 

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James Rothman (Columbia University, NY)

Speakers

Philippe Bastien (EMBL, Germany)
Valerie Culotta (Johns Hopkins, MD)
Tarun Kapoor (Rockefeller University, NY)
Akihiro Kusumi (Kyoto University, Japan)
Jeremy Nicholson (Imperial College London, UK)
Joseph P. Noel (The Salk Institute, CA)
Garry Nolan (Stanford University, CA)
Richard Proia (National Institutes of Health, MD)
Peter Sorger (MIT, MA)
Kai Simons (Max Planck Institute, Germany)
Jack Taunton (UCSF, CA)
Antoine van Oijen (Harvard University, MA)

nature publishing group 

Stem-cell Method Preserves Embryo; Massachusetts Lab Hopes to end Standoff

On August 23, Massachusetts scientists announced that they had created the first human embryonic stem cells using a technique that does not require the destruction of an embryo, an advance that they said could end the political standoff over stem-cell research.

According to the *Boston Globe*, the research by the Worcester laboratories of Advanced Cell Technology (ACT), showed that a single cell from an early embryo can be used to generate embryonic stem cells

“My hope is that this will jump-start this field,” said Dr. Robert Lanza, a scientist at Advanced Cell Technology who oversaw the research. “We really need to get past all of this politics.”

Reacting to the announcement, a White House spokesman called the research a positive step but said the President’s position has not changed. “This technique does not resolve” serious ethical concerns about the use of embryos in research, said spokesman Peter Watkins, “but it is encouraging to see scientists at least make serious efforts to move away from research that involves the destruction of embryos.”

The new technique does not address all of the ethical concerns raised by critics of embryonic stem-cell research.

Scientists called the work an important advance, but said other researchers must verify the findings. Scientists say human embryonic stem cells, which have the capacity to become any cell in the body, are a powerful tool for research that could lead to new insights into diseases and, potentially, cures.

The President’s ban on using federal funds to study stem cells created after August 2001, has drawn complaints from scientists that few batches of the older stem cells have proved useful. At

the same time, scientists around the world have been looking for a way to make cells that behave like embryonic stem cells, but do not require the destruction of an embryo. Earlier this year, German scientists reported they could modify cells found in the testes of mice and create cells that behave like embryonic stem cells, and Japanese scientists recently reported that they could make human skin cells take on some of the characteristics of embryonic stem cells.

The ACT technique, however, is the first to yield human embryonic stem cells, and would not require using more than one of the embryo’s cells, leaving a viable embryo capable of starting a pregnancy.

The technique builds on a procedure known as preimplantation genetic diagnosis (PGD). With it, after technicians fertilize an egg, they allow it to grow into an embryo of approximately eight cells. They then remove one cell and test it for signs that the embryo carries chromosomal abnormalities or genes for diseases such as cystic fibrosis. The results guide decisions about which embryos to implant in a woman.

To create stem cells, the ACT team proposes working in the future with couples already having PGD performed. The key difference is that after the cell is removed, it would not be immediately tested, Lanza said. Instead, it would be allowed to grow in a laboratory dish overnight, in the hope that it would divide, creating two cells. One of the cells would be used for the genetic testing and the other to create a batch of embryonic stem cells.

In the company’s testing, a single cell divided overnight 58% of the time, according to the paper. All told, the company used 91 single cells taken

from 16 embryos, and from those it was able to produce only two batches of human embryonic stem cells. The team performed a standard set of tests to demonstrate they were embryonic stem cells. The scientists used frozen embryos that a fertility clinic planned to discard, Lanza said.

Before the technique could be used as a part of PGD, doctors would have to be confident that it would not interfere with the genetic testing, according to Richard Scott, Director of Reproductive Medicine Associates of New Jersey, in Morristown.

Lanza said his team is working to resolve these issues with an American fertility clinic. The clinic, which he declined to name, contacted him after he published a paper last year that showed the technique worked in mice.

One potential ethical complication is the question of whether a single cell from an embryo could develop into a human life. In the paper, the authors state that single cells taken from the stage of embryo used “have never been shown to have the intrinsic capacity to generate a complete organism in any mammalian species.” However, critics have said this does not entirely prove the case. Last year, the President’s Council on Bioethics issued a report on alternative methods of creating embryonic stem cells, which criticized the idea used by ACT as raising ethical questions, including whether one cell could become a human.

In addition, a provision of federal law known as the Dickey-Wicker amendment may prohibit funding the creation of stem cells using the ACT technique, because the law prohibits funding certain kinds of embryo manipulation, according to Alta Charo, a prominent bioethicist and professor at the University of Wisconsin at Madison ☞

How Retired Scientists Are Helping Educate Our Youth

For many scientists, the joy of retirement can be diminished by concerns about the loss of activity, the thrill of discovery, and opportunities for contact with other scientists. However, since its foundation in 1991, the RE-SEED (Retirees Enhancing Science Education through Experiments and Demonstrations) program has provided professionals with scientific backgrounds the opportunity, as volunteers in middle schools, to help teach students about the physical sciences.

Created by Professors Alan Cromer, Christos Zahopoulos and Michael Silevitch, along with retired engineer executive Frank Madden, and based at Northeastern University in Boston, RE-SEED has enrolled nearly 500 volunteers, including scientists, engineers and physicians. It has been active in about 100 school districts in some 10 states, and is currently widespread throughout Massachusetts and Maine. In total these volunteers have offered about 500,000 hours of service, and have become deeply involved members of the schools in which they work.

The training for the volunteers is thorough, and mandates completion of 13 4-hour workshop sessions. Volunteers visit a school on average once a week, and use hands-on activities to help demonstrate the concepts of physical science, using the SEED Sourcebook for material. Supported by a grant from the National Science Foundation, the Sourcebook was created in 1993 for volunteer training by Doctors Cromer and Zahopoulos and has further developed since then. Over 200 inexpensive activities for 6th to 8th grade students are listed for topics such as motion, electromagnetism, and Earth as a planet.

In addition to increasing student interest in science, the expertise of the volunteers is useful to enrich teachers' knowledge as well. It is estimated that only 17% of middle school science teachers hold a science degree, and in low-income schools students have less than a 50% chance of having a math or science teacher with a license or degree in their professional fields. In such settings, RE-SEED volunteers can use their experience and knowledge to bring the excitement and thrill of science into the classroom, and also be available to act as mentors if needed.

The next scheduled training programs will take place in Santa Clara County, California, on October 2-5, and in Boston, Massachusetts, on Mondays from 10-2 starting in October. If you are interested in receiving more information about RE-SEED and its training programs, visit their website, www.reseed.neu.edu, call 617-373-5860 or 888-742-2424, or email Deirdre Weedon at D.Weedon@neu.edu. ❧

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Scientists Coax Nerve Fibers to Regrow After Spinal Cord Injury

In tests on rats, researchers at Johns Hopkins and the University of Michigan have developed a treatment that helps spinal cord nerves regrow after injury. The findings were published in the July 18 issue of the *Proceedings of the National Academy of Sciences*.

The researchers treated experimental nerve injuries in rats with the enzyme sialidase. Four weeks later, more than twice as many nerves in the spinal cords of sialidase-treated rats grew new nerve fibers compared to nerves in untreated rats. The experimental injury in rats mimicked an injury in humans that may occur during childbirth or in motorcycle accidents when an arm is pulled violently away from the body. This injury causes nerves to be yanked out of the spinal cord. Without these nerves, the arm loses feeling and muscle tone. Without muscle tone, the body cannot support the weight of the arm, and many health problems can develop.

While surgeons can sometimes reattach the yanked nerves to the spinal cord, this treatment is not as effective as physicians or patients would like. This is, in part, because nerves in the brain and spinal cord, unlike those in the rest of the body, fail to grow new nerve fibers.

"Molecules in the environment of the injured spinal cord are specifically instructing the nerve end not to regrow," says the study's director, Ronald Schnaar, Ph.D., professor of pharmacology and neuroscience in the Institute of Basic Biomedical Sciences at Hopkins.

"The brain and spinal cord are extremely crowded with nerves and nerve fibers, which may be why we have developed careful controls that tell cells to stop making new connections. The crowded central nervous system has ways to say 'OK, we're done' to keep nerves from sprouting

ASBMB member Ronald L. Schnaar is a Professor in the Department of Pharmacology and Molecular Sciences and in the Department of Neuroscience at the Johns Hopkins University School of Medicine. He is also Director of the Pharmacology Graduate Program. He obtained his B.S. in Cellular Biology from the University of Michigan in 1972 and his Ph.D. in Biology from the Johns Hopkins University in 1976. He was a post-doctoral fellow at the Johns Hopkins University in 1977 and at the National Institutes of Health from 1978 to 1979. In 1980, Schnaar began as Assistant Professor in the Department of Pharmacology and Molecular Sciences and Department



Dr. Ronald L. Schnaar

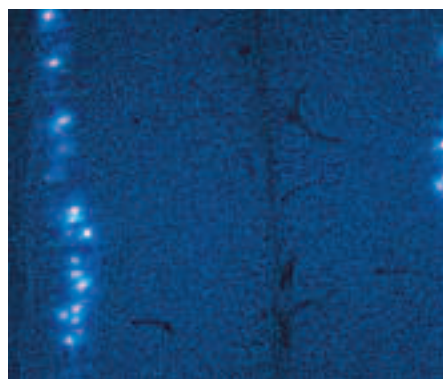
of Neuroscience at Johns Hopkins, and from 1984 through 1990 he held the position of Associate Professor.

Schnaar has served on many advisory panels and has authored or co-authored over 120 publications. In addition to earning research awards from the American Cancer Society and a graduate student teaching award from The Johns

Hopkins University in 1988, he is also a recipient of the National Institute of Neurological Disorders and Stroke Javits Neuroscience Investigator Award from 2005-2012. A leading researcher in the field of glycobiology and the emerging area of neuro-glycobiology, his recent research has focused on the interactions of myelin-associated glycoprotein, or MAG, and gangliosides.

willy-nilly and making inappropriate connections. But in gaining the ability to crowd nerves close together, we have given up flexibility - the ability to heal after injury."

Axon regeneration inhibitors, or ARIs, are molecules in the spinal cord



Horizontal section of the spinal cord showing highly fluorescent spinal motoneurons retrogradely labeled with fast blue and hydroxystilbamide (left) or fluoro gold (right) dyes. L. J. S. Yang, unpublished.

that stop nerve fibers from growing. "Treatments that eliminate ARIs might allow the nerve ends to regain their natural regenerative abilities, as they do in the periphery, and improve recovery," says Schnaar.

The researchers surgically severed nerves that normally extend from the spinal cord to the shoulder of anesthetized rats. They then transplanted a nerve from the hind leg of the same animal into the spinal cord to reconnect the injured nerve ends.

To coax the injured nerve ends to grow fibers and connect to the transplanted nerve, they used an implanted pump to bathe the area with one of three different enzymes known to destroy ARIs. Four weeks after transplantation and enzyme treatment, the researchers injected dyes into the nerves to see whether, and how many, nerve fibers grew from the injured cells of the

Continued on next page

Key Process in Cell Death Occurs As Single, Quick Event

The release of mitochondrial intermembrane space proteins into the cytosol is a key event that occurs during apoptosis. Using *in situ* fluorescent labeling, scientists at St. Jude Children's Research Hospital have now demonstrated that this phenomenon occurs as a single, quick event, rather than as a step-by-step process.

Results of the study indicate the formation of pores in the mitochondrial membranes is a rapid process that allows a nearly simultaneous, rather than sequential, release of many apoptosis proteins, according to Douglas Green, chair of the St. Jude Department of Immunology. Green is senior author of a report on this work that appears in the August 1 issue of *Proceedings of the National Academy of Sciences*.

The process of pore formation, or mitochondrial outer membrane permeabilization (MOMP), allows mitochondrial intermembrane space

spinal cord into the transplanted nerve.

Rats treated with one of the three enzymes tested, sialidase, showed well over twice the number of new nerve fibers than rats treated with saline. Moreover, the researchers saw that the new fibers were made by nerve cells residing in the spinal cord.

Having established the ability of sialidase to improve spinal nerve regeneration into transplanted peripheral nerves, Schnaar and his research team at Hopkins are testing the same treatment to see whether it will help nerve regeneration in other types of spinal cord injuries.

"Even a small improvement might mean a lot. People with spinal cord injuries generally are not looking to play football," says Schnaar, "but to regain basic functions. A modest improvement in nerve regeneration might make a big improvement in a patient's quality of life." ❧

proteins to escape and orchestrate the cell's destruction. MOMP is controlled by a family of proteins called Bcl-2; some of these support apoptosis and others interrupt the process. The pro- and anti-apoptotic Bcl-2 proteins cooperate to weigh and balance cell signals that promote survival or death. During apoptosis, these proteins are either already on the mitochondrial membranes or migrate to the membranes, where they trigger MOMP.

Using *in situ* fluorescent labeling of proteins tagged with a short tetracycline-containing sequence, the researchers were able to follow the release of the apoptotic proteins Smac, Omi, adenylate kinase-2, cytochrome *c*, and apoptosis-inducing factor (AIF) during apoptosis.

The team found that, after cells were treated with a chemical that triggers apoptosis, it took 3 to 10 minutes for cytochrome *c*, Smac, Omi and adenylate kinase-2 to escape together immediately following MOMP. However, AIF escaped from the mitochondrial membrane much more slowly and incom-

pletely, starting with the release of cytochrome *c* but continuing during the next few hours. The researchers concluded that, while AIF is known to regulate other cellular processes, the protein itself is not involved in triggering apoptosis.

"The slow, continuous release of apoptosis-inducing factor (AIF) suggests that the pore formed during MOMP remains open for many hours," Green said. "Our finding of nearly simultaneous, rather than sequential, release of the mitochondrial membrane proteins helps to explain the timing of the movement of these apoptosis proteins following MOMP. The findings also suggest that release of these proteins is not controlled by multiple levels of regulators, but rather occurs as a single event."

The study also highlights the importance of the Bcl-2 family in regulating the formation of pores in the mitochondrial membrane and emphasizes how critical the formation of these pores is to the regulation of apoptosis, Green said. ❧

ASBMB member Douglas R. Green holds the Peter C. Doherty Endowed Chair of Immunology at St. Jude Children's Research Hospital in Memphis, Tennessee. He received both his B.S. and Ph.D. in biology from Yale University in, 1977 and 1981 respectively. He was tenured at the University of Alberta



Dr. Douglas Green

in the Department of Immunology from 1989 to 1991, then served there as Adjunct Professor until 1993. From 1990 to 2005 Green served as member and head of the Division of Cellular Immunology at the La Jolla Institute for Allergy and Immunology. He con-

currently worked as Adjunct Professor in the Department of Biology at the University of California, San Diego, from 1994 to 2005.

Green is a prominent scientist in the field of apoptosis and is well known for his research on how a breakdown in this process can trigger cancer in lymphocytes and other types

of cells. In 2002 he received the MERIT Award from the National Institute of General Medical Sciences. Green holds several patents and is a member of the American Association of Immunologists and the American Association for Cancer Research.

Diana Beattie Appointed Dean Of Oman Medical College

Dr. Diana Beattie has been appointed Dean of the undergraduate campus of the Oman Medical College in Muscat, Sultanate of Oman. Oman Medical College, which has a partnership agreement with WVU, is a seven-year program, with the first three undergraduate years taught at the campus in Muscat, and the four-year medical school program taught in Sohar. Beattie was the long-time Chair of the Department of Biochemistry, and subsequently Chair of the Department of Biochemistry and Molecular Pharmacology, at West Virginia

University School of Medicine, and is a former Chair of the AAMC's Council of Academic Societies.


Dr. Beattie, who had been hoping to find a position in Oman following her visits to the college, said the decision to resign from her current role was an easy one to make. "After serving 21 years as chair, it was time for a change," Beattie said. "I needed to seek new interests and challenges."

Beattie, who was involved in the early planning stages for designing Oman Medical School, spent five weeks teaching and interacting with the stu-



Dr. Diana Beattie

dents in the spring of 2005 and 2006. "I enjoyed learning the culture and working with the students," Beattie said. "I look forward to bringing my experiences to Oman."

WVU's partnership with Oman Medical College offers WVU faculty members from the School of Medicine, School of Pharmacy and Eberly College of Arts and Sciences an opportunity to develop curriculum and provide instruction to premedical, medical, and pharmacy students in Oman. The school has nearly 400 students from Oman and several neighboring countries. As Dean, Beattie will be responsible for designing the curriculum, mentoring and managing faculty, and interacting extensively with students. 

Russell DeBose-Boyd Named Distinguished Young Scholar

ASBMB member Russell DeBose-Boyd, assistant professor of molecular genetics at University of Texas Southwestern Medical Center, has been named a Distinguished Young Scholar in Medical Research by the W. M. Keck Foundation. The grant accompanying this honor will support DeBose-Boyd's work on HMG CoA reductase, an enzyme that controls


the production of cholesterol and is the direct target for cholesterol-lowering drugs such as Zocor and Lipitor.

The W. M. Keck Foundation established the Distinguished Young Scholars in Medical Research Program to give young scientists the resources they need to pursue potentially breakthrough research projects in biomedicine. Since 1999, the program has awarded grants



Dr. Russell DeBose-Boyd

of up to \$1 million to each of five junior faculty investigators at research universities and institutions annually. Originally conceived as a five-year project, the program has been extended through 2008. Nominations from institutions are accepted on an invitation-only basis.

The four other 2006 fellows are: Dr. Luis Amaral of Northwestern University, Dr. Seth Blackshaw of Johns Hopkins University, Dr. Jonathan Bogan of Yale University School of Medicine and Dr. Amy Pasquinelli of the University of California, San Diego. 

Nicolas G. Bazan To Receive Proctor Medal

The Association for Research in Vision and Ophthalmology (ARVO) has selected ASBMB member Nicolas G. Bazan, M.D., Ph.D., to receive its Proctor Medal. This is ARVO's highest honor and it is presented annually for outstanding research in the basic or

clinical sciences as applied to ophthalmology. The award will be presented to Bazan during ARVO's Annual Meeting in Fort Lauderdale, Florida, in May 2007.

Bazan was chosen as the recipient of the Proctor Medal for his elucidation of the lipid pathways in the retina, his discovery of the DHA derivatives named docosanoids, his work on the platelet activating factor in inflammatory responses, and his design of

agents to prevent apoptosis arising out of this work.

Bazan is currently Director of the Louisiana State University of Health Sciences Center and Neuroscience Center of Excellence in New Orleans. He is also the Ernest C. and Ivette C. Villere Professor of Ophthalmology.



Dr. Nicolas G. Bazan

Emory School of Medicine Names Glycomics Expert New Chair of Biochemistry


Emory University School of Medicine has named Richard D. Cummings, a nationally recognized expert in the emerging research field of glycomics, as the new Chair of the Department of Biochemistry. Before joining the Emory faculty in June, Dr. Cummings was George Lynn Cross Distinguished Research Professor of Biochemistry and Molecular Biology at the University of Oklahoma Health Sciences Center. He held the Ed Miller Endowed Chair in Molecular Biology, was a professor of biochemistry and molecular biology, and was director and founder of the Oklahoma Center for Medical Glycobiology. An ASBMB member, he also served on the board of *The Journal of Biological Chemistry*.

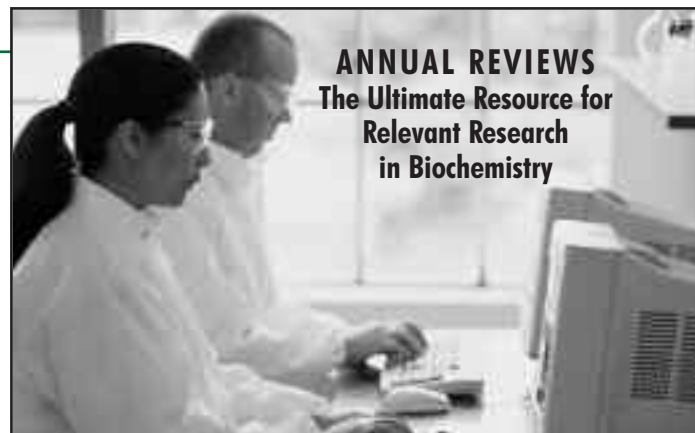


Dr. Richard Cummings

In 1999, Cummings was appointed co-director/coordinator of the newly established University of Oklahoma Bioengineering Center. Before joining the University of Oklahoma, he was professor of biochemistry at the University of Georgia and associate director of the UGA Complex Carbohydrate Research Center.

His research focuses on glycoconjugates, the carbohydrate molecules and their associated proteins that permit cells to communicate with, and adhere to, each other—transmitting and receiving chemical, electrical and mechanical messages that underlie all cellular and bodily functions. His research has a particular emphasis on the role of glycoconjugates in cardiovascular biology, autoimmune diseases, and parasitology. A hallmark of his research team has been the promotion of collaborative studies and training in glycobiology, and he has partnerships with more than a dozen other laboratories.

Dr. Cummings received his bachelor's degree in 1974 from the University of Montevallo in Montevallo, Alabama and his doctoral degree in 1980 from The Johns Hopkins University. He then was a postdoctoral fellow in Hematology/ Oncology at the Washington University School of Medicine in St. Louis. 



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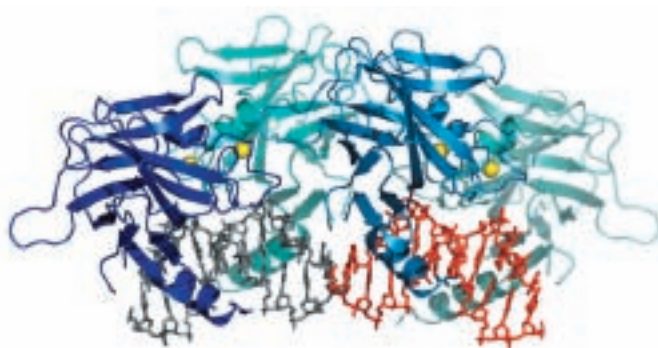


Structure of the p53 Core Domain Dimer Bound to DNA

William C. Ho, Mary X. Fitzgerald, and Ronen Marmorstein

J. Biol. Chem., 2006 281: 20494-20502

p53 is a transcription factor that regulates the cell cycle and thus functions as a tumor suppressor. In its active form, p53 forms a dimer of dimers. In this article, the authors use a cross-linking strategy to trap a p53 core domain dimer bound to DNA for structure determination by x-ray crystallography. Their 2.3 Å structure reveals the molecular details of cooperative dimeric p53 binding to DNA that involves a zinc-binding domain. The researchers also discovered that a hot spot for tumor-derived mutations maps to the dimerization region, reinforcing its functional importance. Interestingly, residues associated with p53 dimer formation on DNA are poorly conserved in the p63 and p73 paralogs, possibly due to their functional differences. The authors also used the dimeric protein-DNA complex to model a dimer of p53 dimers bound to icosamer DNA. Their model suggests that the p53 core domain dimer-dimer contacts are less frequently mutated in human cancer than intra-dimer contacts.



A p53DBD tetramer bound to icosamer DNA.

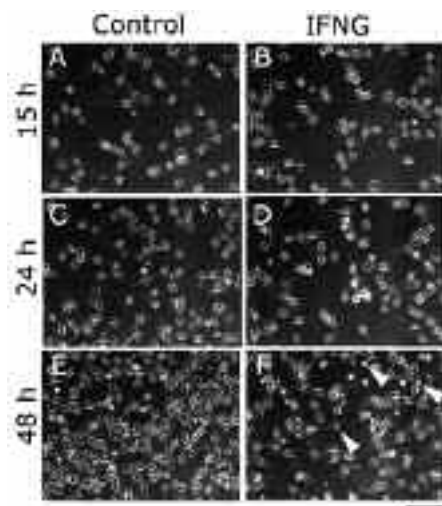
MEK-ERK Signaling Is Involved in Interferon-Induced Death of Oligodendroglial Progenitor Cells



Makoto Horiuchi, Aki Itoh, David Pleasure, and Takayuki Itoh

J. Biol. Chem. 2006 281: 20095-20106

Inflammatory reactions in the central nervous system play important roles in the pathogenesis of white matter diseases such as multiple sclerosis and periventricular leukomalacia. These reactions result in the exposure of oligodendrocytes to various cytokines that affect oligodendroglial survival, proliferation, and differentiation. One of these cytokines, interferon- (IFNG), has been reported to have both a deleterious and protective role in myelin synthesis. Here, the authors show that simultaneous activation of the signal transducers and activator of transcription (STAT) pathway by IFNG and of the extracellular signal-regulated kinase (ERK) pathway by exogenous trophic factors plays a role in interferon-induced cytotoxicity in proliferating oligodendroglial progenitors. The effect is developmental stage-specific in that non-proliferating immature and mature oligodendrocytes are protected from interferon-induced cell death.

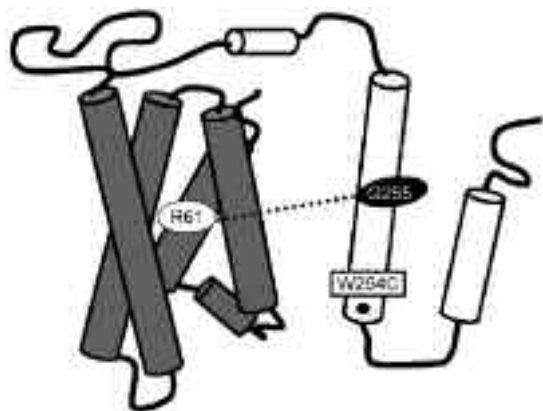


IFNG induces cell death in oligodendroglial progenitor cells.

C-terminal Interactions of Apolipoprotein E4 Respond to the Postprandial State

Sarada D. Tetali, Madhu S. Budamagunta, John C. Voss, and John C. Rutledge

J. Lipid Res. 2006 47: 1358-1365.



Schematic representation of apolipoprotein E4 (apoE4) structure.

Apolipoprotein E (apoE) is involved in the assembly, processing, and removal of plasma lipoproteins. Its C-terminal domain has a high affinity for lipid and is responsible for lipoprotein lipid binding. Several studies have suggested that apoE undergoes a conformational change upon association with lipids, and that it adopts distinct conformational and oligomeric states when associated with different classes of lipid particles. In this paper, the authors take advantage of the fact that the postprandial state offers a dramatic change in the levels and ratios of lipoprotein particles and investigate the structural changes in the apoE4 C terminus in the preprandial and postprandial states. Using electron paramagnetic resonance (EPR) spectroscopy they are able to detect rearrangements of apoE4 induced by the postprandial state and specifically by triglyceride-rich lipoprotein (TGRL) lipolysis products.

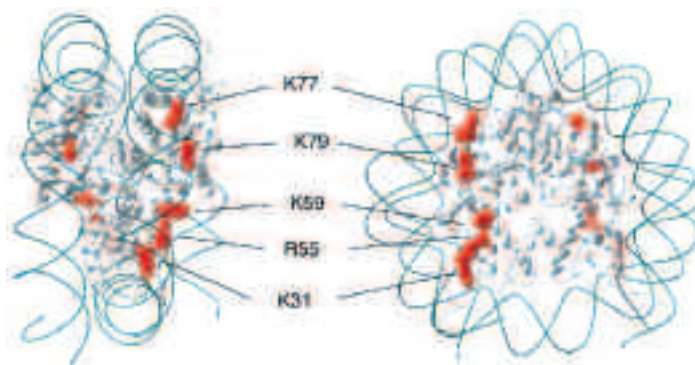


Quantitative Proteomic Analysis of Post-translational Modifications of Human Histones

Hans Christian Beck, Eva C. Nielsen, Rune Matthiesen, Lars H. Jensen, Maxwell Sehested, Paul Finn, Morten Grauslund, Anne Maria Hansen, and Ole Nørregaard Jensen

Mol. Cell. Proteomics 2006 5: 1314-1325.

A central mechanism for regulating chromatin activity is the reversible covalent modification of histones. The combinatorial nature of these modifications constitutes the "histone code" that dictates chromatin structure and function during development, growth, differentiation, and homeostasis of cells. However, deciphering the histone code is hampered by the lack of analytical methods for monitoring the combinatorial complexity of reversible multi-site modifications of histones. To address this problem, the authors of this paper used LC-MS/MS technology and Virtual Expert Mass Spectrometrist software for qualitative and quantitative proteomic analysis of histones extracted from human small cell lung cancer cells. They were able to locate a total of 32 modifications in the four core histones, including seven novel modifications. The authors also performed a quantitative proteomic study of the dose-response effect of a histone deacetylase inhibitor on histone acetylation in human cell cultures and found that the inhibitor affects acetylation in a site-specific and dose-dependent manner.



Localization of the novel modification sites identified by LC-MS/MS on nucleosome crystal structure.

by John D. Thompson, Editor

Drug Makers Urge Congress to Increase FDA Funding

James C. Greenwood, former congressman and now President of the Biotechnology Industry Organization, stood before an audience at the Massachusetts Biotechnology Council's annual meeting recently and outlined his next challenge: getting Congress to increase Food and Drug Administration funding.

Greenwood displayed a chart indicating zero growth in congressional appropriations to the FDA from fiscal years 1986 through 2006. In sharp contrast, he said, fees paid by the drug industry to the FDA to speed promising treatments to market have doubled from 1998 to 2005. Now, the FDA wants pharmaceutical companies to pay even more to help it better ensure drugs that patients take are safe.

He and others in the industry acknowledge that the FDA is chronically

under-funded and stressed by new responsibilities such as planning for a flu pandemic, and they agree that adequate funding for drug safety is an admirable goal. They just don't want all of the money coming out of the industry's pocket. However, getting Congress to adopt the industry's point of view could be a hard sell, and as more and more new drugs come into the testing pipeline debate is intensifying over who will foot the bill.

"Very simply, FDA is drowning under the weight of its added responsibilities and budget woes, and it sees user fees as its life line," said Greenwood. In fiscal 2004, drug companies paid \$232 million in fees to the FDA, accounting for 53 percent of the agency's \$436 million budget for new-drug review. The 2007 budget includes

\$320.6 million in fees to be paid by drug makers.

Every 100 additional reviewers shortened the time it took the FDA to consider a new drug application by 3.4 months, according to Harvard University government professor Daniel Carpenter. Even the most promising life-saving drugs took 16.3 months to review in 1993. Last year, the median time for review and approval was six months for high-priority drugs. Many would like to see the same kind of progress made in how the FDA monitors the safety of drugs after they go to market.

Negotiations between the FDA and drug makers over user fees were likely to continue behind closed doors through Labor Day, but some are taking the battle public through fiery statements like those Greenwood made in Boston.

Does Success Mean New Challenges for Ireland?

For Western Europe's nations, Ireland's success is a sore point. Why? Those Western European nations that poured billions of dollars into "poor struggling, little Erin," now see Ireland marketing its products and looking to the U.S. and Britain for needed investment capital. To Western European eyes it's a bit like seeing the youth they raised and showered with euros leave home, without even a thank you, to reap the rewards of American dollars and British pounds. It's not only the sense of being shunned for richer markets by their protégé that riles Western Europeans, it's the fact that Ireland's booming economy is attracting hordes of ambitious and talented immigrants yearly, the bulk of them from Eastern Europe and not from the high unem-

ployment nations of Western Europe.

Ireland's success, however, has come with some new problems. While it has fueled an expansion of high-value exports and manufacturing, the nation's domestic economy remains uncompetitive and the public service sector inefficient. That translates into high costs for energy, distribution, business services, and the basic cost of living. Still there is ground for hope.

In the first half of 2005 Ireland attracted 25% more venture capital than in the same period of 2004. Then, to give more impetus to Ireland's economic growth, the state's Industrial Development Agency made a proposal to invest some \$90 million to create a National Institute for Bioprocessing, Research and Training. The new facil-

ity is intended to demonstrate Ireland's commitment to be a leader in this industry.

Further support for biotech has come from the Higher Education Authority's allocation of \$380 million for bioscience research and nearly \$40 million for such specific research operations as the Alimentary Pharmabiotic Center which partners with Procter and Gamble, and other centers in Cork, Dublin, and Galway.

Adding to Ireland's prospects is a reverse brain-drain, which finds many of the nation's best-and-brightest, who left the country for better opportunities elsewhere in the 1980s and 90s getting back on the plane for a return trip. The skills and talent they gained abroad will be coming home with them.

Momenta Inks Deal with Novartis' Sandoz

Generic drug giant Sandoz has purchased a \$75 million stake in Momenta Pharmaceuticals Inc., a small biotechnology company that develops generic, genetically engineered drugs. Under terms of the deal, the two companies will develop four generic drugs.

Cambridge, Massachusetts-based Momenta specializes in the sequencing and design of complex sugars to develop improved versions of existing drugs, as well as developing new drugs. Sandoz, the generics branch of Switzerland-based Novartis AG, will pay Momenta \$75 million upfront to buy

4.7 million shares at a price of \$15.93 each. Momenta is also eligible for a total of \$188 million in additional payments if all its milestone achievements are reached. Following news of the deal, shares of Momenta surged, rising \$4.97, or 38.1%, to close at \$18.02 on the NASDAQ.

The companies have had a partnership since November 2003 on the generic drug M-Enoxaparin, which is used to treat deep vein thrombosis and several cardiovascular conditions, and Momenta CEO Alan Crane said that collaboration helped pave the way for this latest deal. "That relationship has

demonstrated to Sandoz both the strength of our technology and the strength of our broader capabilities as a company, as well, to execute on and move products forward," he said in a conference call with analysts.

Sandoz CEO Andreas Rummelt said both companies will benefit from their combined technologies and Sandoz's global marketing and manufacturing infrastructure. "Our intent is to set new standards for the characterization of complex drugs and for bringing follow-on versions to the market as quickly as possible. That will contribute to reducing healthcare costs," he noted.

California Biotechs Freed from Sharing Stem-Cell Research Data

A panel to decide what benefits California taxpayers will receive from their \$3 billion investment in stem cell research recently agreed to remove a discovery-sharing requirement that the biotech industry vigorously opposed. Biotech leaders had argued that being forced to freely share their patented inventions with California research institutions could stymie stem cell research, by removing financial incentives for companies to get involved.

"We do not want to hurt this industry," agreed Jeff Sheehy, a member of the Intellectual Property Task Force of the state's stem cell agency. "We have a policy that industry has told us will not work for them."

Yet the task force did not give industry leaders everything they

wanted. It decided to keep intact requirements that a share of royalties be returned to state coffers, that companies adopt plans to provide access to medical therapies for uninsured people, and that the stem cell agency have the right to suspend exclusive licensing agreements if companies do not make inventions available to the public in a timely manner.

The policy deals only with firms that obtain a licensing agreement with a university or non-profit institution. A separate policy for firms that receive stem cell research grants directly from the state will be set at a later date.

At issue is what return California taxpayers will receive on their sizable investment in stem cell research, which was approved with the passage of Proposition 71 in 2004.

JK Agri to Develop New Generation Bt Cotton

India's Financial Express reports that Hyderabad-based JK Agri Genetics is planning to develop a new generation of Bt cotton, using the Cry 1 EC gene grown by the National Botanical Research Institute (NBRI), Lucknow.

NBRI is a public sector research institution, affiliated with the Council of Scientific and Industrial Research (CSIR). Recently, it transferred the technology to JK Agri Genetics to develop and commercialize a new generation of Bt cotton. JK launched its indigenous Bt cotton this summer, after getting clearance from the Genetic Engineering Approval Committee (GEAC).

The new Cry 1 EC gene developed by NBRI would be pyramided with the recently released material, containing Cry 1 Ac gene, said official sources. The company claimed that the proposed new generation Bt cotton will give broader insect resistance coverage, particularly against Spodoptera (tobacco caterpillar).

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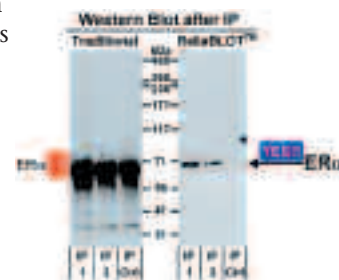
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Calendar of Scientific Meetings

SEPTEMBER 2006

7th Siena Meeting from Genome to Proteome: Back to the Future

September 3–7 • Siena, Italy www.unisi.it/eventi/proteome/

29th European Peptide Symposium

September 3–8 • Gdansk, Poland
www.29eps.univ.gda.pl; E-mail: 29eps@chem.univ.gda.pl
Ph: 48-58-3450363

47th International Conference on the Bioscience of Lipids — ICBL-ELIFE-ILPS Joint Meeting

September 5–10 • Pécs, Hungary
For information contact: www.cbi12006icbl2006.hu/

5th European Conference on Computational Biology

September 10–13 • Eliat, Israel
www.eccb06.org/; E-mail: eccb06@diesenhaus.com
Ph: 972-3-5651313

American Chemical Society National Meeting and Expo

September 10–14 • San Francisco
For Information: Department of Meetings & Expositions Services; Kathleen Thompson, Assistant Director, Melissa Redd, Assistant
Fx: 202-872-6128; Ph: 202-872-6061
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5th European Congress of Biogerontology

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

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

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

2nd International Conference: Metzincin Metalloproteases in Health and Disease

September 24–29 • Monte Verità, Ascona, Switzerland
Information, registration and abstract submission: www.metzincin.unibe.ch
Organizers: Erwin Sterchi, Judith Bond, Walter Stoecker
Contact: erwin.sterchi@mci.unibe.ch

OCTOBER 2006

4th Euro Fed Lipid Congress

October 1–4 • Madrid, Spain
www.eurofedlipid.org/meetings/madrid/index.htm
Email: amoneit@eurofedlipid.org

International Conference of Immunogenomics and Immunomics

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

3rd Annual Scientific Forum of the Midwest Lipid Association

October 20–22 • Kansas City, MO
www.lipid.org/chapters/mwla; Email: ssheridan@lipid.org

Asilomar Conference on Mass Spectrometry

October 20–24 • Asilomar Conference Center, Pacific Grove, CA
Fundamentals of Gas Phase Ion Chemistry: Experiment and Theory
Program Chairs: Frantisek Turecek and Thomas Morton
For information contact: ASMS
Ph: 505-989-4517; Email: asms@asms.org; www.asms.org

FEBS Special Meeting: European Lipidomics Initiative

October 21–25 • Noordwijkerhout, The Netherlands
www.febslipid2006.chem.uu.nl/

4th International Conference on Structural Genomics

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

NHUP0 5th Annual World Congress

October 28–November 1 • Long Beach, CA
www.hupo2006.com; E-mail: Wehbeh.Barghachie@mcgill.ca
Ph: 514-398-5063

The Liver Meeting 2006— 57th Annual Meeting of the American Association for the Study of Liver Disease

October 27–31 • Boston, MA
www.aasld.org/eweb/DynamicPage.aspx?webcode=2006_AnnualMeeting

NOVEMBER 2006

Transcriptional Regulation by Chromatin and RNA Polymerase I I

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

43rd Japanese Peptide Symposium/4th Peptide Engineering Meeting

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

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

November 9–10 • Catamaran Resort, San Diego
Program Chairs: Victor Ryzhov and Richard Vachet
For information contact: ASMS
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NIH 4th Symposium — Functional Genomics of Critical Illness and Injury

Surviving Stress: Organ Systems to Molecules

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

Annual meeting of the Society for Glycobiology

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

The 19th Annual Tandem Mass Spectrometry Workshop

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

DECEMBER 2006

Second ISN Special Neurochemistry Conference: Neural Glycoproteins and Glycolipids

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

19th World Diabetes Congress

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

American Society for Cell Biology 46th Annual Meeting

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

JANUARY 2007

Sanibel Conference

January 19–22 • Sundial Beach Resort, Sanibel Island, Florida
Imaging Mass Spectrometry
Program Chairs: Richard Caprioli, Ron Heeren, and Markus Stoekli, For information contact: ASMS
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MARCH 2007

U.S. HUPO 2007

March 4–8 • Seattle
For information contact: www.ushupo.org
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Association for Biomolecular Resource Facilities

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

APRIL 2007

Second Workshop on Biophysics of Membrane-active Peptides

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

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

April 28–May 2 • Washington, DC
Contact: ASBMB 2007, 9650 Rockville Pike, Bethesda, MD 20814-3008
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www.kenes.com/prediabetes2007;
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