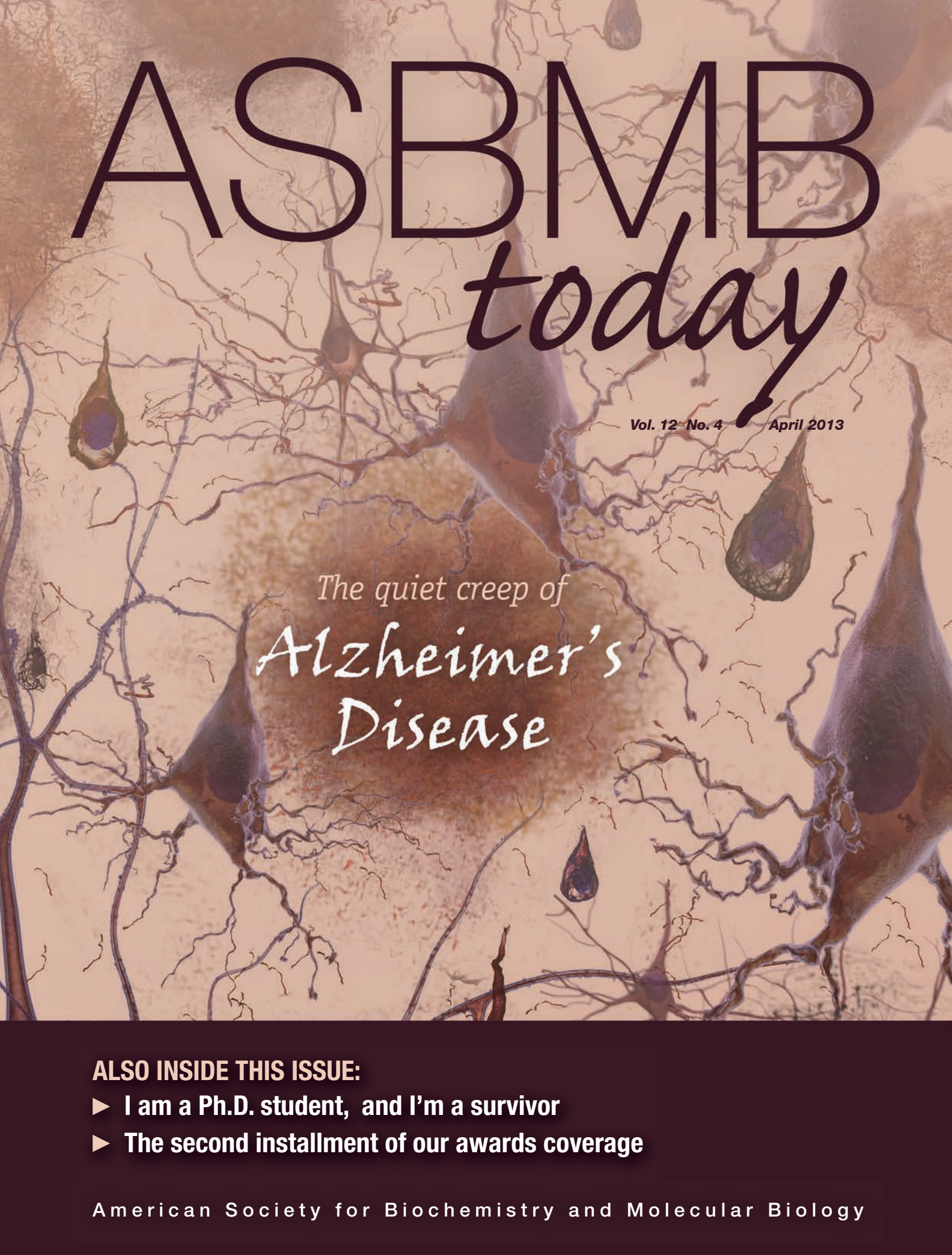




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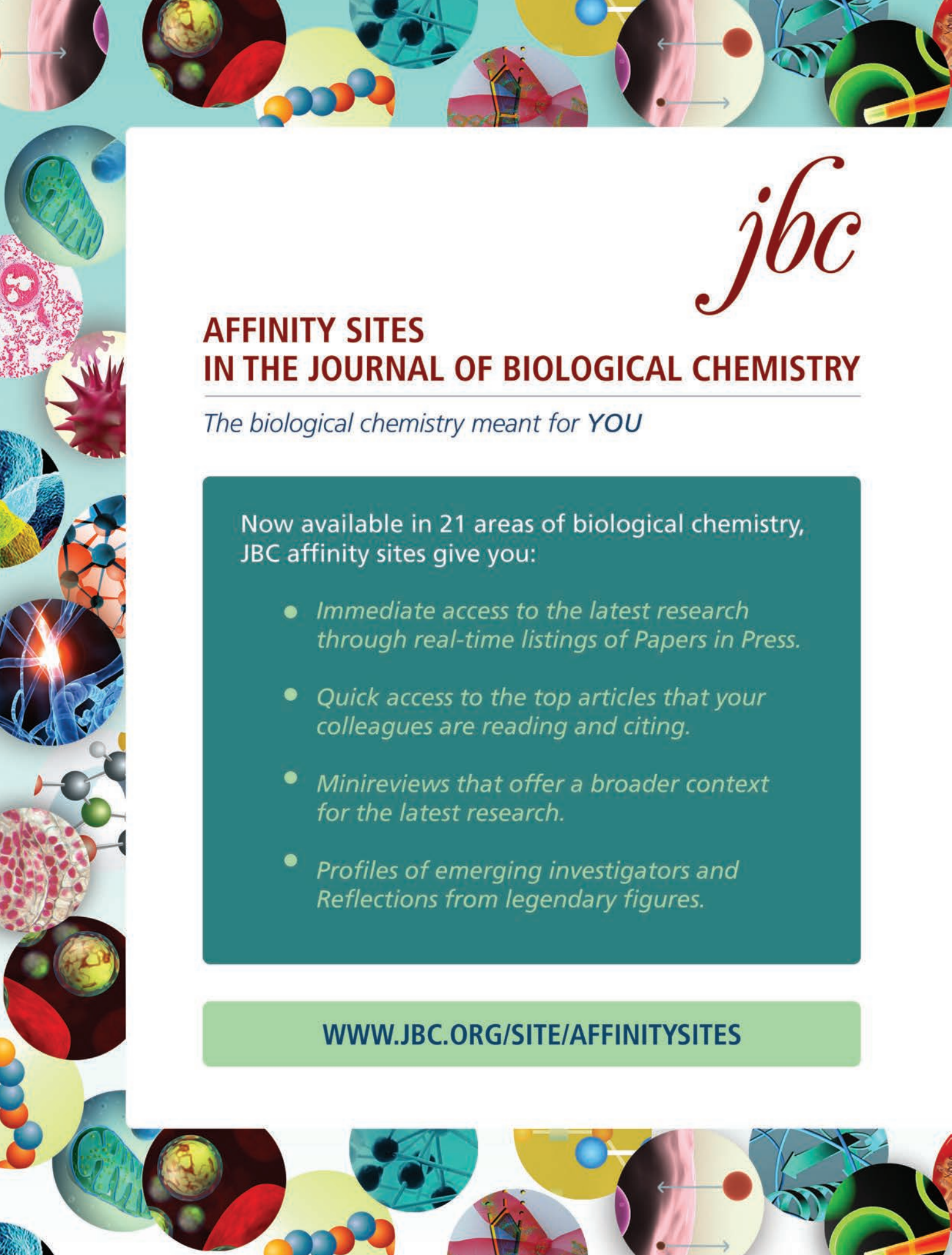
Vol. 12 No. 4 April 2013

The quiet creep of
**Alzheimer's
Disease**

ALSO INSIDE THIS ISSUE:

- ▶ I am a Ph.D. student, and I'm a survivor
- ▶ The second installment of our awards coverage

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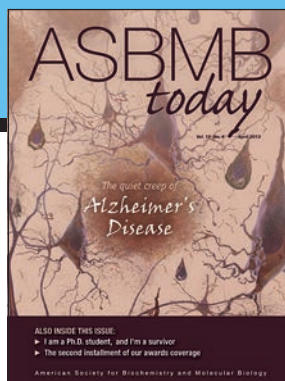
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ASBMB Today

Angela Hopp *Editor*
ahopp@asbmb.org

Rajendran Mukhopadhyay
Sr. Science Writer/Editor
rmukhopadhyay@asbmb.org

Marnay Harris *Designer*
mharris@asbmb.org

Andrew Harmon *Science and Technology
Publishing Manager*, aharmon@asbmb.org

Nancy J. Rodnan *Director of Publications*
nrodnan@asbmb.org

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On the Wild Types blog

It very well may be possible that compromised leukotriene signaling is one of the reasons Neanderthals are not among us today. In a recent paper in the *Journal of Lipid Research*, investigators compared the genome sequences of *Homo neanderthalensis* and *Homo sapiens* to see if the two hominid subspecies shared genes for the biosynthesis of leukotrienes and other inflammatory mediators. The investigators found that the Neanderthal genome contained six genes encoding for six different lipid-peroxidizing isoenzymes called lipoxygenases (LOXs). Previous work has shown that we too have six LOX genes. However, the cDNA for two of the enzymes contained premature stop codons in the Neanderthal sequence, suggesting that expression of these enzymes was compromised. **Read more on the ASBMB Today blog, Wild Types.**

New Chi Omega Lambda inductees

The American Society for Biochemistry and Molecular Biology's Biochemistry and Molecular Biology Honor Society, Chi Omega Lambda, recognizes exceptional undergraduate juniors and seniors pursuing degrees in the molecular life sciences at colleges and universities that are members of the Undergraduate Affiliate Network. Students are recognized for their scholarly attainment, research accomplishments and outreach activities. Sixteen students were inducted into Chi Omega Lambda in 2013. **See the full list online.**



Hill Day coverage

In mid-March, the ASBMB public affairs office escorted members to Capitol Hill to promote funding for scientific research. **Visit our website for photos and more.**

ASBMB Today takes summer break

Please note that ASBMB Today will publish a combined June/July issue this summer. Monthly publication will resume in August.



Sequester

Impact on the national debt: minimal

Potential damage to many research laboratories: priceless

BY JEREMY BERG

My column is due near the first of each month. As I was starting to write this column in late February, I was following the news regarding the automatic spending cuts set to take place March 1, also known as sequestration or the sequester. Given the amount of brinksmanship that we have seen over the past few years, I expected lots of public posturing with behind-the-scenes negotiations leading to some last-minute act that would at least postpone the implementation of the sequester. I was wrong. March arrived, and there was no deal and, it appears, no serious negotiation.

In many phases of our lives, we struggle to find effective ways to motivate ourselves and others around us. This usually involves some combination of rewards and punishments: If my daughter completes her paper, then she can go to the mall with her friends; if I don't complete my workout, then the Wii will criticize me (and, for some strange reason, I care).

The sequester was intended to be such a motivational tool. Across-the-board cuts affecting both nondefense and defense spending were designed to be so undesirable to members of Congress on both sides of the aisle that the threat alone would motivate them to work out a compromise to reduce the deficit without the sequester going into effect. By design, the sequester was intended to be bad policy; cuts would be made to all affected programs equally without analysis of the quality of the programs or the impact of the cuts.

What is the impact of the sequester on the national deficit and debt?

In fiscal 2012, the U.S. budget was \$3.803 trillion, of which 62.8 percent involved mandatory spending (such as interest on the debt, Social Security, Medicare and Medicaid) and 37.2 percent involved discretionary spending appropriated annually by Congress.

Discretionary spending can be divided further into defense discretionary spending and nondefense discretionary, or NDD, spending. In 2012, NDD represented 36.7 percent of discretionary spending, or 13.7 percent of

the budget.

The sequester is a budget reduction of \$85.4 billion from March 1 to Sept. 30 and is focused largely on discretionary spending, with more than 80 percent of the cuts coming from the NDD or defense discretionary categories. Thus, the sequester represents a cut of \$85 billion out of \$3.8 trillion (although the final budget for fiscal 2013 is not yet settled), or approximately 2.2 percent of the budget (with the sequester in effect for only seven of 12 months of 2012).

The sequester is not a one-year adjustment but applies from 2013 to 2021. Over this period, the sequester is expected to avoid \$1 trillion in spending, with additional savings in interest payments, to yield a total savings of \$1.2 trillion. However, because mandatory spending, particularly Medicare expenditures, is anticipated to continue to rise, the national debt (the accumulated result of budget deficits) is projected to rise from \$16 trillion to \$26 trillion over this period.

What is the anticipated impact of the sequester on science funding?

Cuts from the NDD category account for \$28.7 billion, or 5.2 percent of the NDD budget. The National Science Foundation budget is \$7.0 billion this year (based on the continuing resolution in effect until March 27), so the sequester cut is \$370 million. The National Institutes of Health budget for this year is \$30.8 billion, and the sequester cut is \$1.5 billion.

How will these agencies handle these budget reductions? NSF Director Subra Suresh issued a statement in late February saying existing grants will not be affected but that the primary impact will be a reduction of 1,000 in the number of new grants awarded (1). Given that the NSF typically awards approximately 13,000 grants annually, this represents a reduction of approximately 8 percent. (NSF fully funds many multiyear awards, so it is difficult to reduce existing awards.)

Prior to sequestration, the NIH released a notice that indicated the agency would use a strategy that has

become all too familiar in recent years: reducing non-competing grants below their previously committed levels to avoid decreasing the number of new and competing awards that can be made (2).

Because the average NIH grant lasts four years and the funds for almost all grants are disbursed incrementally, approximately 75 percent of the NIH budget is committed to grants that already have been awarded. If these commitments were not touched, then the full brunt of sequestration cuts would come down on new and competing grants.

Suppose that 80 percent (corresponding to the fraction of the NIH budget devoted to extramural research) of the \$1.5 billion cut was taken out of new and competing grants. Given an average grant size of \$450,000 (direct and indirect costs), this would correspond to a loss of 2,667 grants. Given that NIH Director Francis Collins testified in June before the U.S. House Energy and Commerce Health Subcommittee that a nine-month sequester totaling \$2.4 billion would result in the loss of 2,300 grants (3), the NIH clearly intends to cut noncompeting grants. On March 4, Deputy Director for Extramural Activities Sally Rockey issued a letter to grantees (4) regarding the NIH's plans. This letter laid out all of the options, to wit:

"Examples of this impact could include: not issuing continuation awards, or negotiating a reduction in the scope of your awards to meet the constraints imposed by sequestration. Additionally, plans for new grants or cooperative agreements may be re-scoped, delayed, or canceled depending on the nature of the work and the availability of resources."

Unfortunately, the letter did not provide much insight into the likely balance of approaches. In fairness, the situation is still quite uncertain, because the government is operating under a continuing resolution that expires on March 27, and the situation could change, for better or worse, as an appropriation for the rest of this fiscal year is developed.

We can estimate that at the NIH the sequester will result in the loss of 1,400 new and competing grants (based on Collins' statement about the larger cuts). This

represents a reduction of 15 percent from the already low number of 9,158 grants made in fiscal 2012. This reduction will further reduce success rates, which are already at historic lows. Many of the lost grants will affect established and productive laboratories competing for their only major source of funding, while others will affect younger investigators launching their independent careers. For some investigators, the loss of these funding opportunities (despite having outstanding peer-review scores) will cause them to leave academic research.

I await April 1 every year with some trepidation, because my wife has been known to catch me with some vicious April Fools' Day tricks. One year she convinced me that the owners of the house for which we had a signed an offer had decided not to sell after all. She let me fume and fuss and get ready to contact our real estate agent and perhaps a lawyer before she let me off the hook. This year, I hope to start April by finding out that the failure to stop the implementation of the sequester was all a joke.

Update: On March 21, the Congress passed a continuing resolution that funds the government through the end of the fiscal year (5). This resolution did not substantially restore the sequester cuts to the NIH budget, although the impact on the NSF budget was reduced to a 2.9 percent cut.



Jeremy Berg (jberg@pitt.edu) is the associate senior vice-chancellor for science strategy and planning in the health sciences and a professor in the computational and systems biology department at the University of Pittsburgh.

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At ASBMB, we believe, as English essayist James Henry Leigh Hunt once said,

"COLORS ARE THE SMILES OF NATURE."

That's why we've eliminated color figure fees for members publishing as corresponding authors in The Journal of Biological Chemistry and Molecular & Cellular Proteomics and reduced color figure fees to \$50 for members publishing as corresponding authors in the Journal of Lipid Research. So, bid farewell to that leaden look and let nature's smiles liven up your manuscripts.

Scientific conferences under threat

BY CHRIS PICKETT

"[M]any insights are possible only because of close, personal interactions among scientists who see each other regularly: those who do not work at the same university or laboratory must rely on interacting with each other at conferences."

– U.S. Rep. Rush Holt, D-N.J., Feb. 27, 2013

Communication and collaboration are cornerstones of the research enterprise, and scientific conferences are essential for the exchange of ideas that lead to breathtaking breakthroughs. Any scientist who attends a conference gains some insight into what others in his or her field are doing and just how unique his or her own work is. A bill under consideration in the U.S. House of Representatives, however, could severely limit the ability of federal scientists to take part in such conferences.

Written in response to a scandal concerning wasteful spending on conferences by those at the U.S. General Services Administration, the Government Spending Accountability Act of 2013 (officially known as House Resolution 313) could cause significant damage to the scientific community if it passes.

While H.R. 313 would, for the most part, pertain to travel for federal employees, such as scientists at the National Institutes of Health, the Centers for Disease Control and Prevention, and the national labs, among others, the American Society for Biochemistry and Molecular Biology is concerned with four provisions in H.R. 313.

The bill states;

1. That a federal employee giving a presentation at a conference also must make that presentation available on the agency's website. This is problematic for scientists, because the reason for giving a presentation at a conference – to exchange ideas and find new paths forward – is very different from the reason for publication, which is to tell a complete story with a clear conclusion. Federal scientists, faced with either publishing their work prematurely or not giving presentations at conferences, may elect not to give presentations and may decide not to attend altogether, thereby limiting these important interactions.

2. That each agency will have a limited amount of funds that it may spend on employee travel expenses. Coordinating meeting attendance at an agencywide level could result in junior scientists missing out on opportunities to attend conferences, meet new mentors and further their careers.

3. That the amount of money a federal agency may use for conferences in a year will be limited. Many smaller conferences depend on federal grants to support meetings. Restricting these funds will reduce the opportunities for scientists to interact, collaborate and make innovative discoveries.

4. That no more than 50 agency employees may travel abroad for international conferences in a year. Some major international conferences, such as international AIDS conferences, are attended by hundreds of researchers from a single agency, and this interaction is important for the global fight against such diseases.

While the above provisions apply to federal scientists, one possible outcome of this bill could significantly affect grant study sections. Because grant reviewers are technically federal employees during a study section, H.R. 313 could limit travel to and from those meetings. Discussions at study sections that hash out the good and bad aspects of grant proposals are a necessary and beneficial part of the peer-review process.

While we at the ASBMB support the federal government's goal to reduce wasteful spending, we favor a more nuanced approach than what is proposed in H.R. 313. We would prefer a bill that applies reasonable restrictions on travel that do not inhibit the mission of the agency. For scientific agencies, this means enough freedom to travel to meetings to ensure that the scientific enterprise is working efficiently. The current one-size-fits-all approach to travel restrictions could damage significantly the scientific enterprise and eliminate an important avenue of scientific collaboration.



Chris Pickett (cpickett@asbmb.org) is the science policy fellow at the ASBMB.

de Lange, Cantley, Yamanaka win Breakthrough Prizes



DE LANGE



CANTLEY



YAMANAKA

Three American Society for Biochemistry and Molecular Biology members in February each won a Breakthrough Prize in Life Sciences, which recognizes achievement and focuses on curing disease and prolonging human life. Titia de Lange, director of the Anderson Center for Cancer Research at Rockefeller University, was recognized “for her work on telomeres, illuminating how they protect chromosome ends and their role in genome instability in cancer.” Lewis C. Cantley, professor and director of the cancer center at Weill Cornell Medical College and NewYork-Presbyterian Hospital, won for the discovery of the PI-3 kinase pathway and its role in cancer metabolism. Shinya Yamanaka, a senior investigator at the Gladstone Institutes and a professor at Kyoto University in Japan, was honored for his work transforming adult stem cells into embryolike stem cells. The prizes are awarded by the Breakthrough Prize in Life Sciences Foundation, a nonprofit dedicated to advancing breakthrough research, celebrating scientists and encouraging the pursuit of science careers.

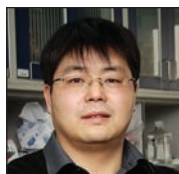
Protein Society recognizes van der Donk, Doudna, Shao



VAN DER DONK



DOUDNA



SHAO

In advance of its annual conference, The Protein Society selected three ASBMB members for honors. Wilfred van der Donk, professor at the University of Illinois at Urbana-Champaign, won the 2013 Emil Thomas Kaiser Award for his use of synthetic organic chemistry and protein chemistry to examine enzymatic reactions. Jennifer Doudna, professor of biochemistry, biophysics and structural biology at the University of California, Berkeley, was one of two recipients of the 2013 Hans Neurath Award for her study of three-dimensional structures of noncoding RNA to investigate the regulation of protein products by ribonucleoproteins. Feng Shao, investigator at the National Institute of Biological Sciences in Beijing, won the 2013 Protein Society Young Investigator Award for his work investigating how pathogenic bacteria modulate signaling cascades and evade

host immunity. The three winners will be recognized at the 27th Annual Symposium of The Protein Society in July.

Guengerich honored with Society of Toxicology Merit Award



GUENGERICH

F. Peter Guengerich has won the Society of Toxicology Merit Award for 2013. Guengerich, a professor of biochemistry at Vanderbilt University School of Medicine, has been pursuing for three decades a better understanding of how cytochrome P450 enzymes metabolize

drugs and carcinogens, the bioactivation of halogenated hydrocarbons and polymerase interactions with carcinogen-modified DNA. Guengerich is an associate editor of Chemical Research in Toxicology and The Journal of Biological Chemistry. He also is on the editorial boards of Critical Reviews in Toxicology and Nature Reviews, Drug Discovery. Guengerich gave his award lecture, “Bioactivation, Covalent Binding, and Toxicity: A Personal Odyssey,” at the society’s annual meeting in March in San Antonio.

Masters named president-elect of TAMEST



MASTERS

Bettie Sue Masters was named president-elect of The Academy of Medicine, Engineering and Science of Texas in February. Masters, a former president of the ASBMB, holds the Robert A. Welch Distinguished Chair in Chemistry in the biochemistry department at the School of

Medicine at the University of Texas Health Science Center in San Antonio. Kenneth L. Kalkwarf, president ad interim of the Health Science Center, emphasized the appointment “recognizes her career contributions as a scientist, her willingness to speak and serve on important issues, both in Texas and on the national and world stage, and not least, her mentorship of younger scientists.” Masters, a member of TAMEST’s founding board of directors, said, “This is, indeed, an honor and a privilege for me to serve in this capacity for TAMEST, a unique organization in the United States ... I hope that this organization increasingly becomes a source of information about science, engineering and technology for the public and our state legislators.” Masters’ term begins in 2014. TAMEST, which promotes education in science, technology, engineering and math and recognition of rising stars in science, as well as examination of energy and environmental issues, has a membership of more than 250 National Academies members and Nobel laureates. Nobel laureates Michael S. Brown and Richard Smalley were its founding co-chairs.

— Compiled by Anna Shipman



ALICE AND C.C. WANG AWARD IN MOLECULAR PARASITOLOGY

Goldberg wins Alice and C.C. Wang Award in Molecular Parasitology

BY NATALIE OSAYANDE

Daniel Goldberg, professor of medicine and molecular microbiology and co-chief of the division of infectious diseases at Washington University in St. Louis School of Medicine, is the winner of the American Society for Biochemistry and Molecular Biology's Alice and C.C. Wang Award in Molecular Parasitology.

The award is given to investigators who are making revolutionary contributions to the field. Goldberg was nominated for his contributions to our understanding of the biology of *Plasmodium* food vacuoles, primarily in *Plasmodium falciparum*, and the roles of proteases in the survival of parasites.

"Dr. Goldberg has defined the complex cellular trafficking of parasite food vacuole proteases and the mechanisms by which these malaria proteases work synergistically within the food vacuole to degrade hemoglobin to provide amino acids for the parasite," explained Kami Kim, a professor at Albert Einstein College of Medicine, in her nomination of Goldberg for the award.

Goldberg's work includes the examination of proteases that play important roles in the egress, the cell cycle and export of surface proteins, said L. David Sibley of Washington University in St. Louis School of Medicine.

"These and other achievements are all the more impressive in that they have been conducted in a system that most would consider intractable," Sibley said. "It is one thing to do elegant molecular biology in yeast and quite another to do so in parasites — and, unlike many parasites, no one would claim that *Plasmodium* is a model for anything!"

Goldberg is regarded as a pioneer of molecular parasitology, having developed molecular methods for transfection of selectable markers, transposon mutagenesis and systems for regulated expression using degradation domains that can be stabilized using molecular ligands.

"In the traditions of the work of Alice and C.C., Dan is a true biochemist at heart who also fearlessly takes on the techniques and problems of related disciplines such as cell biology, molecular biology, genomics or chemical biology, as long as it helps his team solve critical biological questions of parasitology," said Pradipsinh Rathod, professor of chemistry at the University of Washington in Seattle. "Even though Dan has worked on medically important parasites such as *Plasmodium* and *Ascaris*, he has always been more interested in the functioning of the biology of these parasites rather than a pure obsession with developing therapeutics."

Goldberg, who graduated from Harvard University in 1978 and obtained his M.D. and Ph.D. from WUSTL in 1985, has been a faculty member at WUSTL since 1990



"What an honor to receive this award named for the grandparents of molecular parasitology, C.C. and Alice! I like to think in terms of the pre-Wang era, when gentle scientists would collect specimens between gin and tonics and take them back to the lab for a few measurements, contrasted with the Wang era, where scientists use cutting-edge technology to address sophisticated biological questions about these amazing and nefarious creatures."

– DANIEL GOLDBERG

2013 ASBMB YOUNG INVESTIGATOR AWARD

Shan honored for work uncovering mechanisms of signal recognition particle

BY DINU-VALENTIN BĂLĂNESCU

The American Society for Biochemistry and Molecular Biology has named Shu-ou Shan of the California Institute of Technology the winner of the society's Young Investigator Award for 2013.

Shan has led extensive research concerning the specific protein-targeting mechanisms of the signal recognition particle, or SRP, the protein–RNA complex responsible for guiding polypeptide chains from the ribosome to the eukaryotic endoplasmic reticulum or the bacterial plasma membrane during protein biosynthesis. Her quantitative dissection of the kinetics of this complex mechanism established that the overall fidelity of the targeting process — the ability to make sure the right polypeptide is delivered to the proper cellular location — is achieved through the cumulative effects of discrimination at multiple kinetic steps, rather than only at the initial step as had been believed widely.

Shan's research is of clinical significance, as it offers a better understanding of diseases that result from a defective pathway of protein delivery to the endoplasmic reticulum. Furthermore, it could aid in the development of new antibiotics that target bacterial SRP, thereby obtaining a bactericidal effect and an alternative for treating drug-resistant bacteria. Most fundamentally, it provides an elegant example of and roadmap for the dissection of complex biological processes, the need for which will continue to grow as we learn more about biological pathways and the control of and connections between these pathways.

Upon obtaining her Ph.D. at Stanford University in 2000, Shan joined the University of California, San Francisco, for postdoctoral research. It is there that she began her work on the SRP. She joined the California Institute of Technology in 2005.

Caltech colleague Douglas Rees describes Shan as "an exceptional scientist, dedicated mentor and a wonderful colleague."

In his nomination of Shan for the award, Rees lauded

and a Howard Hughes Medical Institute investigator since 1994.

"Dan always has new discoveries in the molecular biology of *Plasmodium*, the most recent of which is the finding of the protease that cleaves proteins destined for the red cell cytoplasm," said Louis H. Miller, head of the malaria cell biology section at the National Institute for Allergy and Infectious Diseases. "He is in the tradition of Alice and C.C. Wang, who created the field of molecular parasitology."

Goldberg will receive his award during the Experimental Biology 2013 conference in Boston, where he will deliver an award lecture. The presentation will take place at 12:30 p.m. April 22 in the Boston Convention and Exhibition Center.



Natalie Osayande (natalie.osayande@spartans.ut.edu) is an undergraduate at the University of Tampa studying biochemistry.



Shan's "ambitious and innovative research program and her uncanny ability to take a system from a cartoon level of understanding and placing it on a rigorous and quantitative mechanistic foundation."

Rees continued: "Shu-ou's impressive mentoring qualities clearly reflect the influence of her graduate adviser, Dr. Daniel Herschlag, who was recognized for his training of younger scientists by receiving the 2010 ASBMB William C. Rose Award."

Shan got off to a quick start in graduate school, publishing three papers in her second year, all in prestigious journals, said Herschlag, now at the Stanford University Medical Center, who described Shan as "a very special and extremely creative scientist."

"It has been remarkable to watch Shu-ou's career develop as a postdoc with Peter Walter, where her understanding of biology grew immensely, and then as head of her own lab, where she has found and pursued important biological problems and have, in absolutely beautiful studies, applied rigorous kinetics and thermodynamics to

reveal elegant mechanisms underlying the biology."

Shan will receive her award during the Experimental Biology 2013 conference in Boston, where she will deliver an award lecture. The presentation will take place at 8:30 a.m. April 22 in the Boston Convention and Exhibition Center.



Dinu-Valentin Bălănescu (dinu.balanescu@yahoo.com) is a medical student at the Carol Davila University of Medicine and Pharmacy in Bucharest, Romania.

AWARD FOR EXEMPLARY CONTRIBUTIONS TO EDUCATION

Ntambi honored for unique contributions to education

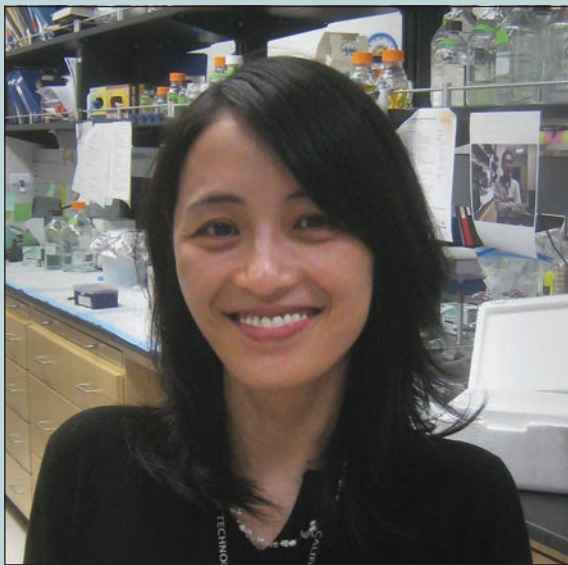
BY NATALIE OSAYANDE

James Mukasa Ntambi, professor of biochemistry and of nutritional sciences at the University of Wisconsin-Madison, is the winner of the American Society for Biochemistry and Molecular Biology Award for Exemplary Contributions to Education.

Ntambi, a native of Uganda, received his bachelor's and master's degrees from Makerere University Kampala and then his Ph.D. from John Hopkins University in 1985. After a postdoctoral fellowship at Hopkins and after working briefly at Georgetown University, he joined the departments of biochemistry and nutritional sciences at the University of Wisconsin-Madison, where he has taught and conducted research for 20 years.

In addition to teaching the metabolism section of the large undergraduate course in general biochemistry, Ntambi teaches a unique Uganda study abroad course called International Health and Nutrition. "The central concept of this course is that UW-Madison undergraduates apply the biochemistry they learn in the classroom to real-world problems," Ntambi explained.

Ntambi and his colleagues first discuss with the students the biochemical basis of nutrition, as well as agriculture, culture, economics, education and public health issues particular to Uganda. This is followed by a three-week trip to Uganda, where they work in villages and see nutritional, environmental and public-health problems



"It is a great honor to receive the ASBMB Young Investigator Award. I stand on the shoulders of many who came before me — great cell biologists like Peter Walter, who discovered this fundamental cellular pathway, and enzymologists like Daniel Herschlag, who defined rigorous conceptual frameworks for understanding biomolecular action. It is truly an honor to be mentored by these great scientists and hence have the opportunity to combine these disciplines."

— SHU-OU SHAN



"It is an immense honor for me to accept this award and to be held in the highest regard as a scientist and teacher. I continue to believe that we should put into practice what we teach and learn both in and out of the classroom – an experience that brings new meaning to the education of all humans."

– JAMES MUKASA NTAMBI

firsthand. They visit rural health centers, HIV/AIDS clinics, child-nutrition centers, agricultural research stations, local markets and rural schools to learn about real-life experiences in the communities.

Through Ntambi's collaboration, there has been exchange in the other direction as well: Makerere University faculty members and students have visited Madison, and some have conducted research in Ntambi's lab. These education and research experiences have helped students and faculty members of the two institutions build international networks and discuss global public-health issues, Ntambi said.

"James' Uganda program has been a tremendous success, with valuable consequences for UW students long after their Ugandan experience," said Ormond MacDougald, director of graduate studies at UWM, in his nomination of Ntambi. "By exposing students to critical health issues, students are provided with opportunities to learn more about themselves as future health care professionals."

Meanwhile, Ntambi's research program in Madison focuses on the biological roles of stearyl CoA desaturase enzymes. Ntambi's pioneering work on the genetic regula-

tion of SCD has led to many new insights into the importance of these enzymes in metabolism and in disease states such as obesity, diabetes, atherosclerosis, inflammation and cancer.

The Ntambi research program has trained 16 Ph.D. students, 15 postdoctoral fellows, more than 30 undergraduate students and several high-school students.

Lacmbouh Ghislain Ade, one of Ntambi's former interns, stated in his nomination letter, "After working with Dr. Ntambi only a couple of days, I still can remember walking into his office and asking him, 'Sir, how can I become like you?' Dr. Ntambi is all you expect of a mentor, and his motivation and dedication to seeing his students succeed is really phenomenal."

Ntambi will receive his award during the Experimental Biology 2013 conference in Boston, where he will deliver an award lecture. The presentation will take place at 12:30 p.m. April 21 in the Boston Convention and Exhibition Center.



Natalie Osayande (natalie.osayande@spartans.ut.edu) is an undergraduate at the University of Tampa studying biochemistry.

MILDRED COHN AWARD IN BIOLOGICAL CHEMISTRY

Doudna wins new Mildred Cohn award

BY MAGGIE WEAR

Jennifer A. Doudna is the inaugural recipient of the American Society for Biochemistry and Molecular Biology Mildred Cohn award in Biological Chemistry. Doudna has been described by those who nominated her for the award as "an outstanding scientist," "a unique scholar," "an exceptionally creative and productive scientist," "a great speaker" and "an accomplished educator."

Beyond the superlatives, Doudna's scientific career speaks for itself. Jack Szostak, her thesis adviser at Harvard University, remarks, "Her thesis work spanned synthetic chemistry, molecular biology and genetics, and was published in 10 papers, including three in *Science* and *Nature*."

Although "conventional wisdom had concluded that large, biologically interesting RNAs were unsuitable for X-ray crystallography," Thomas Cech, Doudna's postdoc-



toral adviser, explains, she “solved the first structure of a large domain of the Tetrahymena ribozyme.”

Building a research program upon this and other structures, which clearly showed how RNA can act catalytically, Doudna moved in 1999 to Yale University, where she continued her crystallography work. “Her time at Yale was intensely productive, with previously recalcitrant RNAs falling like dominoes before the increasingly successful Doudna lab,” Szostak remarks.

Thomas Steitz of Yale University observes, “Her structure of the P4-P6 RNA fragment of the group I intron was the first to show that RNA can have a tightly packed globular fold, not unlike proteins.”

In 2002, Doudna moved to the University of California, Berkeley. There, her lab solved the structure of the siRNA Dicer enzyme from *Giardia*, elucidating its function as a molecular ruler. Today her team is investigating the machinery of the RNA-mediated acquired immune system in bacteria. As Dinshaw Patel of Memorial Sloan-Kettering Cancer Center notes, this latest endeavor represents a “tour-de-force set of structural and biochemical studies.”



“I am thrilled and honored to receive this award that in turn honors the great scientist Mildred Cohn. Dr. Cohn’s outstanding legacy epitomizes the values I hold most dear in science and that I seek to communicate to students: scholarship, passion for discovery and a continuing sense of wonder about the natural world. I want to express my ongoing gratitude to my colleagues and my students for making the journey so much fun.”

– JENNIFER A. DOUDNA

Joan Steitz, in her nomination of Doudna for this award, commented that “just as three-dimensional structures transformed our understanding of proteins, Dr. Doudna’s high-resolution X-ray analyses of large RNAs have had momentous impact.” Doudna, she continues, “is not merely a crystallographer but a consummate molecular scientist who can apply whatever physical or biochemical approach is needed to solve an important problem.”

Szostak concurs, stating that Doudna “has sought out whatever techniques are needed to solve interesting and important but difficult problems.”

Doudna has been the recipient of many prestigious awards, including the Beckman Young Investigator Award in 1996, the National Academy of Sciences Award of Initiatives in Research in 1999 and the Eli Lilly Award in Biological Chemistry in 2001. She was inducted into the National Academy of Sciences in 2002 and has been a Howard Hughes Medical Institute investigator since 2002.

Beyond her impressive scientific achievements, Doudna is known as an exceedingly excellent educator. Szostak observes, “She instills her trainees with the confidence to tackle important problems while conveying to them the knowledge and experience needed to do so successfully.”

Michael Marletta of The Scripps Research Institute said of Doudna’s 2006 Science paper revealing that the micro-RNA processing enzyme Dicer acts as a molecular ruler, “Mildred Cohn would be smiling at these results and the way they were obtained.”

Doudna will receive her award during the Experimental Biology 2013 conference in Boston, where she will deliver an award lecture. The presentation will take place at 9 a.m. April 24 in the Boston Convention and Exhibition Center.

About Mildred Cohn

Throughout her career, from graduating high school at the age of 14 to becoming the first female appointed to the editorial board of the *Journal of Biological Chemistry* and the first female president of what was then the American Society of Biological Chemists, Mildred Cohn was a pioneer and inspiration for female scientists throughout the world. Cohn’s endeavor to understand both enzymatic mechanisms and cellular metabolism resulted in breakthroughs in the development of nuclear magnetic resonance methods that changed the way we study enzymatic reactions.



Maggie Wear (Margaret.Wear@usuhs.edu) is a Ph.D. student in the molecular and cellular biology program at the Uniformed Services University of the Health Sciences in Bethesda, Md.

WALTER A. SHAW YOUNG
INVESTIGATOR AWARD
IN LIPID RESEARCH

Walther lauded as 'hands down the most talented young investigator in lipid biology'

BY DINU-VALENTIN BĂLĂNESCU

Tobias Walther, associate professor at the Yale School of Medicine, is the winner of the American Society of Biochemistry and Molecular Biology's 2013 Walter A. Shaw Young Investigator Award in Lipid Research.

Throughout his career, Walther has worked on numerous cell biology problems, but a main focus is on cellular lipid droplets. His work on the subject has helped to propel lipid droplets into an important interest in cell biology with vast implications in multiple fields.

Together with his scientific partner, Robert Farese Jr. at the Gladstone Institutes and the University of California, San Francisco, Walther's laboratory has been pioneering the domain. The team used a combination of systematic approaches, including RNAi screening, quantitative proteomics and cutting-edge microscopy to work out many mechanisms of LD formation and growth. One of his most recent achievements involved the use of proteomics data leading to the discovery of a feedback system that regulates phosphatidylcholine synthesis and LD growth.

Walther published his first paper as an exchange undergraduate student at the Southern Methodist University in Dallas. He obtained his Ph.D. in 2002 at the Ludwig-Maximilians University of Munich, working at the European Molecular Biology Laboratory, where he identified factors of nuclear pores assembly. He then joined the University of California at San Francisco for his postdoctoral training, during which time he studied plasma membrane organization and began his work on lipid droplets. From 2006 until 2010, he was group leader at the Max Planck Institute of Biochemistry in Martinsried, Germany.

Peter Espenshade, winner of the 2012 award, said in his nomination letter of Walther that in addition to Walther's scientific contributions, "he has an amazing ability to effectively collaborate with others." Farese said of Walther, "Put simply, I think he is an immensely talented scientist



"I feel very honored and humbled by ASBMB rewarding our work on lipid homeostasis and storage with the Walter A. Shaw award. It is great to follow in the tradition of many excellent scientists, and I look forward to the discoveries to come."

– TOBIAS WALTHER

and hands down the most talented young investigator in lipid biology."

Walther will receive his award during the Experimental Biology 2013 conference in Boston, where he will deliver an award lecture. The presentation will take place at 3:45 p.m. April 23 in the Boston Convention and Exhibition Center.



Dinu-Valentin Bălănescu (dinu.balanescu@yahoo.com) is a medical student at the Carol Davila University of Medicine and Pharmacy in Bucharest, Romania.

**Now accepting nominations
for the 2014 awards!**

Deadline: June 3
Visit: www.asbmb.org/awards/2014/

DELANO AWARD FOR COMPUTATIONAL BIOSCIENCES

Berman recognized for her efforts in removing barriers to data access

BY MANASA CHANDRA

Helen Berman has won the 2013 DeLano Award for Computational Biosciences from the American Society for Biochemistry and Molecular Biology. Berman, a professor at Rutgers University and co-founder of the Protein Data Bank, is being honored for her efforts to make data universally available.

"From the earliest point in her career, she worked within the scientific community to set up guidelines to ensure that such universal access would be possible," explained Ann Stock of Rutgers University in her nomination of Berman. She continued: "Motivating these efforts was her strong conviction that it would be possible to create new knowledge from systematic analyses of data. While this concept seems obvious today, it was visionary when she began to promote the idea in the early 1970s."

About 25 new structures now are submitted to the PDB each day, and more than 88,000 structures have been made available since it was established in 1971. (Berman, who today directs the Research Collaboratory for Structural Bioinformatics PDB, has contributed 38 of those structures.) Berman also was involved in creating the Nucleic Acid Database of structures. Her contributions to the PDB and databases of the sort have had a profound impact on how we study biology.

Jane Richardson wrote in support of Berman's nomination for the award, "I think it is incontestable that disappearance of the PDB would leave a larger hole in macromolecular science than the loss of any other single computer-based system and also that Helen is the person who deserves the most credit for that."

Berman began studying crystallography at the Columbia University College of Physicians and Surgeons during her undergraduate years at Barnard College. Her interest followed her to the University of Pittsburgh, where she worked under the direction of George Alan Jeffrey and received, in a mere three years, her Ph.D. in crystallography in 1967. She stayed on at Pittsburgh for a postdoctoral stint and later moved to the Fox Chase Cancer

Center, where she started her own lab and focused for 20 years on nucleic acid crystallography and the interactions between nucleic acids and drugs. In 1989, she joined the faculty at Rutgers and went on to expand her crystallographic program to include the study of collagen and protein-nucleic acid complexes. Berman became director of the PDB in 1998 and in 2003 co-founded the Worldwide PDB organization that now manages the PDB archive.

Education is another facet of Berman's extraordinary career: She has mentored numerous high-school, undergraduate and graduate students and postdoctoral fellows. Janet Thornton at The European Bioinformatics Institute explained, "Obviously, as a professor, she has been involved in education, but her commitment goes well beyond the normal devotion to duty. Within the PDB she has always devoted considerable energy and funds to ensuring that the PDB is accessible to school children and young undergraduates. Her work has made these complex structures much more accessible to these young people, the scientists of the future."

Berman will receive her award during the Experimental Biology 2013 conference in Boston, where she will deliver an award lecture. The presentation will take place at 2:55 p.m. April 22 in the Boston Convention and Exhibition Center.

Manasa Chandra (mchandra@coh.org) is a Ph.D. candidate focusing on gene therapy at the Irell and Manella Graduate School of Biological Sciences at City of Hope Medical Center in Duarte, Calif.



"I am deeply honored to be a recipient of the DeLano award that recognizes the importance of the availability of computational resources for biological research."

– HELEN BERMAN

ASBMB

ANNUAL MEETING

BOSTON, April 20–24, 2013

WORKSHOPS

Computational Tools for Assigning Enzymatic Functions Workshop: Examples from the Glutathione Transferase Superfamily

This workshop is sponsored by the Enzyme Function Initiative (EFI), a large-scale collaborative project from the National Institute for General Medical Sciences.

12:30 p.m. to 2:30 p.m. Sunday, April 21

Co-organizers: John A. Gerlt, University of Illinois
and Patricia C. Babbitt, University of California at San Francisco

Proteomics of Post-translational Modifications Workshop

12:30 p.m. to 2 p.m. Tuesday, April 23

Session chairman: Steven P. Gygi, Harvard Medical School

Presentations followed by a panel discussion and questions from audience.

SPECIAL EVENTS

Professional Development for Graduate Students and Postdoctoral Trainees

Saturday, April 20, Room 253C

** Advance registration required*

Opening Reception

Saturday, April 20, Ballroom West, after the opening lecture

Kick start your meeting experience enjoying refreshments while connecting with friends and fellow attendees.

A Science Revolution: Undergraduate Uprisings



The Biochemists are Coming! Orientation for Undergraduates

11:30 a.m. to noon Saturday, April 20, Room 50



Intercontinental Congress: The 17th Annual Undergraduate Research Poster Competition

1 p.m. to 4:30 pm Saturday, April 20, East Registration Area

** Advance registration required*



Declaration of Independence: Undergraduate Workshop

Beyond College: Coping with Some Common Challenges

4:45 p.m. to 5:45 p.m. Saturday, April 20, Room 50



Boston Tea Parties: Undergraduate Breakfasts with Award-Winning Scientists

7 a.m. to 8 a.m. Sunday, April 21, and Monday, April 22, Room 255

** Advance registration required*



For details, visit **WWW.ASBMB.ORG**

Fostering Partnerships among Colleges: University and K - 12 Workshop

9 a.m. to 1 p.m. Saturday, April 20, Room 251

** Advance registration required*

How Scientists Can Save the World

Sponsored by the ASBMB Public Affairs Advisory Committee

12:45 p.m. to 2:30 p.m. Sunday, April 20, Room 258

Join the leading names in science and public policy as they discuss the massive challenges facing society in the next 100 years. Learn how science holds many of the solutions to problems like hunger, health, and sustainability -- and how to be an advocate for science.

ASBMB Welcome and Networking Reception

Sponsored by the ASBMB Minority Affairs Committee

6:30 p.m. to 8:30 p.m. Sunday, April 21,

Commonwealth Ballroom at the Westin Boston Waterfront Hotel

Open Meeting on ASBMB Certification Program for Bachelor's Degrees

12:30 p.m. to 2 p.m. Monday, April 22, Room 251

From the Lab to the Kitchen Table: Communicating Science to a Lay Audience

Monday, April 22, 12:30 pm - 2:30 pm, Room 255

This interactive program features successful outreach activities and teaches attendees techniques for effectively communicating their science to the lay public

ASBMB Thematic Fermentation Happy Hour

6 p.m. Monday, April 22, outside the ASBMB sessions in the Northeast Foyer

Relax at this casual, post-session happy hour and continue the scientific discussion, meet the speakers and network.

ASBMB Science Cafe: The New Social Networking Event

7 p.m. Monday, April 22, Sauciety Restaurant at the Westin Boston Waterfront Hotel

Y.E.S. Mixer (Young Experimental Scientists)

9 p.m. to 11 p.m. Monday, April 22, Galleria at the Westin Boston Waterfront Hotel

Science Cafes 101: An Interactive Guide to Organizing Your Own Science Cafe

12:30 p.m. to 2:30 p.m. Tuesday, April 23, Room 255

ASBMB Women Scientists Networking Event

6 p.m. to 8 p.m. Tuesday, April 23, Room 257B

Lorsch to become next NIGMS director

Jon Lorsch, a professor at Johns Hopkins University, will be the next director of the National Institute of General Medical Sciences. He'll arrive at the institute in Bethesda, Md., this summer.



Lorsch, a member of the American Society for Biochemistry and Molecular Biology's mentoring committee, will oversee a \$2.4 billion budget that supports primarily fundamental research and scientific workforce training.

"With his reputation of being a broad-minded and visionary thinker with strong management skills, I am confident that Jon will lead NIH's basic science flagship to keep the U.S. at the forefront of biomedical research," Francis S. Collins, director of the National Institutes of Health, said in a statement announcing the appointment on March 25.

Lorsch will take the NIGMS reins from Judith H. Greenberg. Greenberg has served as the acting director of the institute since July 2011, when Jeremy Berg stepped down, after holding the director position for eight years, to become the University of Pittsburgh's associate senior vice-chancellor of science strategy and planning.

Berg, who now is also president of the ASBMB, said he was pleased with the appointment: "Jon is a great choice. He is an outstanding scientist with ideas spanning many disciplines and with great teaching and training experience. He also led the curriculum reform efforts at Johns Hopkins and balanced clinical and basic perspectives very well."

Berg continued: "He is very personable and is a good listener but is not at all afraid of tough issues. Jon is one of a small group of people whom I frequently reached out to when I was NIGMS director for his perspectives and advice. NIGMS will be in good hands."

JBC/Herb Tabor Young Investigator Award winners

BY KYEORDA KEMP



James Duce of the University of Leeds

James Duce, a senior research scientist and group leader at the University of Leeds, received the Journal of Biological Chemistry/Herbert Tabor Young Investigator Award in November at the 2012 Neurodegeneration Conference in Xcaret, Mexico, for his work on iron homeostasis in neurodegenerative diseases.

Duce played a pivotal role in identifying β -amyloid precursor protein, or APP, as a ferroxidase, and its ability to regulate neuron iron efflux. This also requires tau's assistance in its trafficking to the neuron surface.

Abnormal accumulation of APP and tau protein is found in the brains of patients with neurodegenerative diseases such as Alzheimer's, and Duce contends that studying these proteins "may provide pivotal information in discovering why iron-associated oxidative stress is of



such prevalence in a variety of these pathologies.” In the future, he said, he hopes to “establish an underlying mechanism of iron dysregulation in many degenerative diseases that is able to unify a number of the proteins typically associated with pathology and to provide potential sites of action for therapeutic intervention.”



Xu Ding of UCSD

Xu Ding, a project scientist at the University of California, San Diego, was awarded the Journal of Biological Chemistry/Herb Tabor Young Investigator Award in November at the 2012 joint meeting of the American Society for Matrix Biology and the Society for Glycobiology in San Diego for his work with heparan sulfate. Ding first began studying heparan sulfate in graduate school and joined the laboratory of Jeffrey Esko at UCSD as a postdoctoral fellow to further explore the role of heparan sulfate in vascular biology and inflammation.

Recently, he discovered that vascular endothelial heparan sulfate is required for RAGE, which is short for receptor for advanced glycation end products, to signal. RAGE must oligomerize to signal, and heparan sulfate plays a crucial role in this. Furthermore, he and his colleagues have determined the structures of the RAGE/heparan sulfate complexes, leading to the development of antibodies that disrupt RAGE oligomerization and signaling. This is of interest due to the major role that RAGE plays in diseases such as diabetes, atherosclerosis and cancer.

Alexandra Naba of MIT

Alexandra Naba, a postdoctoral fellow at the Massachusetts Institute of Technology, was awarded the Journal of Biological Chemistry/Herb Tabor Young Investigator Award in November for her work on the characterization of the composition of the extracellular matrix at the 2012 joint meeting of the American Society for Matrix Biology and the Society for Glycobiology in San Diego.

Naba first became interested in ECM signaling during the pursuit of her Ph.D., and upon completion she joined Richard Hyne's Lab at MIT to “further explore how extracellular cues control normal and tumor epithelial cell morphogenesis and behavior.” She has played a lead role in the Matrisome Project, which aims to uncover “the complexity of extracellular matrices in tissues and tumors,” she explained, and she has worked with the Proteomics Platform of the Broad Institute to develop protocols and procedures to isolate and analyze ECM proteins from tumor and normal tissues. They are now in the process of analyzing patient samples to discover ways in which the ECM could influence cancer progression.

Naba adds, “We also hope that this approach will allow us to identify biomarkers that could serve as prognostic markers and diagnostic tools and possible novel therapeutic targets for cancer patients.”



Kyeorda Kemp (kyeordakemp2010@u.northwestern.edu) is a postdoctoral researcher at Northwestern University.

I AM A PH.D. STUDENT, AND I'M A SURVIVOR

BY ADITI S. IYENGAR

Why would anyone want to be withdrawn from the real world, overworked, underpaid, chronically stressed and painfully uncertain about his or her life? That, in a nutshell, is the life of a graduate student. So why am I doing this? Believe me, I have had I-just-made-a-huge-mistake moments, and I often ask myself why I didn't simply get a job. But looking back, I realize that though this has been a singularly difficult path for me, there's nothing else I would be better at. I am now in the final year of my Ph.D., and sitting in my lab, having spent all day troubleshooting, I reminisce about the past few years and chronicle the experiences that almost beat me down – but not quite.

YEAR 1

I felt like Bambi. A shy international student taking my first steps into the esoteric world of research, I was green but willing to learn, intimidated but highly motivated. Also, I had chosen to study in New Orleans – the Crescent city, the jazz capital and the land of Mardi Gras. This was perfect. I was going to work hard but party harder. I was going to be a rock-star scientist.

TWO MONTHS INTO YEAR 1

My father passed away. I don't remember much of rushing back to India, dealing with family and friends. Sometimes I wonder how I got through that particularly tough time. It was a nasty jolt. I consid-

ered quitting my program and remaining in India, partly to be there for my family but also because I didn't think I had it in me to go back and do justice to what I had set out to do. I did not want to deal with being alone and isolated. My mother, on the other hand, was made of sterner stuff. Family drama ensued, and after a barrage of tears and lamentations, my mother said, "Do not give up. Finish what you started. Make your dad proud." And so I found myself back in school two weeks after the funeral. I got busy with classes, learning how to culture cells without massive contamination and making reagents without Wikipedia's help. Losing myself to the mayhem of academic science rescued my sanity and saved my soul. Polymerase chain reaction consumed my life, pain passed and I healed.

YEAR 2

The second year of graduate school is, by far, the best time a student has in his or her graduate career. Classes are dwindling down, and you have had the what-is-my-project-I-can't-wait-to-get-started-I-might-cure-cancer conversation with your mentor. Your weekends are still relatively free, and you might still have a social life. And if your experiments don't work, you smile and reassure yourself that you have three more years to figure all this out. Or so I thought.

YEAR 3

Science just got real. After whiling away my previous year testing protocols and attending seminars for free lunches, I realized that I needed to get with the program. In my zest to prove my scientific worth, I

decided to start a project from scratch in an area never explored by my lab. I was going to take our research in another direction. I was going to conduct fantastic experiments and obtain jaw-dropping data. I was going to be the best thing that happened to my field.

Three months later, I found myself in my mentor's office, weeping into a Kleenex. Nothing was working. I am not good enough. I will disappoint the lab. I'm stupid. After allowing 30 minutes of self-loathing, my mentor said to me something that later became my mantra: "This is biological research. Ninety-five percent of the next few years will be a failure; the remaining 5 will be serendipity, luck and chance. The pursuit of that 5 percent is what will get you through graduate school. I know you can do this. I believe in you."

I am eternally grateful to my mentor for putting up with my hysteria. He is one of the reasons I didn't take the next flight back to India.

YEAR 4

The year of restrained frustration. Put plainly, a previous student messed up. We got stuck cleaning up the mess. This was a massive setback. All my data might have been wrong, and I had to start all over again. As I felt the months inch by, a panic settled in the pit of my stomach and refused to leave. When will I ever stop troubleshooting? When will I actually do real work? Why am I even here? As I questioned the meaning of life, the universe and everything over cups of cheap wine shared with my immensely more sensible roommate, I started to grow up. I was waking up from the slumber of muted complacency.

I learned to prioritize, to focus. I learned that whining wasn't getting me anywhere. And so I cleaned up the mess and lost valuable time, but I'm a better scientist for it.

YEAR 5

Final year! It's been quite a journey. I started graduate school thinking I knew what I wanted; I walk away now acutely aware of what I do not want. Cloning taught me self-discipline, Western blotting taught me creativity, and tissue culture taught me patience. More importantly, I learned that this is not just my story. So many graduate students go through more and worse. I have met students who were set back by many years because of Hurricane Katrina and the destruction it left behind. I am acquainted with people who suffer at the hands of mean bosses and obnoxious lab mates. A lot of these people made it through with flying colors, owing to their implicit desire to succeed and further their research. So if you think you got a raw hand, do not be discouraged. To quote my favorite fortune cookie: "Every wrong attempt discarded is a step forward." Do not despair! If you love what you do, you'll find a way.



Aditi S. Iyengar was born and raised in India and earned a bachelor's degree in zoology from Ethiraj College. She arrived in New Orleans in 2007 to pursue at Tulane University a master's in neuroscience, upon completion of which she joined the genetics department at Louisiana State University Health Sciences Center in New Orleans. She now works in the lab of Andrew Hollenbach, and her project includes determining the biological activity of the transcription factor Pax3 and its role in muscle and skin cancer.

The quiet creep of
Alzheimer's
Disease

As caregivers grapple with the grim changes in their loved ones, researchers race to stall this neurodegenerative disorder.

BY RAJENDRANI MUKHOPADHYAY

One day in 2001, Jodi Bottoni came home to find her clothes, shoes, money and small purple beaded lamp on the driveway. Her mother had thrown them out the front door of their home in Venice, Fla. Bottoni, who in her 40s had moved back in with her mother to care for her, was baffled. She assumed her mother, Jacquie Berg, was becoming an alcoholic and blamed scotch for the irrational behavior. But over the course of the next four years, Bottoni realized her mother's increasingly erratic behavior, such as hoarding toilet paper in every nook and cranny of the house, couldn't be attributed solely to alcohol. Something else was wrong. In 2005, Berg was diagnosed with Alzheimer's disease.

Today, Bottoni is one of 15 million Americans who are caring for an Alzheimer's patient at home. As Gregory Petsko of Brandeis University and Weill Cornell Medical College stated in his 2012 TEDMed lecture, caregivers are "the hidden victims" of the disease.

"I've put on tons of weight. I get no sleep. I'm stressed out," says 54-year-old Bottoni, a bartender-waitress recovering from carpal tunnel surgery. "I have no life anymore. I don't get to go anywhere. I don't get to do anything. It's terrible."

Bottoni's now 84-year-old mother, a former acrobat and Arthur Murray dance teacher whose features remind her daughter of the late actress Elizabeth Taylor, talks to her reflection in mirrors, incessantly hums and whistles, hides food in potted plants, and scribbles in coloring books with crayons. Her daughter has child-locked all doors leading out of the house and put a tracking bracelet on her mother's ankle. During the night, Berg gets up four or five times to go to the bathroom. Bottoni's 14-year-old Labrador, Morgan, sleeps in her mother's room, and whenever the elderly woman rises out of bed, "he gets up and shakes his collar," says Bottoni. "He lets me know she's up and moving." If Bottoni doesn't catch her mother in time, she has to clean urine and feces from the floor, because her mother can no longer coordinate her movements and bodily functions.

So far, Bottoni has refused to put her mother, who adopted her as a child, in a nursing home. "I don't trust anyone else to take care of her," she says. "I love her to death. But I don't know if I can do this anymore."

The number of people in Bottoni's shoes will rise if the disease goes unchecked. The global population is aging, and people in their mid-60s begin to become more susceptible to Alzheimer's disease. By 2050, 100 million people around the

world are projected to be stricken with the disease and 300 million people will become their caregivers.

Despite the potentially large number of victims of this fatal disease, Petsko has said, "I hear no clamor. I see no sense of urgency. If you knew an asteroid was going to hit the earth and would kill 100 million people, you would probably expect a worldwide clamor that something be done about it."

The lack of public clamor, Petsko has argued, is at odds with the buzz of activity in Alzheimer's disease research. Scientists are learning more about the molecular details of the disease and are optimistic that they may have drugs within the next five to 10 years to defend against its assault. But lack of public awareness of the disease's impact means that there isn't a push for faster research progress. "We're grossly underspending on research for this field," says Norman Relkin of Weill Cornell Medical College, but "we're facing an epidemic of unprecedented magnitude."



Alzheimer's disease is defined by two histopathological hallmarks in the brain, amyloid plaques and neurofibrillary tangles. These were first described more than a century ago by the Bavarian psychiatrist and neuropathologist Aloisius "Alois" Alzheimer. In 1901, Alzheimer met a 51-year-old female patient, Auguste Deter, at the Frankfurt Asylum. Deter showed strange behaviors, including screaming for hours in the middle of night, and experienced hallucinations. She had been admitted into the institution because her husband, a railroad worker named Karl, could no longer take care of her. Alzheimer became obsessed with her symptoms over the next few years. When Deter died in April 1906, Alzheimer acquired her medical records and brain. He used silver staining techniques to study her brain and later that year described at a conference the plaques and tangles he saw as well as her symptoms.

Alzheimer's disease is mostly an age-related disorder, but "there's been a longstanding debate (about) whether Alzheimer's disease is an obligate consequence of aging," says Relkin. There is evidence against that notion. Rare early-onset forms of the disease, based on autosomal dominant mutations, have been characterized in individuals who, like Deter, begin to display dementia by their 50s. These early-onset forms, which bear clinical and pathological similarities to the late-onset form, underscore that Alzheimer's is a bona fide disease and not a

foregone conclusion of aging.

The plaques, found outside of neurons, are now known to be clumps largely of a protein fragment called beta-amyloid or amyloid-beta. A β is a product of the proteolysis of a larger transmembrane protein called amyloid precursor protein. There are more than 30 types of A β fragments that differ in length, but the one that is found predominantly in plaques has 42 amino acids, A β 42.

The neurofibrillary tangles are inside of neurons. They consist of a microtubule-associated protein called tau in a hyperphosphorylated form. When tau is not hyperphosphorylated, it stabilizes microtubules, which are important for maintaining cellular structure and acting as railroad tracks for the trafficking of various organelles. Tangles are seen in other types of dementia diseases, such as Pick's disease, but in those cases they are not accompanied by plaques.

"There is some early indication that the amyloid, whether it's aggregates or oligomers, triggers intracellular signaling pathways that involve tau and initiate the process of tangle formation," says Paul Fraser at the University of Toronto about Alzheimer's disease. "But the exact mechanism hasn't been completely figured out."

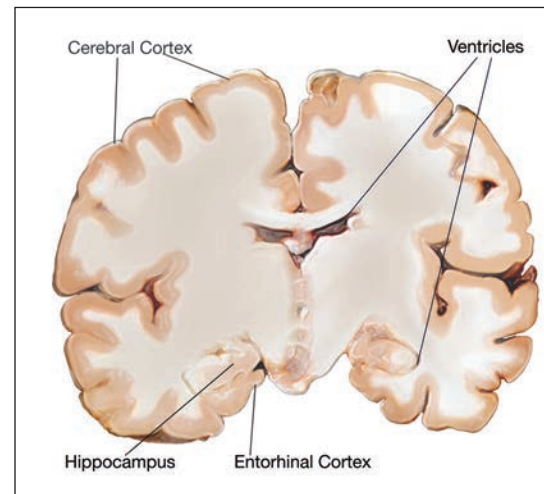


Eilene Wollslager recalls the moment when the enormity of her mother's Alzheimer's disease hit her. A professor of communications at the University of Texas, San Antonio, Wollslager had been watching her 85-year-old mother, Betty, lose her memory little by little over the course of seven years. In 2011, Wollslager had her parents move in with her and her husband so they could take over their care. But six months ago, when she walked into the kitchen, Wollslager was stunned. "My mom is a person who drinks 20 cups of coffee a day. She is a coffee addict. She was standing in front of the coffee maker and could not figure out how to make coffee," Wollslager recalls. "That really hit me – just how far down the road we had come."

Betty Wollslager was an elementary school teacher who taught third- and fourth-graders for 30 years. She was among the first generation in her family to go college. She was a voracious reader and was known as the fam-



Betty Wollslager PHOTO PROVIDED BY: EILENE WOLLSLAGER



Healthy

ily historian. But hints of the disease began to crop up in the mid-2000s. "She had been forgetful for a number of years. She wouldn't remember little things. She couldn't pull up a name or a word. She would go into rooms and forget why she was there," says Eilene Wollslager. "You thought, 'Oh well, she's getting older.' But it just kept going."

When Betty Wollslager became aware that she was becoming forgetful, she began to keep a nightly journal in which she would record the day's events. "At first, she could do it herself. Then she would have to ask Dad for help, because she couldn't remember what happened in the day. Then she had to ask Dad pretty much for everything. Now she doesn't do it," says Eilene Wollslager.

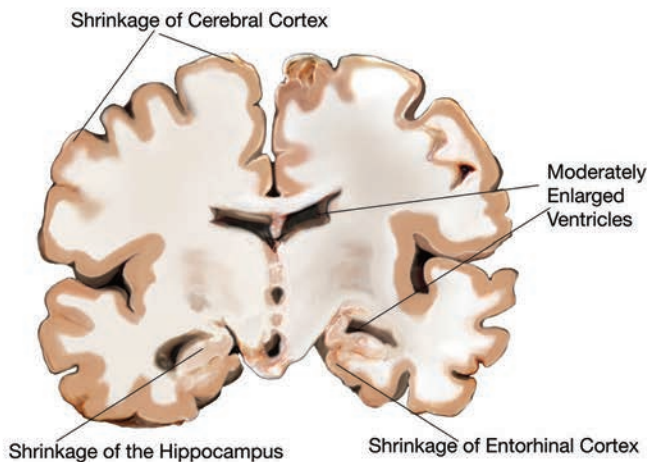
These days, Betty Wollslager recognizes her husband and daughter but doesn't remember her daughter's name. "She cannot tell you where she lives. She can't tell you what year it is and what day it is," says Eilene Wollslager. "She lives totally in the moment."



Alzheimer's disease takes over the brain in a specific pattern. "It's a \$64,000 question of why certain cells die and others don't," says Petsko. "There is no question that the disease is focal. It starts in specific regions of the brain and spreads."

The early stages of the disease rav-

PROGRESSION OF ALZHEIMER'S DISEASE



Moderate

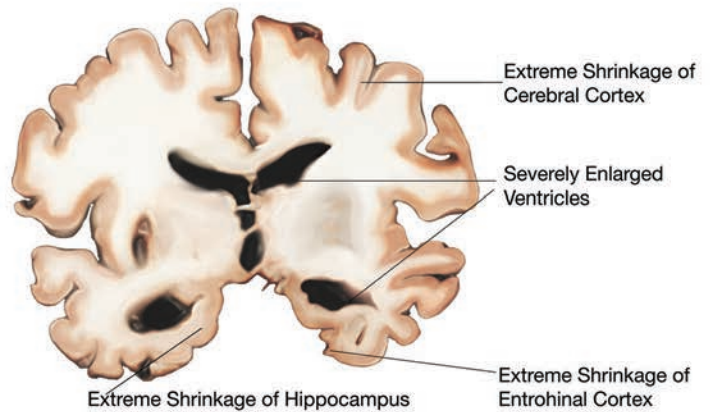
age the entorhinal cortex, a region near the hippocampus. From there, it spreads to other parts of the limbic system, including the hippocampus, which is involved in emotions, behavior and long-term memory. The disease then invades the neocortical areas, at which point patients may begin to show disturbances in verbal fluency, orientation and copying geometric figures.

Autopsies reveal that plaques and tangles show up at different times. Plaques seem to appear anywhere from 20 to 30 years before symptoms. Neurofibrillary tangles seem more closely timed with the onset of disease symptoms but still precede them.

The thinking now is that oligomers of A β and tau, precursors to the plaques and neurofibrillary tangles, trigger neuronal death. Recent evidence suggests that tau can pass from cell to cell in a prionlike fashion, causing its normal counterparts in other cells to misfold and spur cell death.

Larry Sparks of Banner Health Research says autopsies have shown that the brains of elderly people without Alzheimer's can have plaques but no neurofibrillary tangles. The question is why, in some people, these plaques, after a certain load and time, trigger formation of neurofibrillary tangles and cause them to spread through the brain, bringing on clinical symptoms of Alzheimer's.

The neuronal system isn't the only one involved in the disease. A β and tau accumulation are thought to set off reactions involving glucose metabolism and inflammation. There are also links between insulin resistance and Alzheimer's disease. Creighton Phelps at the National Institute of Aging explains that genetics has revealed hints of connections between the immune system and Alzheimer's disease. "It fits that you might inherit a group of genes that make you a little more vulnerable to these protein aggregations because of your immune status,"



Severe

IMAGE CREDIT: NATIONAL INSTITUTE ON AGING

he says.



Both Wollslager and Bottoni say it's so hard to get their mothers to bathe that they only attempt it once a week. "It's the weird things you battle over. My mom always was very fastidious. She always had every hair in place, crisp and clean," says Wollslager. "We have a fight now every week about trying to get her to bathe. She thinks she's already done it."

Bottoni describes showering her mother as hell. "I have the heater in the bathroom to warm it up. I get everything set up in her bedroom with the blow dryer and the chair. I put her bathrobe in the dryer so she will be nice and warm. And then it's a fight, tooth and nail." Her mother refuses to step into the bathtub and recently has taken to screaming curses at her daughter. "Once, she punched me in the stomach. She has a good wallop," says Bottoni with a rueful laugh. "Then another day, she got me in the jaw."



Researchers are certain that in Alzheimer's disease, the processing of APP goes awry. APP, which is about 700 amino acids long and implicated in neuronal guidance and positioning during brain development, gets cleaved at least twice by two different enzymes, the ubiquitous γ -secretase and the more neuron-specific β -secretase, which is also known as BACE. The cleavage produces the series of A β fragments, which include A β 42 and the normally more predominant A β 40. Scott Small of Columbia University says researchers are "legitimately embroiled in the active discussion of which of these fragments is pathogenic."

Experts agree that A β 42 is one culprit. The last two amino



PHOTO PROVIDED BY: BOTTONI

From left: Jodi Bottoni, Jacquie Berg and their friend KC Jones. Morgan, the dog, is behind the cactus next to Bottoni.

acids in A β 42 are alanine and isoleucine, hydrophobic amino acids that confer a strong aggregation property to A β 42 that A β 40 doesn't have. "If we only made A β 40, there probably wouldn't be such a thing as Alzheimer's disease," notes Dennis Selkoe of Harvard Medical School.

Researchers have focused on APP and its processing because of genetic evidence. For example, most people with Down's syndrome develop the signs of Alzheimer's disease if they live to be in their 50s. Those with Down's syndrome, like early-onset Alzheimer's patients, start accumulating plaques in their brains as teenagers or young adults and later develop neurofibrillary tangles. G. William Rebeck at Georgetown University Medical Center explains the gene for APP is on chromosome 21, the extra chromosome those with Down's syndrome carry. With more APP than normal, those with Down's syndrome, not all of whom develop full-blown dementia, may produce more A β fragments than people without the syndrome.

Then there is a mutation called A673T in APP, which is found in a small number of people in Iceland and other Scandinavian countries who never get Alzheimer's disease. The mutation happens at a location right next to where β -secretase cuts on APP. "They not only do not get Alzheimer's disease — they don't even show any mild cognitive impairment," says Petsko. "We now know from in vitro studies that mutation reduces the effectiveness of APP as a substrate for β -secretase. The end result is you make about 50 percent less A β than if you did not have the mutation."

There is a smaller group of a people with a different mutation at the same site where the residue is changed to a valine instead of threonine. That change makes APP more effective as a substrate for β -secretase, and these people get early-onset Alzheimer's disease. "That is, to me, the best smoking gun that says that the processing of APP is relevant to the development of Alzheimer's disease," says Petsko.

Playing into the processing of APP are mutations in the γ -secretase complex, which are also known as presenilins. These presenilin mutations increase the processing of APP to produce more A β fragments, particularly A β 42. For example, a clan of 5,000 people in Medellín, Colombia carries a genetic mutation in presenilins that predisposes them to early-onset Alzheimer's disease; they begin to show cognitive impairment around age 45 and by their 50s have full-blown dementia. Recently, the

medical records and samples of Deter, Alzheimer's first patient, were recovered; she was found to have a presenilin mutation.

But A β 42 and the other fragments are made throughout a person's life, starting from the fetal stage. A mystery in Alzheimer's disease is why all of a sudden these fragments turn rogue. Researchers suspect something goes awry in clearing out the A β 42 fragments, causing them to form oligomers that go on to form plaques. One indication that this may be the case comes from a polymorphism in a gene for apolipoprotein E.

ApoEs circulate in the blood and bind to the LDL-receptor-related protein, which is involved in cholesterol sequestration and modulation of membrane lipids in the central nervous system. ApoE4 reduces the removal of A β fragments from the extracellular space. People who have two copies of ApoE4 are 10 times more at risk of developing Alzheimer's than people who don't carry the polymorphism. In the U.S., Rebeck explains, 1 percent to 2 percent of the population has two copies of the ApoE4 polymorphism. "It's certainly not uncommon and has a strong effect on your risk of disease," he says.



By the fall of 2012, researchers involved in three large clinical trials of potential Alzheimer's drugs announced that the agents did not help patients. Bapineuzumab, known as bapi for short, is a monoclonal antibody that binds A β oligomers and is developed by Pfizer and Johnson & Johnson. The pharmaceuti-

cal company Elan generated the antibody and has a financial stake in the drug. Eli Lilly & Company is developing another antibody against A β called solanezumab, or sola for short, that mostly targets the monomeric form of the protein. Sola failed fully to stop the disease's progress in a recent clinical trial, but there were some signs it could slow cognitive decline in patients with mild Alzheimer's. The clinical trial failures brought on soul searching among researchers: Why did these promising drugs, which worked well in Alzheimer's mouse models, fail in humans?

"Unfortunately, the way any field in medicine and science works, often the tests that are being carried out are tests of a hypothesis that was posed many years before," says Relkin. He says the agents were premised on the original amyloid hypothesis. The hypothesis, put forth in the early 1990s, suggested that the amyloid plaques were the causative agents of Alzheimer's disease. This suggested that simply removing plaques from the brain would be sufficient to halt the disease.

However, researchers now think that the plaques are simply insoluble reservoirs of the more toxic, soluble oligomers of A β , a thinking sometimes called the modified amyloid hypothesis. There is little evidence so far to suggest that simply removing the A β plaques, which the antibody-based therapies do, makes Alzheimer's patients better.

But researchers have not lost hope in the drugs in development. "There are all kinds of indications that these should work," says Phelps. But "these clinical trials were studying the wrong stage of the disease."

So now sola, Roche's gantenerumab A β antibody and a β -secretase inhibitor made by Lilly are being tested on patients predisposed to early-onset Alzheimer's. Phelps explains that researchers at the National Institutes of Health in collaboration with researchers in the Dominantly Inherited Alzheimer's Network are working on prevention trials by studying adults who have a parent with a mutated gene known to cause dominantly inherited Alzheimer's disease. The NIH also is collaborating with Banner Alzheimer's Institute, University of Antioquia in Colombia and Genentech to study an experimental A β antibody treatment called crenezumab on 300 Colombians who are genetically predisposed to develop early-onset Alzheimer's disease. And, by the summer of this year, Relkin says a phase III study he has carried out with the support of the NIH and Baxter Healthcare will reveal whether an antibody preparation called IVIG harvested from the blood of healthy donors is effective in treating Alzheimer's patients.

One of the pressing needs in the field, say researchers, is to develop therapies targeted against tau, not just A β . "They are both attractive targets, but the pharmaceutical industry has to do much more with tau," says Selkoe. Because of some

evidence that tau can exit a neuron and enter others, there is a moment when tau, like A β , is extracellular and can be attacked more easily with a therapeutic molecule. "You can hit the disorder at three or four independent points along the cascade to try and slow down the disease," says Sparks. "Combination therapy is the future of treatment in Alzheimer's disease."

But Relkin, Petsko and others say researchers need to hurry. "Even in the best-case scenario, a newly developed prevention is going to take a decade or more to be approved and disseminated. By that time, we're going to be well into the baby-boomer era, and we're going to have a fairly unmanageable healthcare situation on our hands," says Relkin. "We need demonstrations of drugs that work. It doesn't matter what their mechanisms are. We shouldn't suffer from the hubris that we know what causes the disease, but rather we need to focus on what works."



Both Wollslager and Bottoni say there are moments of joy and pleasure in caring for their mothers. For Wollslager, it's her mother's wit. "The one thing that is still Mom is her sense of humor. She still teases Dad and says things that are deliberately funny," says Wollslager. "That makes it a little more bearable."

But not much else is left of the woman she knew. "I refer to 'Old Mom' and 'New Mom,'" says Wollslager. "My mom I knew growing up isn't there anymore. Now we have New Mom. I compartmentalize her previous self from her current self, because if I continually mourn what was lost, I can't enjoy her in the moment."

Bottoni is grateful that time and the disease haven't touched her mother's beauty. She takes great pleasure in dressing her mother in the clothes she wore as a dance instructor: pastel capris and jackets worn over colorful shirts; accessories on her long, silver-white hair; and lipstick in shades of pink. But Bottoni acknowledges the emotional and physical tolls her mother's care has taken on her. "Unfortunately I don't see an end to it until I put her in an Alzheimer's unit in a nursing home," says Bottoni.

While things are still relatively manageable, Wollslager says she holds onto the small, good moments with her mother. "There will come a day she won't remember how to swallow. You know that's down the road," says Wollslager. "I just try to embrace what we have today."



Rajendrani Mukhopadhyay (rmukhopadhyay@asmb.org) is the senior science writer and blogger for ASBMB. Follow her on Twitter at twitter.com/rajmukhop.

Q&A with Montana Professor of the Year Mike Morrow

BY PREETHI CHANDER



Mike Morrow, professor of biology at the University of Montana Western, was named the Carnegie Foundation 2012 Montana Professor of the Year. Sponsored every year by the Carnegie Foundation for the Advancement of Teaching and the Council for Advancement and Support of Education, this national award honors instructors for their contributions to undergraduate education. He is the fourth professor in a row from the university, located in Dillon, Mont., to win the award. Morrow was instrumental in revamping a predominantly health science program to one that now offers biomedical courses, including biochemistry and molecular biology, along with research experiences for undergraduates. Consequently, student enrollment has risen, new faculty members have been recruited, grant funding has increased, and students of this program have gone on to pursue careers in biomedical fields. In this interview with ASBMB Today, Morrow emphasizes the value of research in teaching. He says, in part, “There’s no better way to teach a biology undergrad student how to be a scientist than to give them research experience. You have to fail, try again, redo your approach, make your mistakes and learn from them.”



Amanda Kortum was a student in Mike Morrow's lab and graduated in 2011. She was the recipient of an American Society for Microbiology fellowship and is enrolled in the DVM/Ph.D. program at North Carolina State University.

What is the focus of your laboratory research?

My lab research focuses on the yeast *Candida albicans*, specifically the eukaryotic machinery involved in the early secretory pathway. There are multiple players that are responsible for translocation of proteins from the cytosol to the endoplasmic reticulum. Using *Saccharomyces cerevisiae*, the brewer's yeast, as a guide to know what players we should look for, we try to locate similar genes in *C. albicans*. Since most of these genes are essential for *C. albicans* survival, our approach has been to generate conditional knockouts for these genes. We then do in vitro assays to see if translocation into the ER still happens when there are reduced amounts of these *Candida* proteins.

What inspired you to teach at an undergrad institution rather than a research-focused university?

I was always drawn to teaching and found that I had an aptitude and could do it effectively; taking complex topics in biology and microbiology and being able to explain those at some level to undergraduate students is extremely appealing to me, and it is very rewarding to help them understand and go on and do great things themselves. So that certainly was part of the major driving force for me to become a teacher. As for the smaller school, I have always wanted that, because I really like to interact closely with my students, get to know them and

help them achieve their goals and be connected enough to feel success through their successes.

Why did you choose Montana?

I was always interested in a smaller school. Our school has about 1,400 students. I wanted a place that was focused on undergrad education. I was always interested in a four-year, baccalaureate-granting institution. My personal interest involves the outdoors, and we certainly have a lot of that. It was a perfect storm of opportunity for me in that there was potential to build a strong biomedical science program. All these factors solidified my decision.

What were the contributing factors to your success at Montana Western?

Most certainly one of the biggest factors has been the funding we have had through the IDeA Networks of Biomedical Research Excellence program of the National Institutes of Health. That helped us modernize equipment, expand course offerings, have up-to-date research experiences for our undergraduates in my research and also use these resources for our courses.

The willingness of the institution to expand in this direction, having a biomedical degree program, was also important.

Another factor certainly has to do with the block scheduling program that we use here. The students take one course at a time, allowing us to do a lot more lab activities as part of the course work. As laboratory scientists, we can do a lot more science – one aspect of the experiment today and then meet tomorrow to do the next part of the experiment. You do not have to wait a week for the next lab to come around to do the next step. It really opens up the doors to different things we can do as teachers.

We changed the curriculum from a health-science-based to a biomedical-focused one. The transition was pretty big. Prior to the establishment of this new degree, the institution didn't offer a pipeline that helped students get into professional health areas like medical school, Ph.D. programs or research careers. So the student population was always in this region, but they really didn't have any options at Montana Western. When we developed this program, a lot of the students started recognizing the things that they could do at UMW, and they started to come when the program was in place. Much like the

rest of the country, the student demand was there, and once we developed the infrastructure and options here, the students came.

Can you speak about the grants that helped you with the restructuring?

The INBRE grant helped us bring in the money to modernize the curriculum and scientific instruments to offer the research experiences to our students. It is a statewide grant designed to build research infrastructure and help increase students in the biomedical pipeline, especially in less populated states that haven't historically had that much National Institutes of Health funding. As a graduate student, I had had some grant-writing experience. During the transition to Montana, I did know that these opportunities were going to be here, and coming to a place that had little infrastructure would have been hard to accept had I not known that the potential was there.

Other collaborators across the state were very willing to help develop proposals and allow me to perform experiments in their labs if they had equipment that we needed. Building collaborations meant there was a lot of assistance for somebody new like me to be part of this statewide initiative.

I was hired on a Biomedical Research Infrastructure Network grant that was a smaller NIH grant that lasted for two years. When the INBRE came along, it was larger and involved a number of institutions throughout the state, and we were one of the beneficiaries.

Could you describe your career trajectory, highlighting your evolution as a scientist?

As I went through my undergrad degree in biology (at Bloomsburg University of Pennsylvania), the idea of being a professor was always there for me, and largely as a teaching professor in an undergraduate institution. Research experience at the University of Pittsburgh between my junior and senior years in college introduced me to scientists and convinced me to go on into a cell/molecular biology area and get a Ph.D. As part of the graduate program at the University of Pittsburgh, I was on a teaching assistantship. I actually got to do a lot more teaching than most graduate students did. My adviser encouraged me to participate in other teaching opportunities in different environments – like I taught molecular biology for a couple of years at the dental school.

Close to graduation, I started looking for teaching postdoc positions that were rare at that time. At Juniata College in Huntingdon, Penn., I got to teach half time and mentored undergraduate researchers part time; it was an awesome experience.

Due to my outdoor activities, I looked for opportunities in the West, and the UMW opportunity presented itself to allow me to do a lot of the same. I don't think that I could have dreamed for it to work out so well for myself.

What would you tell postdocs/Ph.D.s looking to pursue teaching careers in small college settings?

The institution must have the infrastructure or the ability to build infrastructure to establish your own research program and to provide undergrads in your courses with the experiences that you feel they need to have to truly be effective after graduation. You have to certainly love teaching – there's always a heavy demand on teaching, and you have to love it, or else getting up to work will really not be fun. Personally, I think somebody that wants to be successful teaching biology in a small school has to be sold on the idea of marrying research with teaching, because providing undergrads that research experience is the essence of being a biologist. You have to see the educational benefit of teaching someone how to make a buffer or to do a streak plate on a petri dish. You might not have a high-powered research program, but the reward of teaching someone is something you should be looking for. Since the pace of research is much slower here, it is important to find an area that is related, maybe a little less explored with regard to the aspect that you will be focusing on. The challenges with that, of course, are learning a new system, new organism, new literature and new approaches to asking questions experimentally . . . Sometimes the products of your labor are not very tangible; at the end of the day, you don't know if you were really successful at your job or not. I have been fortunate to see a program and the success of the program build in a relatively short amount of time, so there has been a lot of positive reward associated with what we have done.



Preethi Chander (chander.preethi@gmail.com) is a science policy analyst at the National Institutes of Health.



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Thematic series

Glycans: the underappreciated building block of life

BY JOSEPH P. TIANO

Glycans are saccharides that can be attached to a wide variety of biological molecules through an enzymatic process called glycosylation to augment their function. Of the four fundamental building blocks of life, proteins, carbohydrates (glycans), lipids and nucleic acids, glycans have received the least attention from researchers. Glycans are found in Archaea, bacteria and eukaryotes, and their diverse functions contribute to physical and structural integrity, extracellular matrix formation, signal transduction, protein folding and information exchange between cells (and pathogens). A recent issue of the Journal of Biological Chemistry highlighted the important and diverse biological functions of glycans in a thematic minireview series organized by Associate Editor Gerald W. Hart.

In the first article, authors Stevan Springer and Pascal Gagneux discuss how evolution shaped glycan diversity. The regulatory capacity and structural diversity of glycans surpasses that of other biological molecules. The authors argue that glycan diversity stems from their role as mediators of cellular interaction. Glycans are the predominant molecule on the cell surface and serve as the first point

of contact between a cell and other cells, the extracellular matrix and pathogens. The heightened evolutionary pressure of being at the front lines of cellular collaboration and conflict most likely led to the diversification of glycans, the authors argue.

Harald



Nothaft and Christine M. Szymanski discuss the possibilities of exploiting bacterial N-glycosylation to create new vaccines and diagnostics in their review. In the never-ending fight against disease, it is essential that we continue to enhance our repertoire of drugs and vaccines. The most effective and safest vaccines are those in which a polysaccharide antigen is attached covalently to a protein carrier molecule. However, the process of generating these conjugate vaccines is expensive, time consuming and sometimes inefficient. The authors review the possibility of using bacteria to glycoengineer effective compound vaccines similar to how human insulin is genetically engineered in *E. coli*.

Duy T. Tran and Kelly G. Ten Hagen in their review focus on the role of mucin-type O-glycosylation during eukaryotic development to shed light on its role in human disease and disability. O-Glycosylation is one of the two most abundant forms of glycosylation (N-glycosylation being the other) and plays an essential role in protein secretion, stability and function. O-glycans are found in distinct locations within developing tissues and even show developmental stage-specific changes in branching. A handful of human diseases as well as tumor formation and progression are attributed to defects in O-glycosylation. The authors argue that a better understanding of O-glycosylation during development will lead to a better understanding of human disease.

In the penultimate review, Lance Wells highlights the role of glycans in the group of debilitating and life-shortening disorders known as congenital muscular dystrophy, or CMD. Both membrane proteins and the ECM are highly glycosylated, and O-glycans are essential for proper ECM function and communication between cells and the ECM. Several forms of CMD are known to result from dysfunctional O-glycosylation of membrane and ECM proteins; however, one-third of CMDs arise from an unknown genetic etiology. Future studies are needed to appreciate fully the complex role O-glycosylation plays in cell-to-ECM communication and CMD.

In the last review, Hudson H. Freeze discusses bridging the gap between identifying genes responsible for disease and explaining the molecular mechanisms causing the disease. Historically, genes responsible for glycan disorders were discovered by biochemical analysis. Now, gene sequencing and high-tech gene mapping techniques can identify identifying slightly more than 50 percent of responsible genes. A new collaboration between geneticists and glycan biochemists will lead to the discovery of many more glycosylation disorders, their molecular causes and new treatments for existing glycosylation disorders.

Joseph P. Tiano (tiano233@hotmail.com) is a postdoctoral fellow at the National Institute of Diabetes and Digestive and Kidney Diseases in Bethesda, Md.

Swapping cousins: deuterium for hydrogen

BY PREETHI CHANDER

What do snake venom, sperm maturation and pancreatic fluid have in common? Phospholipase A₂, a group of enzymes that release fatty acids from phospholipid components of biological membranes. The released fatty acids, especially arachidonic acid, play regulatory roles in inflammatory responses, thus making phospholipases highly valuable for therapeutic purposes.

PLA₂ enzymes associate with membranes and interact with their substrates to perform their function. Unfortunately, examining the dynamics of these interactions at the molecular level has been challenging due to by the insolubility of the phospholipid substrates and large molecular size of PLA₂. In a recent minireview in the Journal of Biological Chemistry, Edward Dennis and co-workers at the University of California, San Diego, describe hydrogen–deuterium exchange mass spectrometry as a useful method for examining this set of proteins, despite the technique's shortcomings.

Hydrogen atoms that are part of the protein backbone are frequently exchanged with hydrogen atoms in the surrounding water. Deuterium is a heavier isotope of hydrogen and is interchangeable with hydrogen. Harnessing these properties, the DXMS technique has been used to examine regions of PLA₂ that contact membranes, substrates and inhibitors.

The authors of the minireview describe the advantages and technical challenges of using DXMS to probe the dynamics and variety of membrane-PLA₂ association modes. Highly disordered regions and inhibitor binding sites on PLA₂ can be detected by DXMS. These regions are not visible in crystal structures due to high flexibility and problems with co-crystallization. On the other hand, extremes of ordered and disordered regions are beyond the detection sensitivity limits of DXMS. Also, they emphasize, careful experimental design is required to overcome challenges with localization of DXMS changes onto the protein as a whole.

DXMS gives moderate-resolution data; but, the authors say, in conjunction with high-resolution protein structure data, molecular models, site-directed mutagenesis and other biochemical evidence, it can serve as an efficient method to glean a more complete understanding of membrane proteins dynamics.

Preethi Chander (chander.preethi@gmail.com) is a health science analyst at the National Institutes of Health.



Tomatoes with mimic of good-cholesterol peptide benefit mice

BY RAJENDRANI MUKHOPADHYAY



Researchers have come up with genetically engineered tomatoes that could stave off heart attacks and strokes. In a recent paper in the Journal of Lipid Research, a team led by Srinivasa Reddy and Alan Fogelman at the University of California, Los

Angeles, described fruit that contained a protein that helps to halt atherosclerosis, the buildup of arterial plaques that leads to heart attacks and strokes.

One way to treat atherosclerosis is to give patients apoA-I mimetic therapy. The 243-amino-acid apolipoprotein A-I is the main component in high-density lipoprotein, also known as good cholesterol. In animal models and humans, infusions of apoA-I have been associated with improvements in atherosclerosis. But its length makes it an expensive protein to manufacture, and it has to be given intravenously.

Mimics of apoA-I of 18 to 26 amino acids have been produced. They don't have sequence similarities with apoA-I, but they bind lipids in the same way. Reddy, Fogelman and their colleagues have been studying an apoA-I mimetic peptide called 4F, which has been demonstrated in animal models to reduce atherosclerosis and disease processes associated with inflammation. The animal studies spawned two clinical trials; data from these trials indicated that 4F was most effective at preventing atherosclerosis when taken orally and processed in the digestive system.

But the problem was that the oral dose was too high to be cost effective. "The 4F peptide can only be made by chemical synthesis," explains Reddy. "The cost of produc-

ing enough 4F peptide by chemical synthesis to achieve efficacy prevented this from being pursued as a therapy in humans.”

The investigators searched for a peptide that could be synthesized in a biological rather than chemical manner. The apoA-I mimetic peptide 6F seemed to fit the bill, so the investigators decided to see if they could produce it in tomatoes. “We wanted to produce the peptide in a plant that could be eaten without cooking, because we felt that cooking the peptide might denature it,” says Fogelman. “The tomato was a convenient and tasty choice.”

The investigators genetically engineered tomatoes to produce 6F, freeze-dried them and ground them into a powder. They then added the powder to a high-fat, high-cholesterol Western diet for mice. “We found that, some hours after feeding the peptide, it was still intact in the small intestine,” says Reddy. “Markers of inflammation in the blood were significantly reduced, HDL-cholesterol and HDL function were significantly improved, and atherosclerosis of the aorta was significantly reduced.”

The investigators say the work demonstrates that tomatoes engineered to produce an apoA-I mimic could potentially reduce inflammation and atherosclerosis when eaten. Because the engineered tomatoes can be eaten whole, there is no need to extract and purify the peptide from the fruit.

Rajendrani Mukhopadhyay (rmukhopadhyay@asbmb.org) is the senior science writer and blogger for ASBMB. Follow her on Twitter at twitter.com/rajmukhop.

MCP MOLECULAR & CELLULAR PROTEOMICS

Blood proteins may forewarn of pregnancy complications

BY RAJENDRANI MUKHOPADHYAY

No pregnant woman gets excited about invasive procedures. But for the diagnosis of some complications, pregnant women have to undergo procedures such as amniocentesis. Researchers have been aiming to come up with less invasive tests. In a paper in a recent issue of *Molecular & Cellular Proteomics*, scientists describe how a class of proteins can be tracked reliably in blood samples taken from pregnant women and found hints that changes in expression patterns of this class of proteins may indicate if a pregnant woman is at risk for premature delivery.

The class of proteins that Danielle Ippolito and colleagues at the Madigan Army Medical Center studied

was apolipoproteins. “Apolipoproteins are lipid-transport proteins in plasma,” which, along with lipids and cholesterol, increase exponentially in pregnancy to support fetal development, explains Ippolito. Apolipoproteins exist stably in the blood plasma, suggesting they can be reliable indicators of different biological processes.

Ippolito says she, her colleagues and other researchers had found earlier that maternal plasma had different concentrations of apolipoprotein subtypes depending on whether or not the women developed preeclampsia. Based on that finding, the investigators reasoned that if they could track these various subtypes, they would be able to find proteins that signaled early on whether a pregnancy was going to be complicated.

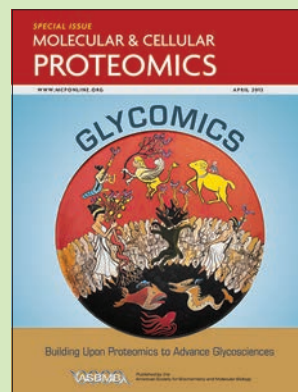
To see if their hypothesis bore out, the investigators analyzed by mass spectrometry the plasma collected from women at different time points of their pregnancies. They found that modified subtypes of an apolipoprotein called Apo A-II were significantly higher in plasma from mothers who delivered preterm babies than those who didn’t.

Ippolito says that as the next step, her team intends “to analyze plasma from patients with different obstetric outcomes to compare apolipoprotein profile in patients who deliver without complications versus women who develop gestational diabetes, severe preeclampsia and preterm premature rupture of the membranes leading to premature birth.”

Rajendrani Mukhopadhyay (rmukhopadhyay@asbmb.org) is the senior science writer for ASBMB Today and the technical editor for the *Journal of Biological Chemistry*. Follow her on Twitter at www.twitter.com/rajmukhop.

MCP presents

Building upon proteomics to advance glycosciences



The April issue of *Molecular & Cellular Proteomics* offers a thematic review series on glycomics. Read the introduction by Lance Wells and Gerald W. Hart and nine other articles at www.mcponline.org.

15 tips for effective interviewing

BY PETER J. KENNELLY

As a job candidate, your No. 1 goal is to present and, when possible, highlight for your evaluators the strong points of your training, experience, ability and potential. Here are some tips to help you achieve that goal. But keep in mind that an interview should be an exchange, a dialogue – not a theatrical performance or a confidence scheme.

1. The foundation of a successful interview is a strong set of qualifications. While marginally qualified candidates do get offers on the basis of exceptional sales pitches, their success is to a great degree reliant on the naiveté of the evaluator. Experienced interviewers quickly learn to probe beneath the surface.

2. It is your interview. Whatever happens will be associated with you (not the interviewer), so do your best to make sure things run smoothly.

3. You are always on the record during an interview. Treat everyone you encounter with courtesy and respect. Avoid controversial subjects, such as politics. Avoid cracking “jokes.”

4. Greet each person with a firm handshake and some words of greeting. While the interviewer should take the initiative, if he or she does not, you should.

5. Dress professionally. Carry a handkerchief and use it if necessary. Avoid distracting the interviewer with the excessive use of makeup, fragrances or jewelry.

6. Use effective body language. Face the interviewer. Turn your chair if necessary. Sit up. Lean forward. Avoid defensive postures, such as crossed arms. Avoid uninterested postures, such as leaning back or slouching. Avoid distracting mannerisms, such as twisting a ring, probing your ears or tapping your fingers. When not using them to make a point, clasp your hands together to prevent them from wandering.

7. Answer questions directly and concisely. Practice to find the sweet spot between rambling and producing only monosyllabic grunts. Work to eliminate repetitive verbal ticks (such as “you know,” “well,” or “um”). Videotaped mock interviews can be very helpful.

8. Not everyone you meet will be prepared; don't let this throw you. Keep a copy of your schedule within easy reach. If your host asks, “How much time do we have?” or “Who do you see next?” you will have the answers at hand, which also is a practical demonstra-

tion of your preparedness and organizational ability.

9. Do your homework. You need to gather information about the employer or program to help inform your decision should you receive an offer. Also, you may need to carry the conversation at times. Fairly or unfairly, awkward silences will reflect on you.

10. Ask questions! Many interviewers interpret the absence of questions as lack of interest in the position, lack of preparedness or poor teamwork skills. Prepare a handful of probing but neutral-sounding questions (e.g., “What do you think distinguishes this school/company from its competitors?”).

11. Be ready to answer these and similar questions: “What stimulated your interest in our program?” “How did your project fit into the overall research program of your adviser?” “Where do you see yourself five years from now?”

12. Make sure your résumé is accurate and free of exaggeration or embellishment. Anything you have mentioned on your résumé is fair game.

13. If a meal is on your interview itinerary, chose food that is easy to handle. You don't want to wear your lunch to your afternoon appointments. Minimize the consumption of liquids, as you may have few opportunities to relieve yourself.

14. Do not bring up compensation. You want to avoid creating the impression that money is your only motivation. Arrive prepared to discuss salary expectations, but your host may not raise this topic until after he or she has decided to extend an offer.

15. Arrive well-rested. An all-day interview can be taxing. You want a clear head and the physical and mental stamina to roll with the unexpected and engage the eighth person you encounter with the same energy and enthusiasm as the first.

Bonus: Follow up. When you return home from your interview, send an email or note thanking your host. Doing so demonstrates good interpersonal skills and reinforces your interest in the position.



Peter J. Kennelly (pjkenne@vt.edu) is a professor and the head of the department of biochemistry at Virginia Polytechnic Institute and State University and chairman of the ASBMB Education and Professional Development Committee.

Teaching science in the 21st century: as easy as 1, 2, tweet

BY GEOFF HUNT

The Internet has a way of being distracting. The plethora of available information and facile social interactions make staying focused a serious challenge for even the most dedicated professionals, let alone teenagers. But for Nashville-based high-school teacher Adam Taylor, it is exactly those properties that are appealing.

“As a science teacher, I am constantly looking for ways to engage students,” he says. Rather than trying to fight an uphill battle, Taylor is instead using the social-media platform Twitter to incorporate online scientific information into his lessons.



Follow Adam Taylor, founder of #scistuchat, at www.twitter.com/2footgiraffe

Science at the 140-character level

Taylor's motivation for turning to social media came about out of necessity. “It is difficult to find scientists and get them into your classroom,” he laments. However, locating experts in the ether of the Internet was much more straightforward. Still, Taylor worried

whether the level of interaction that was taking place online was sufficiently informative for high-school science students. A 2011 news story in the journal *Nature* (1) convinced him that valuable scientific discussions were being conducted in real time via Twitter, exactly the kind of interaction that he was looking for. “Once I found lists of scientists on Twitter, I had my students follow different scientists and start interacting with them. Some scientists even responded to student questions,” Taylor says.

Encouraged by these interactions, Taylor decided to codify them into a formal event. Thus was born #scistuchat, a real-time, moderated question-and-answer session open to the Twitter-sphere. Taylor's one-off event was a success. “Both students and scientists seemed to enjoy themselves, so I decided to make it a monthly event,” he recalls.

Version 2.0

Now held the second Thursday of every month, #scistuchat has grown to involve students from high schools in Kentucky, New York, New Mexico, Kansas, Arizona and Canada. Even more impressive, the list of participating scientists includes researchers hailing from as far away as Australia, New Zealand and Germany – which, to Taylor, is gratifying: “The goal is to connect students with scientists. I can raise engagement of students with scientific professionals, as well as connect them to the outside world.”

Taylor gets the discussion rolling by coming up with questions on surprisingly sophisticated topics chosen by his students, such as cloning and genetically modified food. In a typical chat, he sends out an initial question (e.g., “What is DNA?”) and asks anyone who is following to respond and include the “#scistuchat” hashtag in his or her tweet. Anyone logged on to Twitter can join the conversation; others can follow along by searching for the #scistuchat hashtag.

As the discussion progresses, students follow up with their own increasingly in-depth questions for scientists (e.g., “How can you change your DNA to change

your skin color or eye color?”). This format, says Taylor, “keeps students involved and keeps them from getting frustrated” at not getting responses to all their queries. To keep things moving, Taylor says, each topic is discussed for no more than 10 minutes.

Reaction to the chat has been overwhelmingly positive. According to Taylor, “the students who participate get into it.” They are not the only ones: Past #scistuchat discussions have drawn more than 1,300 tweets in 60 minutes, a rate that had the #scistuchat hashtag ranked as one of the most popular discussion items on Twitter.

Responses from participating scientists have been similarly enthusiastic. Kristopher Hite (@thorsonofodin), a postdoctoral fellow at Emory University, once tweeted, “#scistuchat is one of the coolest things I have ever participated in my life.” Khadijah M. Britton (@KMBTweets), founder of Boston’s BetterBio, wrote, “This chat rocks!”

Into the great wide open

Taylor sees #scistuchat as part of a larger effort to encourage the use of social media in the classroom.

“Hopefully these kinds of interactions will help students become more interested in the world around them,” he says. To emphasize that point, he recently invited members of the Tennessee state legislature to his class to observe and participate in one of the #scistuchat events. “Our visitors were excited about the level of engagement from the students,” he says.

Ultimately, the *raison d’être* of #scistuchat is getting Taylor’s students interested in science. “Talking to actual scientists can help to open students’ minds to the opportunities that are out there in the world of science,” he points out.

Getting scientists involved goes a long way toward achieving this goal. “The more scientists we have on #scistuchat, the more students will feel a connection.” Even the smallest interaction makes a difference, he insists. “If nothing else, (the students) will remember the time they had a question answered by a scientist on Twitter.”



Geoff Hunt (ghunt@asbmb.org) is ASBMB’s outreach coordinator. Follow him on Twitter at www.twitter.com/goodbyeshoe.

REFERENCE

1. Reich, E. S. *Nature*. **474**, 431 (2011).

A sampling of recent #scistuchat tweets

MARCH 14: EVOLUTION

Question 1: “What is evolution?”

@TimBarzyk: A1 When random mutations persist enough through generations to become established traits. #scistuchat Q1 What is evolution?

@HeidiKayDeidi: A1: change of characteristics over time #scistuchat

@pusk52: Q1. What is evolution? A1. Heritable change in populations over time. Remember- that change is measured in genes and alleles #scistuchat

@morganfaith6: Evolution is a gradual process where something changes into a different and usually more complex or better form #scistuchat

@TaylorJackson0: #scistuchat How long would it take for us to evolve after a huge change?

@DoctorZen: @TaylorJackson0 We have a long generation time - 20 years - and a LOT of mixing of populations. So, a long time. #scistuchat

Question 2: “How does evolution work?”

@DoctorZen: Q2. How does evolution work? A2. Several ways, the major being natural selection. #scistuchat

@MsTaylorLHS: A2 There are several mechanisms of evolution, including natural selection, sexual selection, genetic drift, etc. #scistuchat

@thorsonofodin: A2.) Evolution happens when a change in the DNA code persists through generations. If the change is an advantage it stays. #scistuchat

@PaleoRomano: A2 Evolution is not a driving force, but a reactionary force. #scistuchat

@pusk52: Q2: How does evolution work? A2 are there any easy questions #kidding #scistuchat

It's meeting time!

BY DANIEL RABEN

Now that April is upon us, everyone is (hopefully) gearing up for the American Society for Biochemistry and Molecular Biology annual meeting later this month in Boston. Everyone has seen the highlights of the various programming themes, so I won't explore those here. I do want to highlight some newsworthy items that are of interest to those of us in the lipid research community.

Big news for POPG



One of our premier lipidologists, Dennis Voelker at National Jewish Health, just received a U.S. patent for the lipid POPG (1-palmitoyl-2-oleoyl-sn-glycero-3-phosphoglycerol), which reduces inflammation and inhibits infection in the lungs, and various related compounds.

Dennis has been a leader in lipid biochemistry and has maintained an interest in the lung lipids. Another advance for the clinical importance of lipids!

White, brown — and now beige — fat



Lipids have gotten more colorful. As many of you know, last year Bruce Spiegelman of Harvard Medical School and the Dana-Farber Cancer Institute identified a new fat cell — beige fat! These interesting cells are sort of a hybrid between white adipocytes and brown adipocytes. In adults,

these cells are scattered beneath the skin near the collarbone as well as along the spine. These cells originally were identified in 2008, and they now have been shown to be specifically targeted by irisin, which is expressed by muscle cells during exercise. In addition to stimulating the conversion of white fat into brown fat, irisin improves glucose tolerance and stimulates weight loss in obese, prediabetic mice. It will now be exciting to identify the role of beige cells in these mice in diet-induced obesity. Will this be a new therapeutic target for obesity?

The gut microbiome and weight control

Speaking of obesity, interest is increasing regarding the role of those little microbes growing in your gut. Recent studies have implicated the composition of the gut microbiome in playing a significant role in weight gain. The composition of the gut microbiome, which can be influenced by diet, demonstrates a complex system of interactions that plays a role in regulating body weight. So if you're going to the annual meeting and friends want to go out for lunch, think about yogurt!

And let's not forget our young'uns

This year's winner of the Walter A. Shaw Young Investigator Award in Lipid Research is Tobias Walther of the Yale School of Medicine. He will give a talk titled "Cell Biology of Neutral Lipid Storage" at 3:45 p.m. April 23 at the ASBMB annual meeting in Boston. For more about Walther, see page 12.

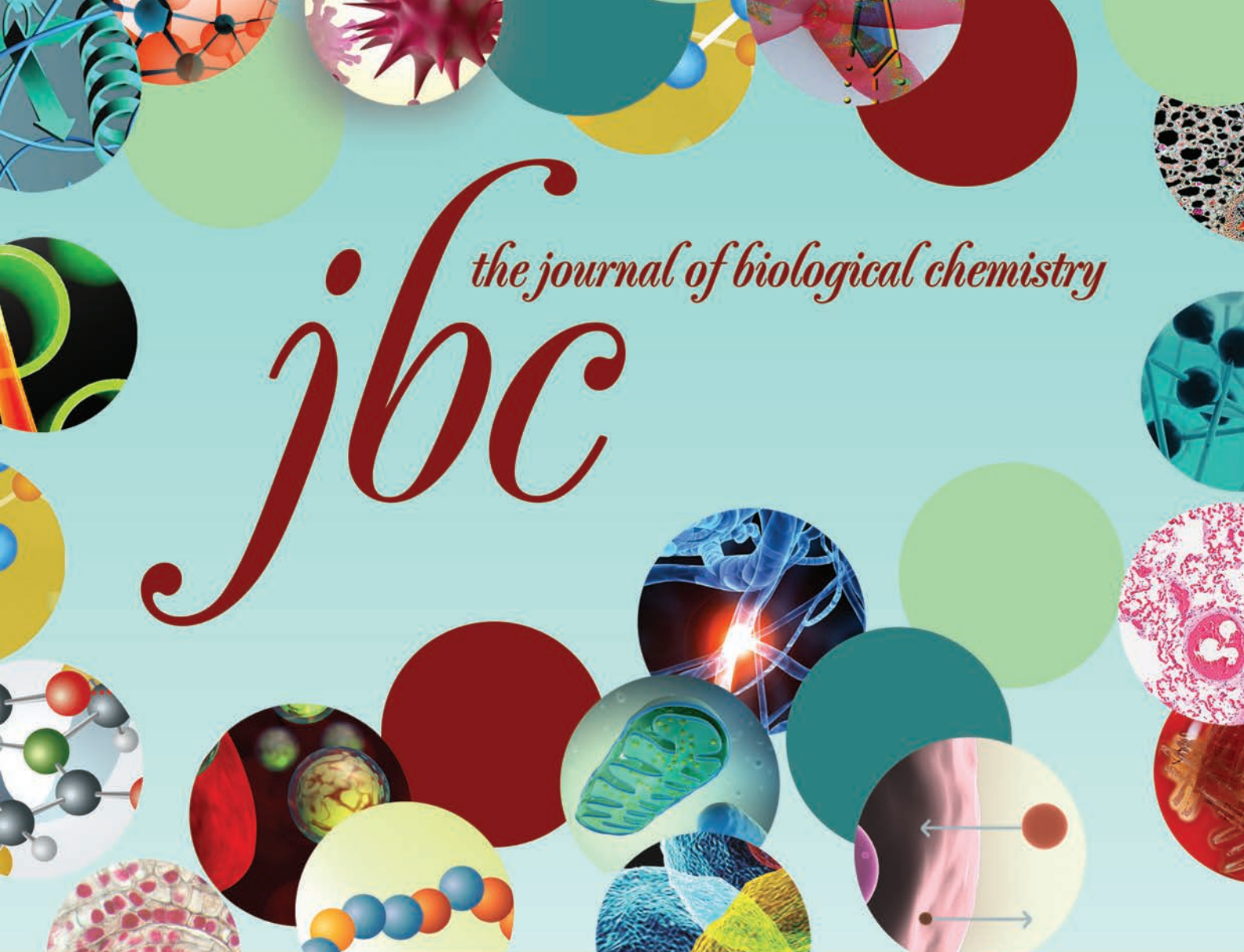
Lipids always float to the top!



Daniel Raben (draben@jhmi.edu) is director of the ASBMB Lipid Division and a professor in the department of biological chemistry at the Johns Hopkins University School of Medicine.

MCP issues guidelines and checklist for reporting glycomics data

The journal Molecular & Cellular Proteomics has established guidelines and a checklist for reporting glycomics data to help authors ensure that they are clearly communicating how they did their analyses and to ensure that reviewers and readers can understand their experiments. For more information, visit <http://www.mcponline.org/>.



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The Journal of Biological Chemistry's editors are pleased to announce that 22 papers have won Best of 2012 designations. The Best of 2012 manuscripts were selected from the more than 4,000 papers published last year. One Best of 2012 paper was chosen from each of the journal's Affinity Groups for its excellence and potential impact on the field.

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THEMATIC SESSIONS

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Genome Replication and Repair #DNArep

Glycan Regulation of Signaling Pathways #sugarsig

Lipids and Membranes #lipids

Mechanisms of Gene Transcription and Regulation #genetxr

Mechanisms of Signal Transduction #cellsig

Protein Modification, Trafficking and Degradation #proteinlife

RNA Function and Protein Synthesis #RNAfxn

Transitions, Education and Professional Development #stemcareer

Triple-Negative Breast Cancer #breastcancer

How Scientists Can Save the World

Sponsored by the ASBMB Public Affairs Advisory Committee. Sunday, April 20, 12:45 - 2:30 p.m., Room 258

Join the leading names in science and public policy as we discuss the massive challenges facing society in the next 100 years. Learn how science holds many of the solutions to problems like hunger, health, and sustainability — and how to be an advocate for science.

Panelists:

- Tania Baker, Massachusetts Institute of Technology
- Darlene Cavalier, Science Cheerleader
- Craig Mello, University of Massachusetts

Moderator:

- Jeremy M. Berg, ASBMB President
University of Pittsburgh