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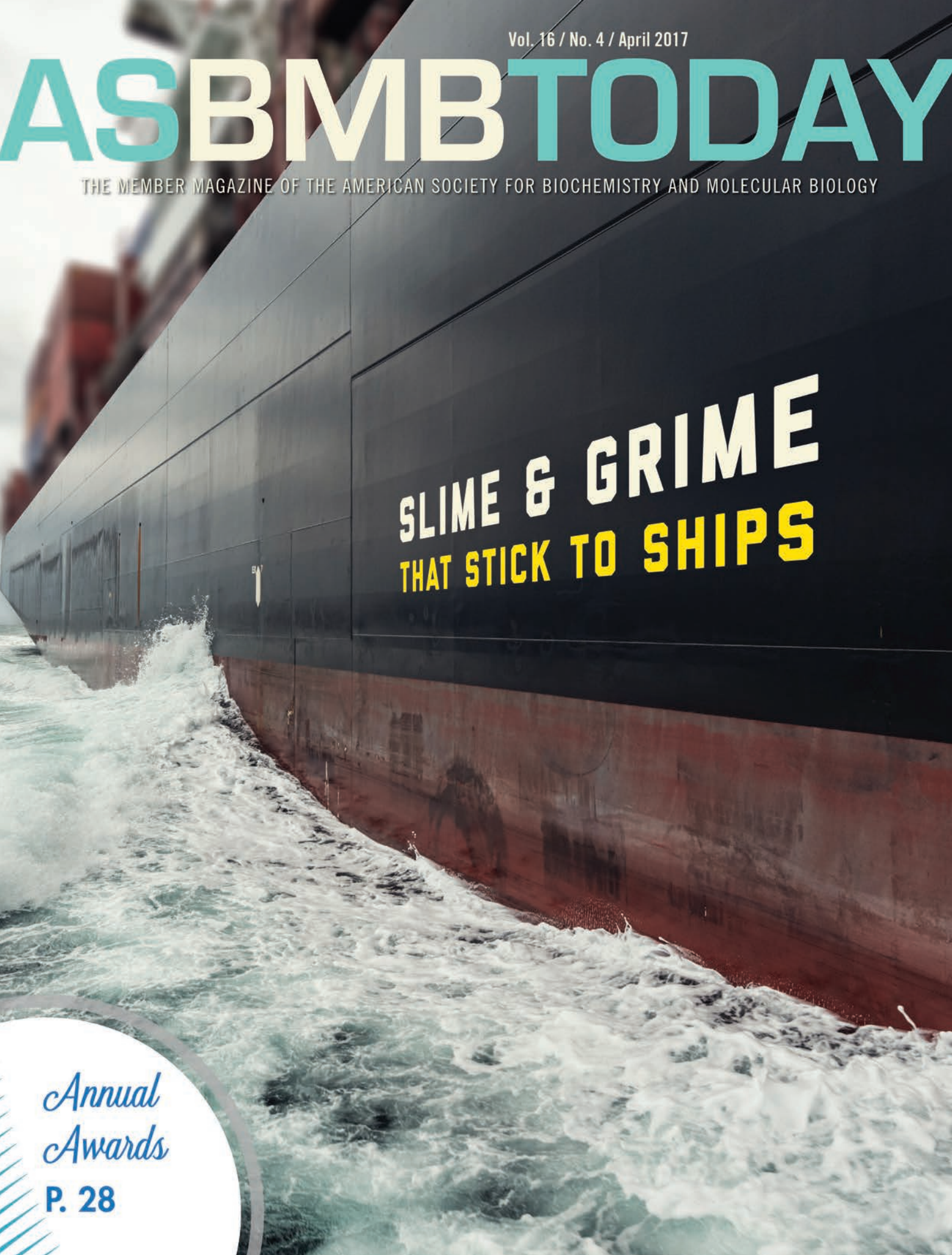
ASBMB TODAY

THE MEMBER MAGAZINE OF THE AMERICAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY



SLIME & GRIME THAT STICK TO SHIPS

Annual Awards

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2017 ASBMB career-development opportunities

Webinars

If you registered for a webinar before March 8, please re-register.

Research careers in industry
April 6

Careers beyond the bench in industry
May 11

Developing successful application materials
June - date TBD

No registration fee! Space is limited.
Register at www.asbmb.org/webinars

Online resources

- Short tutorials on career topics such as informational interviews
- Videos describing the types of careers open to Ph.D.s
- The course "The Art of Science Communication"

www.asbmb.org/careers

Workshops

Interactive Mentoring Activities for Grantsmanship Enhancement
June 22–24 | Washington, D.C.
Assistant professors and postdoctoral scholars transitioning into independent faculty positions receive feedback on their research proposals and mentoring. **Apply by May 5.**

Save the date: Catalyze Your Career
Sept. 21–22 | Portland, Ore.
Learn about careers open to Ph.D.s; improve your communication skills, application materials and interview skills; network; and plan for your career.

Save the date: Preparing Science Professionals
Sept. 29–30 | Lexington, Ky.
Gain insight into different STEM career paths and take part in interactive training in communication, outreach and professional development.

Save the date: Catalyze Your Career
Oct. 20–21 | Tucson, Ariz.
Learn about careers open to Ph.D.s; improve your communication skills, application materials and interview skills; network; and plan for your chosen career.

More information at www.asbmb.org/workshops

ASBMB
American Society for Biochemistry and Molecular Biology

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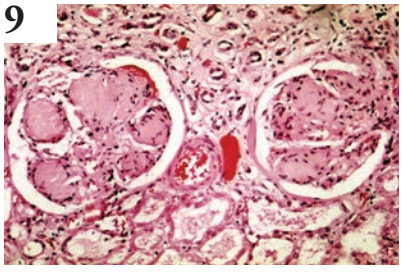

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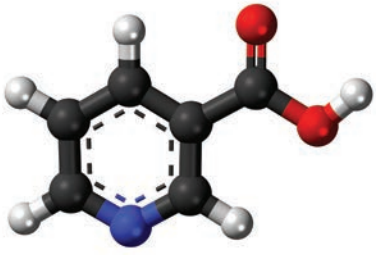

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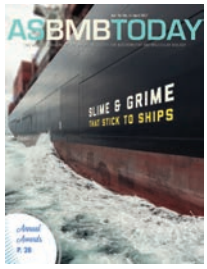
Researchers are trying to exploit the biochemistry of marine organisms to stop the persistent problem of biofouling on ships.



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Science matters

By Natalie Ahn

On April 22, people will gather in more than 300 cities across the U.S. and around the world for the March for Science (www.marchforscience.com). The goals of the march are many, but in general, it aims to celebrate science and its vital public service role, promote respect for research and the scientific method, and defend the importance of evidence-based thinking and decision-making.

The American Society for Biochemistry and Molecular Biology wholeheartedly supports the efforts of the March for Science. Scientists at all career stages work every day to maintain the flow of new knowledge through evidence-based research and logic. We train young scientists in critical thinking skills and the scientific method. While interpretations of experimental observations can and do vary, we hold ourselves to high standards for rigorousness and impartiality, and we strive to reach unbiased conclusions as much as humanly possible.

Science and the scientific method are unquestionably nonpartisan. The political affiliations of the scientists doing the experiments should have no bearing on the interpretations of data. Likewise, illnesses and disease are equal opportunists — they welcome members of all political parties.

Everyone deserves to benefit from the discoveries of science; everyone benefits when science is supported in a bipartisan manner, as has been the case for the past 72 years. Vannevar Bush's report "Science — the Endless Frontier" is just as relevant today as it was in 1945: "It has been basic United States policy that Government should foster the opening of new frontiers. It opened the seas to clipper ships and furnished land for pioneers. Although these frontiers have more or less disappeared, the frontier of science remains. It is in keeping with the American tradition — one which has made the United States great — that new frontiers shall be made accessible for development by all American citizens." Our mandate was then and still remains to uncover the realities of how the world works and train new generations to take this knowledge farther.

That science increasingly is distorted for political gain is deeply troubling to me. It's not that I'm surprised when people sometimes make irrational statements with no basis in fact. I've raised teenagers and have been a teenager myself, so I'm not pointing any fingers. But, as a scientist, I worry when people started believing things that are obviously false. As scientists, we hold ourselves to standards dictating that statements must be grounded in evidence, and it is unacceptable to discount or reject facts without evidence-based justification.

So what can we, as individuals, do? First, attend the March for Science wherever you will be on April 22 and show the world how important science is to everyone. Then, in the coming years, become an ambassador for science. Engage with nonscientists and policymakers to explain what you do, how research expands the frontiers of knowledge and why science is essential for national prosperity.

For many of us, reaching out to nonscientists and policymakers isn't our strong suit. We'd rather spend 12

hours analyzing data than two minutes chatting with the other person in the elevator. The ASBMB has developed many resources to help you begin. An online course developed by the Public Outreach Committee will help you learn how to communicate your work to nonscientific audiences (www.asbmb.org/Outreach/Training/ASC).

The Public Affairs Advisory Committee organizes yearly Hill Day visits to Capitol Hill, an effective way for American citizens to communicate the importance of science to policymakers. The PAAC's Advocacy Toolkit will teach you how to work with Congress; write letters to senators, representatives and local newspaper editors; and host visits by members of Congress to your lab and institution (www.asbmb.org/Advocacy/PAAC/). Finally, this magazine is a great way to publish your ideas and thoughts on the importance of science in society. You can submit your pieces to asbmbtoday@asbmb.org.

If you're attending the 2017 ASBMB Annual Meeting in Chicago, both the POC and PAAC have organized events to help you get started in engaging with nonscientists and policymakers. On April 22, the POC will hold outreach events for meeting attendees in Chicago, which will have a satellite March for Science; you can find out about these events on the POC's website (www.asbmb.org/Outreach/). You also can attend the PAAC town hall on April 24, where we will discuss the impacts of the political landscape on biomedical research (for more details, see page 26).

Many people I've met have told me how they've resolved to stand up actively for science. If ever there has been a time to reinforce the importance of scientific knowledge and evidence-based thinking to policymakers and to the world, it is now. Let's build a bridge between marching for science today and establishing lifelong

Natalie Ahn (natalie.ahn@colorado.edu) of the University of Colorado, Boulder, is president of the ASBMB.

Sumter is first Winthrop provost's faculty fellow


SUMTER

Winthrop University has named Takita Felder Sumter, professor of chemistry, as its inaugural provost's faculty fellow.

The Provost's Faculty Fellows Program recognizes outstanding faculty members by providing them with opportunities for administrative development and leadership. Sumter will represent the office of the provost on special projects and routine office operations.

Sumter is the chair of the American Society for Biochemistry and Molecular Biology's Minority Affairs Committee. She is involved in the Interactive Mentoring Activities for Grantsmanship Enhancement initiative to assist early-career scientists who are transitioning to independent faculty positions.

Ravid earns Fulbright Scholar Award


RAVID

Katya Ravid, professor of medicine and biochemistry at Boston University School of Medicine, is a Fulbright Scholar Award recipient. Established in 1946, the Fulbright Program promotes excellence in scholarship through grants supporting academic collaboration and exchange around the world. Ravid will matriculate later this year.

Ravid will serve as an adviser at the University of Strasbourg in France, where she will lead interdisciplinary research in hematopoiesis and functions of megakaryocytes and platelets.

Ravid is the founding director of the Evans Center for Interdisciplinary Biomedical Research and of the Interdisciplinary Biomedical Research Office at Boston University. Her research focuses on investigating blood and vascular pathologies.

Wilson heads tribal health research office


WILSON

David R. Wilson has been appointed as the director of the tribal health research office at the National Institutes of Health.

Established in 2015, the tribal health research office was created to ensure participation and collaboration on NIH policies and programs with tribal nations.

Wilson previously served as a public health adviser and the American Indian/Alaska Native policy lead at the Department of Health and Human Services Office of Minority Health. He also served on the ASBMB's Minority Affairs Committee and is an adjunct faculty member of the Center for American Indian Health at the Johns Hopkins School of Public Health.

Vrabel recognized for academics and athletics


VRABEL

In December, Jarret Vrabel of Westminster College was named the Student-Athlete Advisory Committee's male scholar-athlete of the month at the Presidents' Athletic Conference.

The committee selects one male and one female student-athlete each month who excels both on the field and in the classroom.

A three-year starter on the school's basketball team, Vrabel is the team's second-leading scorer while ranking fourth in the conference in rebounding through the team's first 15 games.

The Westminster College junior is a biochemistry major with a 3.55 cumulative GPA. He is a member of the All-College Honors Program and chemistry club. He also serves as the treasurer of the institution's student chapter of the ASBMB.

Brunori elected vice president of the Accademia Nazionale dei Lincei


BRUNORI

Maurizio Brunori, professor emeritus of biochemistry at Sapienza University of Rome, was elected vice president of the Accademia Nazionale dei Lincei in Italy and president of the class of physical sciences.

Brunori's primary research interests lie in the study of protein structure, function, folding and dynamics. Among his many honors, Brunori is a member of the American Academy of Arts and Sciences, a member and fellow of the Biophysical Society, and a member of the European Molecular Biology Organization.

The Accademia Nazionale dei Lincei, founded in 1603, was named after the lynx to represent the acute perception needed for greater scientific insight.

Charpentier and Doudna win Japan Prize

Emmanuelle Charpentier and Jennifer A. Doudna have received the 2017 Japan Prize for their development of the genome-editing tool CRISPR/Cas9. Awarded since 1985, the Japan Prize recognizes outstanding contributions to science and technology that also promote peace and prosperity for humankind.

CRISPR/Cas9 has revolutionized genetic engineering by allowing for faster and more efficient editing of parts of the genome. CRISPR/Cas9 has shown the potential for a wide range of applications that greatly would affect genetic research.

Charpentier is the director at the Max Planck Institute for Infection Biology in Germany and a visiting professor at Umeå University in Sweden. Doudna, a professor of chemistry and of molecular and cell biology at the University of California, Berkeley, is the Li Ka Shing chancellor's chair in biomedical science and an investigator with the Howard Hughes Medical Institute.

CHARPENTIER

DOUDNA

Erik Chaulk (echaulk@asbmb.org) is a peer-review coordinator and digital publications web specialist at the ASBMB.

Upcoming ASBMB events and deadlines

MAY

May 2: Special Symposium: Evolution and Core Processes in Gene Expression oral abstract deadline
May 5: IMAGE Grant Writing Workshop application deadline
May 9: Special Symposium: Evolution and Core Processes in Gene Expression early registration deadline
May 9: Special Symposium: Transforming Undergraduate Education in the Molecular Life Sciences early registration deadline
May 11: Webinar: Careers beyond the bench in industry
May 15: The Marion B. Sewer Distinguished Scholarship for Undergraduates application deadline
May 24: Special Symposium: Evolution and Core Processes in Gene Expression poster submission deadline
May 31: Special Symposium: Transforming Undergraduate Education in the Molecular Life Sciences poster submission deadline

JUNE

June 8: Special Symposium: Evolution and Core Processes in Gene Expression registration deadline
June 15: Special Symposium: Transforming Undergraduate Education in the Molecular Life Sciences registration deadline
June 22: Special Symposium: Membrane-Anchored Serine Proteases oral abstract deadline
June 22–24: IMAGE Grant Writing Workshop
June 29: Special Symposium: Membrane-Anchored Serine Proteases early registration deadline

JULY

July 13–16: Special Symposium: Evolution and Core Processes in Gene Expression, Kansas City, Mo.
July 20–23: Special Symposium: Transforming Undergraduate Education in the Molecular Life Sciences, Tampa, Fl.
July 20: Special Symposium: Membrane-Anchored Serine Proteases poster abstract deadline

Glycan selection in the brain

By Amber Lucas

Humans always have had an interest in understanding how we evolved to be what we are today. One of the ways that humans are different from most other mammals is loss of an enzyme called CMP-Neu5Ac hydroxylase, or Cmah for short. In a paper in the **Journal of Biological Chemistry** that was selected as one of the Editors' Picks, Yuko Naito-Matsui and others in Ajit Varki's lab at the University of California, San Diego, explored how the suppression of Cmah in the brain may confer subtle evolutionary advantages. "This is a complicated evolutionary story — it is difficult to prove what really happened during millions of years of evolution," says Varki. "However, this complexity may also attract readers, because all of us are the results of such evolution."

Neu5Ac is a sialic acid found on the surface of all vertebrate cells. Most other mammals use Cmah to convert Neu5Ac into a similar sialic acid known as Neu5Gc in all tissues except in the brain, where Cmah is not present in neural cells. The tissue-specific repression of Cmah in the neural cells of mammals and the complete loss of Cmah in humans led to the idea that the loss of this enzyme may hold evolutionary advantages.

To begin to understand what selection processes led to the suppression of Neu5Gc production in the vertebrate brain, Varki and colleagues created a mouse model called NCmahTg that expressed Cmah in the brain. These mice showed a dramatic increase in the Neu5Gc/Neu5Ac ratio as well as incorporation of Neu5Gc into neural cell-surface structures, such as gangliosides and polysialic acid.

Once they had achieved a functional model system, Varki and colleagues wanted to test whether ganglioside activity was affected by the incorporation of Neu5Gc. Gangliosides are glycosphingolipids that contain sialic acids and, in their Neu5Ac state, interact with myelin-associated glycoprotein, known as MAG, which is thought to mediate the preservation of myelination and axonal outgrowth.

"This is a complicated evolutionary story — it is difficult to prove what really happened during millions of years of evolution." — Ajit Varki

Varki's team collaborated with the team of Ronald Schnaar at Johns Hopkins University. By staining with an antibody containing the ganglioside binding domain of MAG, the investigators found that Neu5Gc incorporation disrupted the interaction between gangliosides and MAG. Images of the major axon tract in the central nervous system also showed a significant reduction in myelination, with some large axons showing complete loss of myelination.

Next, Varki and colleagues conducted a series of neuronal and behavioral tests to determine if the NCmahTg mice had any neurological impairments. Neu5Ac is a component of several neural cell-surface structures; myelination is important for motor coordination and balance. NCmahTg mice displayed slight defects in hind-limb extension and a shorter stride compared with wild-type mice. The NCmahTg mice also were shown to have impaired memory. While the phenotypes were mild, they did show a negative impact on neuronal function of Neu5Gc production in the brain.

The mild neural phenotypes did not seem to explain fully the evolutionary selection for suppression of Cmah expression in the vertebrate brain, so Varki and colleagues decided to explore whether Neu5Gc production in the brain increased susceptibility to microbial toxins. Sialic acids are known to be recognized by virulence factors, such as bacterial adhesins or viral agglutinins, and previous work by James and Adrienne Paton of the University of Adelaide in Australia identified subtilase cytotoxin as a bacterial toxin that preferentially recognized Neu5Gc. The investigators administered subtilase cytotoxin to wild-type and NCmahTg mice and monitored their survival rate. Indeed, NCmahTg mice showed higher susceptibility to subtilase cytotoxin, indicating that loss of Neu5Gc in the brain may have evolved as a protective measure against such virulence factors targeting the brain.

Protection of brain function is paramount to survival and reproductive fitness. It makes sense that, over millions of years, vertebrates evolved mechanisms that provide even a small protective advantage. "Humans completely lack Neu5Gc because of a mutation in the Neu5Gc-synthesizing enzyme," says Varki. "The present work could potentially contribute to understanding how human brains became different from those of other related species."

Amber Lucas (aluca685@gmail.com) is a graduate student at Carnegie Mellon University.

Deciphering how barbiturates work

By Nathalie Gerassimov

Modern medicine would be unthinkable without the use of anesthesia during surgical procedures. In a recent paper in the **Journal of Biological Chemistry** that was selected as one of the Editors' Picks, Marc Delarue of the Pasteur Institute in France and colleagues solved how a class of drugs called barbiturates function to induce anesthesia. "This work describes, for the first time, the three-dimensional structure by X-ray crystallography of barbiturates bound to the target receptor," says Zaineb Fourati, one of the first authors on the paper. She adds, "Understanding the mechanism underlying anesthetics' action is the first step toward the conception of better ones that are more specific and with less side effects."

Barbiturates, a class of drugs derived from barbituric acid, made their debut in Germany in 1903. Their name is thought to be a fusion of "Barbara" and uric acid, a component of urine. Sources are divided on which female inspired the name, who could have been St. Barbara or someone less than a saint.

The first commercially available form of barbiturates was marketed as sleeping pills. Other medical applications of barbiturates soon emerged, including as anesthetics, sedatives and anticonvulsants, which led to their widespread use in the first half of the 20th century. However, the side effects of barbiturates led to their regulation and decreased popularity.

"Uncontrolled use of barbiturates could have hazardous consequences, from addiction to death by overdose," says Fourati. "One concrete example is the presumably intentional death of the famous Marilyn Monroe due to a barbiturate overdose in the early 1960s." Nevertheless, barbiturates remain essential in the treatment of epilepsy and as an intravenous general anesthetic.

Anesthetics act by decreasing the relay of information in the nervous system. Specifically, anesthetics affect several types of membrane-embedded ion channels. The principal targets of barbiturates are thought to be anionic and cationic pentameric ligand-gated ion channels, known as pLGICs. Although there is some evidence to show that barbiturates bind to the cylindrical pore of the ion channel, the exact nature of this interaction is unclear.

To understand this interaction better, Delarue and colleagues used the bacterial pH-gated receptor GLIC, which has high structural similarity to mammalian pLGICs. They first showed that the bacterial version of the channel behaved much like its eukaryotic counterpart by demonstrating that a specific barbiturate called pentobarbital can inhibit the electrical current of the bacterial GLIC channel.

Next, the investigators used three barbiturate derivatives — bromobarbital, thiopental and selenocyanobarbital — that were modified chemically such that they displayed an unusual electron scattering behavior to help assign bound ligands in the X-ray structure unambiguously. The investigators showed that all three barbiturate derivatives bound to the closed GLIC channel deep within the central pore, and they used computational simulations of the channel in open and desensitized states to support their findings further.

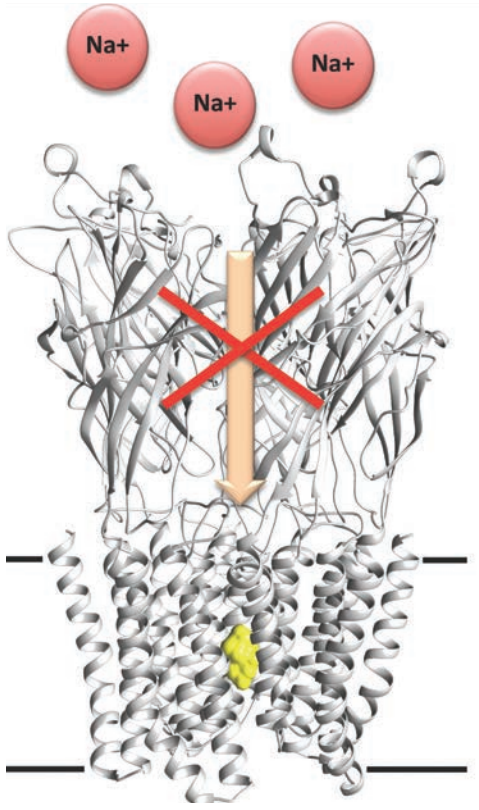


IMAGE COURTESY OF MARC DELARUE

Researchers unveil barbiturates' mechanism of action.

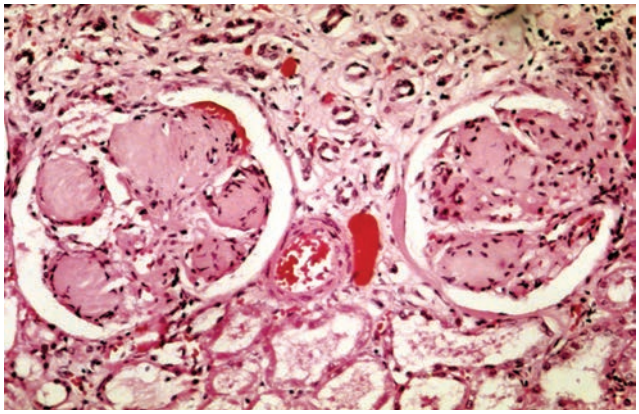
"This work partly reveals how barbiturates induce anesthesia," says Fourati. "As these drugs act on the nervous system, it is very important to address the exact mechanism of their action, and the structure provides a direct proof of a molecule binding to a given site."

She and her colleagues think that the knowledge gained in this study can help design a new generation of barbiturates not only for anesthesia but also for psychiatric and neurological disorders.

Nathalie Gerassimov (nathalie.gerassimov@gmail.com) is a Ph.D. student at Johns Hopkins School of Medicine.

Tracing damage pathways in diabetic kidney disease

By John Arnst



Patients with diabetic kidney disease can have kidneys with scarred glomeruli.

Diabetic kidney disease is the most common cause of kidney failure and can occur as a complication of both Type I and Type II diabetes. In a paper published in the journal of **Molecular & Cellular Proteomics**, researchers at the University of Toronto and the Institut Hospital del Mar d'Investigacions Mèdiques in Spain report a link between the proteomic changes caused by high levels of male sex hormones and impaired energy metabolism in diabetic kidney disease.

"It's been reported in previous clinical studies that male sex hormones predispose (patients) to many kidney diseases and lead to a more rapid progression and increased severity of kidney disease," says Sergi Clotet, the first author on the paper and a postdoctoral researcher in the laboratory of Ana Konvalinka at the University of Toronto. "Our findings proposed this novel link between male sex (hormones), energy metabolism and oxidative stress in the kidneys of diabetic animals, which are indicators of mitochondrial damage."

Diabetic kidney disease causes the clustered kidney capillaries, known as glomeruli, to leak high levels of proteins, such as albumin, into urine. The leakage eventually leads to anemia, chemical imbalance in the bloodstream and overall decline in kidney function. The disease can be stemmed if caught early on but may be exacerbated in men by elevated androgens.

Clotet and colleagues used a variation of the SILAC technique for their study. In traditional SILAC, which stands for stable-isotope labeling of amino acids in cell culture, the amino acids in cells are labeled with nonradioactive isotopes and quantified by mass spectrometry.

The SILAC variant, known as "spike-in," enabled the investigators to bypass difficulties involved with labeling the primary proximal tubular cells, which don't grow well in SILAC media, by labeling only transformed proximal tubular cells. The labeled transformed cell lysates then were combined with the unlabeled and stimulated primary cell lysates.

The investigators stimulated primary cells with 100 nanomolar doses of a sex hormone, either estradiol or dihydrotestosterone, and subjected them to mass spectrometry to identify the changes in metabolic regulation caused by each hormone.

After performing subsequent bioinformatics analyses, the researchers noticed that energy metabolism was impaired in the cells exposed to dihydrotestosterone. They validated their results by examining the expression of key enzymes involved in metabolism in kidneys of male and female normal and diabetic animals. The researchers then performed an enrichment analysis of their own data and a publicly available gene-expression data set generated from kidneys of patients with diabetes. With the analysis, the researchers were able to group and visualize significant functional categories. They found that oxidative stress was elevated significantly in the testosterone-treated samples from their dataset as well as in the kidneys of diabetic patients.

The researchers then decided to analyze oxidative stress levels and the sex differences in their animal groups, finding that the stress levels were increased in males of both normal and diabetic animals. "It's a result that suggests that male sex is associated with more severe kidney disease," says Clotet.

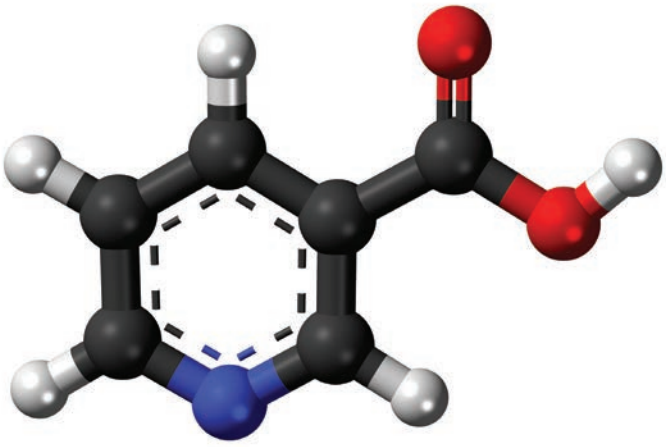
Clotet and colleagues intend to collect kidney biopsies from diabetic and nondiabetic male and female donors to validate their findings further as well as to look for biomarkers that could indicate early onset of diabetic kidney disease. "That could be a noninvasive way to predict sex-specific progression of diabetic and nondiabetic kidney diseases," says Clotet. "At the end of the day, we want to explore more personalized treatments for kidney disease."

John Arnst (jarnst@asbmb.org) is ASBMB Today's science writer. Follow him on Twitter at twitter.com/arnstjohn.

Making the most of niacin treatment

By Sapeck Agrawal

Niacin, better known as vitamin B3, acutely lowers plasma levels of free fatty acids, which helps to deter insulin resistance. However, tolerance to niacin is known to develop over time and can cause FFA levels go back up to, or even become higher than, pretreatment levels. Long-term use of niacin also is associated with worsening of glycemic control, casting doubt on the value of the niacin treatment.



A ball-and-stick model of niacin

In a recent study published in the **Journal of Lipid Research**, a team led by Tobias Kroon at the Swedish University of Agricultural Sciences and AstraZeneca in Sweden explored strategies to develop a niacin-dosing regimen that would maximize metabolic control and minimize tolerance to niacin in a rat model of Type 2 diabetes.

Niacin is known to lower plasma FFA levels by functioning as an agonist to G-protein–coupled receptors that regulate lipolysis. In diabetics, high amounts of FFA, triglycerides and glucose logjam into the blood after a meal and overwhelm the insulin machinery, leading to insulin resistance. Kroon's team wondered how niacin could be made to work most efficiently to lower these high amounts of FFA and glucose in blood.

To find out, Kroon and colleagues conducted a two-part study. The first part examined the effects of a sudden or gradual withdrawal of niacin treatment on metabolism. The second part used the better withdrawal strategy alongside feeding or fasting periods to identify the most synergistic treatment regimen.

For the first part, the investigators delivered niacin via subcutaneous implants to the rats for 12 hours. They gave half of the animals an infusion of glucose to simulate a meal. The other half didn't receive any glucose. At the 13th hour, they either abruptly stopped the niacin treatment or gradually stepped it down until completely terminating it at the 16th hour.

The investigators analyzed the metabolic responses by measuring plasma levels of FFA, glucose, insulin and triglycerides. As the team had hypothesized, plasma FFA rebounds were lowest among animals that experienced a gradual termination of niacin treatment during glucose infusion. Plasma insulin levels showed the same trend. They concluded that niacin treatment should be terminated gradually while the animals are in the fed state.

For the second part of the study, the investigators treated two sets of animals with niacin for five days — one in the feeding phase, the other in the fasting phase — followed by gradual termination of the treatment. A set of control animals received saline treatment. The investigators found that the plasma FFA and triglyceride levels as well as triglycerides in the heart and liver were lowest among animals that experienced treatment during the feeding phase. Glucose control also improved markedly in those animals. The master regulatory genes responsible for de novo lipogenesis were downregulated in the liver of these animals.

While the exact reason behind this phenomenon is unknown, the investigators believe it has something to do with the fact that reduced FFA supply resulting from niacin treatment provided much-needed insulin sensitivity during feeding, when there is an influx of dietary carbohydrate in the system.

"The main challenge (with niacin treatment) that we overcame in our work was finding this magic right way," says Kroon. At the moment, niacin is prescribed to be taken at bedtime, which, for most people, is the fasting phase. "The data raise the question of whether alternative dosing schedules in humans might improve the current clinical use of niacin," explains Kroon, although he cautions, "The ultimate proof of these ideas will require future studies in people with diabetes."

Sapeck Agrawal (sapeck.srivastava@gmail.com) is a science writer.

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The slime and grime that stick to ships

Barnacles, tubeworms and bacteria use a bevy of biochemical signals and tethers to inhabit ship hulls at great cost to maritime industries

By John Arnst

When a barnacle wants to stick to something, it opens up a capillary and bleeds. The sticky torrent of enzymes and fibrous tissues that follows gloms onto a ship hull, dock or any other submerged surface and calcifies into a permanent scab. While this is great for the barnacle, who now has a home and can begin seriously to consider starting a family, it's bothersome for a ship's operators, as the growing masses of sea creatures stuck to the vessel add drag, increase wear and tear on the ships and make trips more expensive.

This phenomenon, known as biofouling, has addled ships for as long as humans have roamed the seas. The ancient Phoenicians, one of the earliest seafaring societies, reportedly coated the bottoms of their tublike ships with pitch or copper to repel the organisms that liked to colonize them. Ship design has evolved greatly over the millennia, but biofouling organisms remain a significant problem even in the age of supercarriers and stealth destroyers. A study published in 2010 estimated the annual cost of biofouling to the entire U.S. Navy to range from $180 million to $260 million.

There are two dominant methods for handling biofouling organisms: kill the adhering microorganisms with antifouling coatings before they can settle or provide a foul-release surface that they'll slide off once the vessel is operating at high speed. However, each comes with its own drawbacks. To overcome the shortcomings of foul-release and antifouling coatings, a third variety of coatings, which exploit the biochemistry that fouling organisms use to stick to hulls, are being developed. But experimental coatings can be difficult to assess in the dynamic environment of the ocean; even the most promising coatings being tested in laboratories are leagues away from making their way to the ship hulls being colonized in thousands of ports worldwide.

So, in the meantime, fouling is an inevitability.

First contact

Barnacles and tubeworms are some of the most prominent and visible biofouling organisms that gather on marine surfaces, but the sticky microbial films that they share space with almost always beat them to the hulls.

"When a new surface is put in the water — for example, a brand-new, newly painted ship — it's colonized by microbes very rapidly," says Benjamin Van Mooy, a chemical oceanographer at Woods Hole Oceanographic Institution. Once attached, the microbes quickly establish a slimelike coating. "That's like a green, slippery film, and it's all made of microbes," he says.

Like many biofilms, the slime

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IMAGE COURTESY OF WIKIMEDIA COMMONS

A Phoenician ship carved on the face of a sarcophagus that dates from the 2nd century.

IMAGE COURTESY OF MICHAEL HADFIELD

The calcarerous tubes created by Hydroides elegans

CONTINUED FROM PAGE 13

provides a matrix for signaling and other interactions among bacteria, and it is held in place by a sticky, transparent polysaccharide glue. But because of the diversity of bacterial species in the film, not all of those interactions are in the best interest of a bacterium's neighbors.

"For every group of organisms that's trying to get a competitive advantage by signaling and coordinating by signaling, there's also another group of organisms that's trying to get a competitive advantage by disrupting the signaling of their competitors," explains Van Mooy.

The members of that bacterial community, which can include photosynthetic cyanobacteria, diatoms and other heterotrophic bacteria, all have evolved adaptations for a relatively stationary life on a surface. "They're not the same microbes that are floating around in the water, so it's not like the ship hull is flypaper," says Van Mooy.

The slime coatings are "the Achilles heel of antifouling and fouling-release coatings," says Mike Schultz, professor and director of the hydrodynamic laboratory at the U.S. Naval Academy. Even if a coating can shear organisms off at 10 knots, it doesn't do much good when a ship is in port for months at a time, which is standard operating procedure in ports from Norfolk, Virginia, to Oahu, Hawaii.

Teeming with tubules

Hyroides elegans is a tubeworm that would love to settle down on the underside of a ship's hull. In their mature form, the tubeworms, also known as hydroides, form tubes made of calcium carbonate that are a major contributor to biofouling in temperate waters around the world.

According to Michael Hadfield, a professor at the University of Hawaii at Mānoa, these calcareous "squiggly tubes" beleaguer dock workers tasked with maintaining the ships at Pearl Harbor. He says that port workers report the tubes "build up on the big propellers of an aircraft carrier a layer a couple of centimeters deep in a matter of one to two months."

The tubeworms have a quick

lifecycle and are capable of settling down to begin developing their crusty tubes within five days of hatching from an egg. The metamorphosis from a free-swimming filamentous larvae to a stationary, plume-crested adult begins when a tubeworm is jabbed by a nanometer-scale harpoon fired by Pseudoalteromonas luteoviolacea, a bacterium sometimes found in marine biofilms.

Once hit by a bacterial harpoon, a tubeworm quickly secretes a mucus strand to tether itself to the surface, followed by a primary tube made up of proteins and fibers. Within this tube, the tubeworms undergo metamorphosis and swap out the bands of cilia that had allowed them to swim and feed for a large plume of tentacles. After completing metamorphosis, the worms build a calcified tube that sticks perpendicularly to the surface of the ship or dock.

If the juvenile tubeworms are unable to find a surface, they can remain as larvae for seven weeks or longer. This relative longevity is essential, as the interactions between hydroides and the harpooning bacteria occur by chance, in the complete absence of signaling molecules from the bacteria.

The lack of signaling molecules wasn't confirmed until Hadfield and his colleagues published a paper in Science in 2014. Up to that point, "people were under the impression that bacteria were just producing passive molecules," says Nicholas Shikuma, an assistant professor at San Diego State University who was a co-author on that paper. "We figured out that the bacteria were producing these very complex structures that are syringelike," says Shikuma. He and colleagues hypothesize that these nanometer-scale syringes poke the tubeworm larvae and act as "the stimulant for settlement and metamorphosis."

While P. luteoviolacea isn't the only bacteria that can induce hydroides to undergo metamorphosis, it is the only bacteria known to use the syringe mechanism. In the journal Scientific Reports, Hadfield and his colleagues recently described the mechanisms that other common members of marine biofilms use to spur hydroides metamorphosis.

Regardless of which bacteria help to initiate it, the entire metamorphosis process for the tubeworms takes less than 12 hours, and before long the mature tubeworms are ready to start reproducing again. Under ideal circumstances, the turnaround time for a generation is about three weeks. The worms are prolific breeders too. "The females will spawn over and over, and they spawn about 2,000 eggs every time they do," says Hadfield. "They're little reproductive machines."

Combined with the surge in global shipping routes, this has made the tubeworms' presence both ubiquitous and genetically indistinct in major ports around the world.

"This sort of warm-water biofouling community is just about uniform all the way around the world," says Hadfield. "These organisms have been spread by ship traffic around the world since the late 1400s."

Blistering, bleeding barnacles

Barnacles are arguably the organisms most associated with biofouling, in part because the hundreds of different species colonize a wide variety of marine surfaces, ranging from rocks and docks to whale fins. Their interior base and stalks are hidden by hard, calcified plates and encircled by a cuticle made of chitin.

Barnacles begin their lives as one-eyed larvae that consist of a head and tail. The barnacles swim, eat and pass through a series of five molting phases, winding up as antennae-wielding larvae in search of homes.

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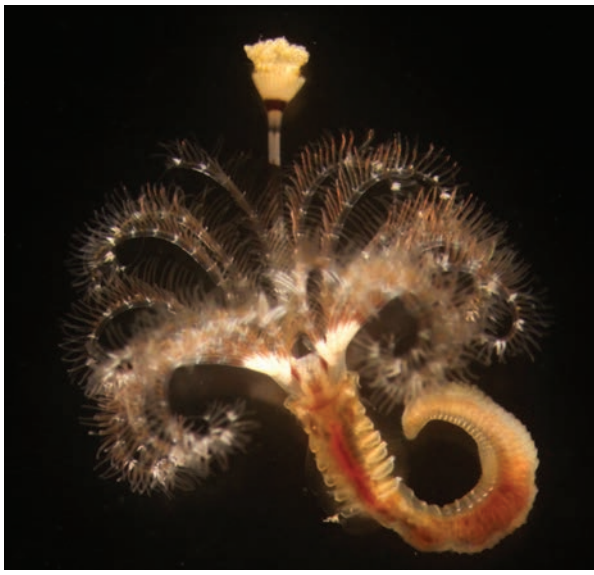

IMAGE COURTESY OF MICHAEL HADFIELD

An adult Hydroides elegans tubeworm

Barnacles on the propeller and rudder of a ship

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During this exploration process, the barnacles produce an adhesion protein that alerts them to a suitable surface. Once a surface has been found, "the larvae will stop exploring, settle and send out signals to attract other larvae," says Julius Vancso at the University of Twente in The Netherlands. "Then, a colonization of the surface starts."

After a barnacle has settled, it will eventually grow and molt, producing a new cuticle in a manner similar to an insect producing a new exoskeleton. As this cuticle widens, it cuts into both the underlying surface and the barnacle, causing the crustacean to bleed into the space below. The blood forms a clot, which sticks to the underlying surface. As the molting continues, the barnacle also forms open-ended capillaries whose secretions spot-weld the crustacean in place to the surface beneath.

While the blood is flowing, "all the enzymes you can imagine in your blood clotting system are there," says Daniel Rittschof at Duke University, who helped pioneer models for studying barnacle growth. This immune response helps to wipe out the bacterial populations near the barnacle. Some species of barnacle also calcify the area beneath them to stick more firmly in place.

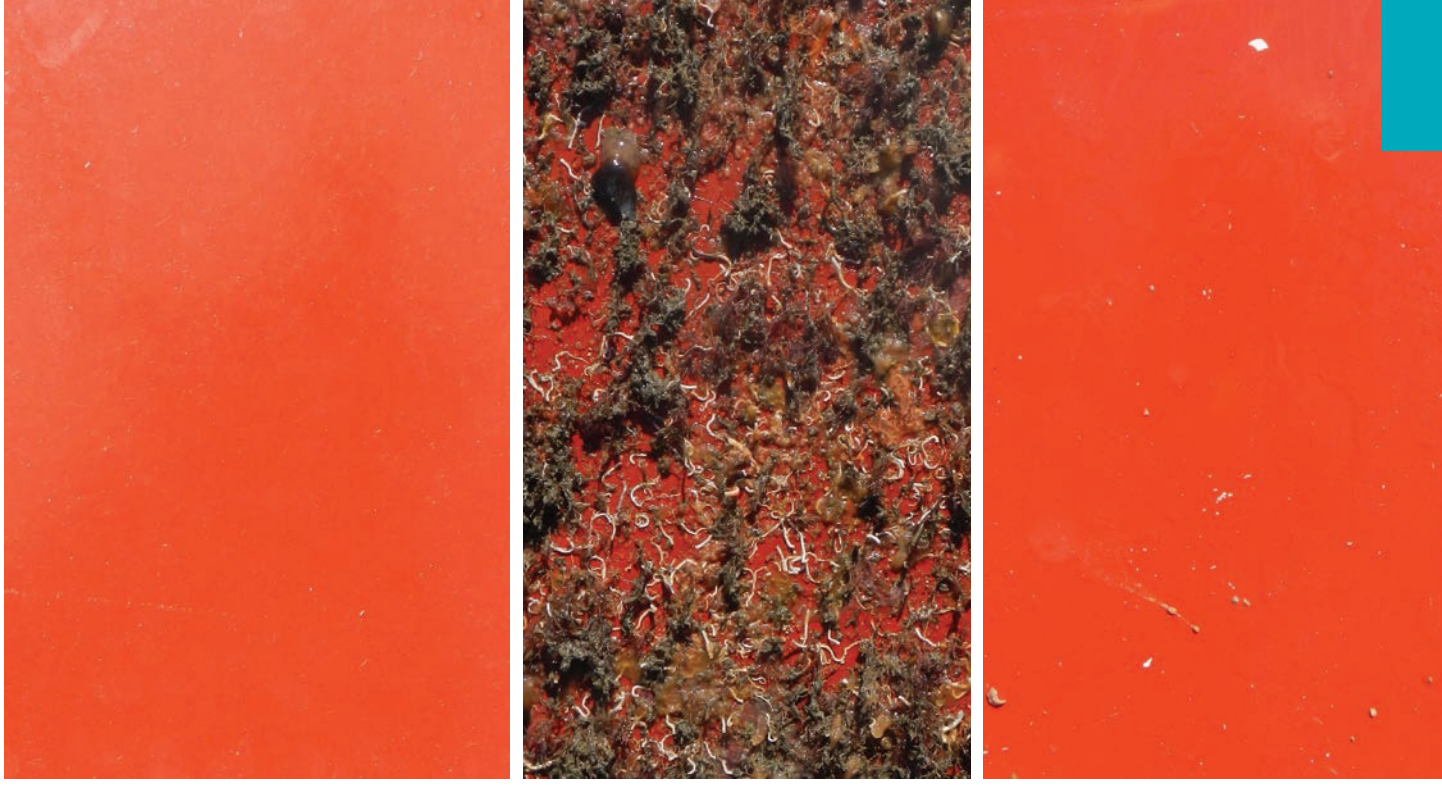
"It's like you've cut yourself. You've got a scab, and your body reworks underneath that scab," says Rittschof. "In the barnacles, the scab never falls off."

Kill or release

The global uptick in ocean travel following the 15th century spurred innovation for keeping ships free from biofouling organisms. Around the mid-18th century, the pitch-based mixtures that had been the most widely used coatings were supplanted by copper as the official standard for the United Kingdom's Royal Navy.

According to Stephen McElvany, the program officer overseeing the Office of Naval Research's antifouling and fouling release coatings program, the U.S. Navy uses two primary types of coatings, which are copper oxide-based ablatives and silicone-based foul-release coatings.

The copper-oxide coating is a remnant of the older way of tackling biofouling that Rittschof calls "mix and kill." In this method, biocidal compounds that leach into the water



IMAGES COURTESY OF KELLI HUNSUCKER AT THE FLORIDA INSTITUTE OF TECHNOLOGY

From left: A foul-release surface; same surface after seven months in a marine environment; and immersed surface after a cleaning that sloughs off marine organisms

surrounding the ship are mixed into the paints applied to the hull.

Copper primarily works by poisoning metabolic pathways, such as photosynthesis, says Van Mooy. The pathway is essential for the cyanobacteria and diatoms that make up a marine biofilm. On commercial vessels, this approach reached an apex of sorts with organotins, which are compounds based on tin linked with hydrocarbons.

At concentrations on the microgram level, organotins, which were highly effective at keeping hulls clean, work by interfering with an organism's energy production. However, at thousand-fold lower concentrations, the compounds cause female mollusks to transform into males, which had devastating effects on the French oyster fishery in Archaron Bay in the 1970s. The compounds, which weren't ever implemented in U.S. Navy ships, were banned in 2001 by the International Maritime Organization's International Convention on the Control of Harmful Anti-fouling Systems on Ships.

The less harmful silicone coatings, which are used in both military and commercial vessels, are rubbery and similar to caulk coatings in our homes.

"It's not exactly like your bathtub, but what happens then is the fouling that gets on there is easy to peel off," says McElvany. "If a barnacle sits on there, you can just peel it off easier than (if it were on) a steel surface or a copper paint surface." Once a ship leaves port and hits speeds of 10 to 20 knots, he says, the hydrodynamic motion of the water against the ship hull shears off the film and crust.

While they're more environmentally friendly than the copper ablative coatings, silicone paints are more expensive and less durable. When some species of barnacles move in and the surfaces get scratched, the easy-release properties diminish.

"Barnacles are famous for digging under softer coatings and getting a grip that way," says Anne Meyer at the University of Buffalo. "They actually cut into the coatings."

However, if barnacles can be prevented from attaching altogether, the cutting and glomming processes can't damage coatings. Working with colleagues at the Agency for Science, Technology and Research in Singapore, which is known as A*STAR,Vancso used atomic force microscopy to determine the isoelectric point of the adhesion protein that

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PHOTO COURTESY OF GLENN BATUYONG

A ship receives a high-power wash to remove barnacles and other marine hitchhikers.

CONTINUED FROM PAGE 17

barnacles use to stick to surfaces.

The researchers can use this information to engineer a surface that exploits electrostatic attraction to interfere with barnacle adhesion. "We've reasoned that if we can control the adhesion between this adhesion protein … and eventually prevent that adhesion, then these larvae would explore, see nothing and then die, because they stay alive in the metamorphic cycle for a couple of days," says Vancso.

This type of surface and the experimental coatings being pursued by the Office of Naval Research both belong to a third category of coatings that seek to make ship surfaces essentially invisible to the organisms by exploiting their biochemistry.

According to McElvany, these experimental coatings include polymers that are amphiphilic, containing both hydrophobic and hydrophilic entities on their polymer chains. These each repel certain swaths of fouling organisms. Other coatings involve zwitterions, which cause a positive and negative charge separation at a distance of two or three carbon atoms. This charge separation creates a highly ordered water layer at the paint surface due to hydrogen bonding with the water molecules, which prevents organisms, such as barnacles, from sensing and settling on the hull.

But the experimental coatings are just that, for the time being. The coating that exploits isoelectric points developed by Vancso's colleagues at A*STAR has been tested only on small substrates under controlled laboratory conditions. "It is not uncommon for the technology to take decades, 10 or 20 years, to develop," says Anbanandam Parthiban, a colleague of Vancso's at A*STAR.

For one or all

Biofouling continues to exact a cost on vessels in a number of ways. (See box "Scenes inside tubes.") "Mariners have been faced with ship-hull fouling since they went to sea, and so it just kind of goes to show what an incredibly enduring problem this is, that it's persisted for centuries," says Van Mooy, who is exploring antifouling solutions for deep-sea sensors used in scientific research.

"Various antifouling strategies have been tried, but there's been no magic bullet," he says. "I wouldn't say 'boutique antifouling' methods, but I think that certain types of shipping are going to have to have certain types of fouling-release and antifouling formulations that are best suited to what they're doing."

Like many of his colleagues, Vansco is concerned about the matter of specificity versus targeting all biofouling organisms with a single coating in the future.

"The next question is, all right, we've prevented attachment of barnacles, which is an important fouling species, but what about other fouling species?" says Vancso. "Can we ever have a generic recipe for a universal coating which, if you would apply to the hull of a ship, would prevent attachment of all these fouling species? And that's a big question that still needs some answering."

Scenes inside tubes

Biofouling is also a problem in the coolant pipes of a ship's engine. "If you get too much barnacle growth in your inlet pipes, then you can restrict the flow of coolant, which will limit how fast you can run until you can clean it out," says Vic Raines, a chief engineer with American President Lines, an international container shipping and ocean freight provider.

The primary method of dealing with clogged engine tubes is overengineering. Raines gives the ship's cooling system as an example. "Cooling is one of those things that only really matters when you run out of it," he says. Through overengineering, or building more pipes than the engine requires to be cooled at its highest speeds, the cooling system can afford to lose efficiency due to barnacles choking off pipes.

Other methods of dealing with fouling organisms in the coolant pipes include running electrical currents through copper plates to release ions and using electrical currents to generate sodium hypochlorite, also known as bleach, from the oxygen and sodium chloride in sea water.

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ANNUAL MEETING HIGHLIGHTS

ASBMB '17

ANNUAL MEETING

CHICAGO | APRIL 22 – 26

PROGRAM AT A GLANCE

Saturday, April 22

8:30 a.m. – 4:30 p.m.
Graduate Student and Postdoc Career Development Event
Advance registration required
11 a.m. – 4 p.m.
Undergraduate Orientation and Poster Competition
Advance registration required
4 – 5:15 p.m. & 6:30 – 7 p.m.
Science Outreach Posters
4:15 – 5:15 p.m.
Exploring Careers Speed-Networking Event for Undergraduates
5:30 – 6:30 p.m.
Herbert Tabor Research Award Opening Lecture
7 – 8:30 p.m.
EB Welcome Reception

Sunday, April 23

8:45 – 9:45 a.m.
ASBMB Award Lectures
10 a.m. – 12 p.m.
Scientific Symposia, Issues in Depth, Education & Professional Development
12 – 2:30 p.m.
Posters, Networking, and Meet the Speakers
12:30 – 1:30 p.m.
ASBMB Award for Exemplary Contributions to Education Award Lecture
1 – 3 p.m.
Tang Prize Award Lecture and Reception
2:30 – 5:45 p.m.
Spotlight Sessions, Education & Professional Development
6:15 – 7:45 p.m.
Learning Labs & Success in Science Workshop Series
7:30 - 9 p.m.
ASBMB Welcome Reception
Sponsored by the Minority Affairs Committee

Monday, April 24

8:45 – 9:45 a.m.
ASBMB Award Lectures
10 a.m. – 12 p.m.
Scientific Symposia, Issues in Depth, Education & Professional Development
12 – 2:30 p.m.
Posters, Networking, and Meet the Speakers
12:30 – 2 p.m.
Advocacy town hall
Presented by the Public Affairs Advisory Committee
2:30 – 4 p.m.
Alice and C.C. Wang Award in Molecular Parasitology Symposium
2:30 – 5:45 p.m.
Spotlight Sessions, Education & Professional Development
6:15 – 7:45 p.m.
Learning Labs & Success in Science Workshop Series
7 p.m. and 9 p.m.
Nothing Academic: A Night of Science-Themed Improv
9 – 11 p.m.
Young Experimental Scientists (Y.E.S.) Mixer

Tuesday, April 25

8:45 – 9:45 a.m.
ASBMB Award Lectures
10 – 12 p.m.
Scientific Symposia, Issues in Depth
12 – 2:30 p.m.
Posters, Networking, and Meet the Speakers
12:30 – 2 p.m.
NIH Workshop: Funding Opportunities
2:30 – 5:45 p.m.
Spotlight Sessions
6:15 – 7:45 p.m.
Learning Labs & Success in Science Workshop Series
7:30 – 9 p.m.
Women Scientists Mentoring and Networking Event

Wednesday, April 26

8:45 – 9:45 a.m.
ASBMB Award Lectures
10 – 12 p.m.
Scientific Symposia
12 – 2:30 p.m.
Posters, Networking, and Meet the Speakers

Rare bacterial outbreak in the Midwest

By Hailey Gahlon

In November 2015, a deadly outbreak of a rare bacterial species began in the U.S. Midwest. The affected states included Wisconsin, Illinois and Michigan. The outbreak led to 20 deaths of elderly or immunocompromised persons; it remains unclear whether the bacterial infection alone or a combination of the infection and pre-existing health conditions was the cause of death. The culprit was a little-known bacterial species called Elizabethkingia anophelis.

A group of researchers from Peter Hoyt's laboratory at Oklahoma State University now are reporting on the transposon genome rearrangements that are unique to the Elizabethkingia strains from the outbreak.

According to the Centers for Disease Control and Prevention, the outbreak was a rare occurrence, as only a few cases of E. anophelis typically are confirmed in the U. S. every year.

From November 2015 to January 2016, 67 cases were reported to the Wisconsin Division of Public Health; of those, 63 were confirmed. With the outbreak spreading across state lines, the CDC began an investigation last year to understand the outbreak better. They found that the median age of the patients infected was 72 years old, and 47 percent were female. The infections presented predominantly in the blood. They resulted in sepsis and were highly resistant to several antibiotics.

To this day, the source of the outbreak remains unknown. There is no evidence to link the outbreak to contamination in health-care products, food, water or patient-to-patient transmission.

Starting research on Elizabethkingia

The genus of Elizabethkingia first was described in 1959 by bacteriologist Elizabeth O. King while she was working at the CDC; the bacteria were named in her honor. Elizabethkingia are Gram-negative bacteria commonly found in soil and water. The strain responsible for the outbreak, E. anophelis, first was isolated in the midgut of the Anopheles gambiae mosquito from the McCarthy Islands in Gambia in 2011. Before that time, only two other species of Elizabethkingia had been proposed based on 16S rRNA sequence similarity studies, E. meningoseptica and E. miricola. In 2011, another species, E. endophytica, was reported in some specimens of sweet corn. All Elizabethkingia strains isolated to date are resistant to many common antibiotics.

The Hoyt lab was interested in Elizabethkingia years before the outbreak. Hoyt explains his department head, John Gustafson, got him interested in these bacteria. At the time, the Gustafson group was isolating bacteria that cause mastitis in dairy cows. The Hoyt lab was interested in trying to separate the different species of Elizabethkingia; it's still unknown how many species may exist in the genus.

Hoyt says that he found the genomes of Elizabethkingia intriguing. According to him, the bacterial genes effectively move like "liquid;" they are highly mobile and able to rearrange rapidly.

Studying the outbreak strains

Hoyt explains that he and his colleagues read about the outbreak and knew that the CDC would get involved. Gustafson contacted the CDC and was put in touch with Ainsley Nicholson, a microbiologist, who had been working on sequencing the outbreak strains and looking for underlying genetic clues as to what caused the outbreak. Nicholson provided the Hoyt laboratory with some of the outbreak strains.

Rita Flores, a graduate student in the Hoyt lab, and colleagues identified a homologous double transposon region in a few of the strains from the outbreak. Their data suggested that these DNA rearrangements were recent genetic events. The conserved homologous region was about 63 kilobases in length and was flanked by typical mobile genetic elements.

Further, there was variation in the length of the conserved region depending on the strain. For example, the shortest conserved region was found in the E. anophelis PW2809 strain, while the longest was found in E. anophelis NUHP-1, miricola ATCC and the four strains from the outbreak. Importantly, the location of these transposons in the CDC outbreak strains was different from other strains. The affected genetic location occurs in a gene coding for an A/G-specific adenine glycosylase, which is much like the E. coli MutY that is involved in DNA repair. This genetic change in the outbreak strains could have resulted in aberrant glycolytic function or increased mutagenic potential.

Additionally, Flores and colleagues identified a toxin–antitoxin complex unique to the strains given to them by the CDC. This toxin–antitoxin, along with the disruption in the adenine glycosylase function, could have increased the survival of E. anophelis, possibly underlying the phenotypic changes associated with the outbreak strains.

The aftermath of the outbreak

Hoyt explains that some event,

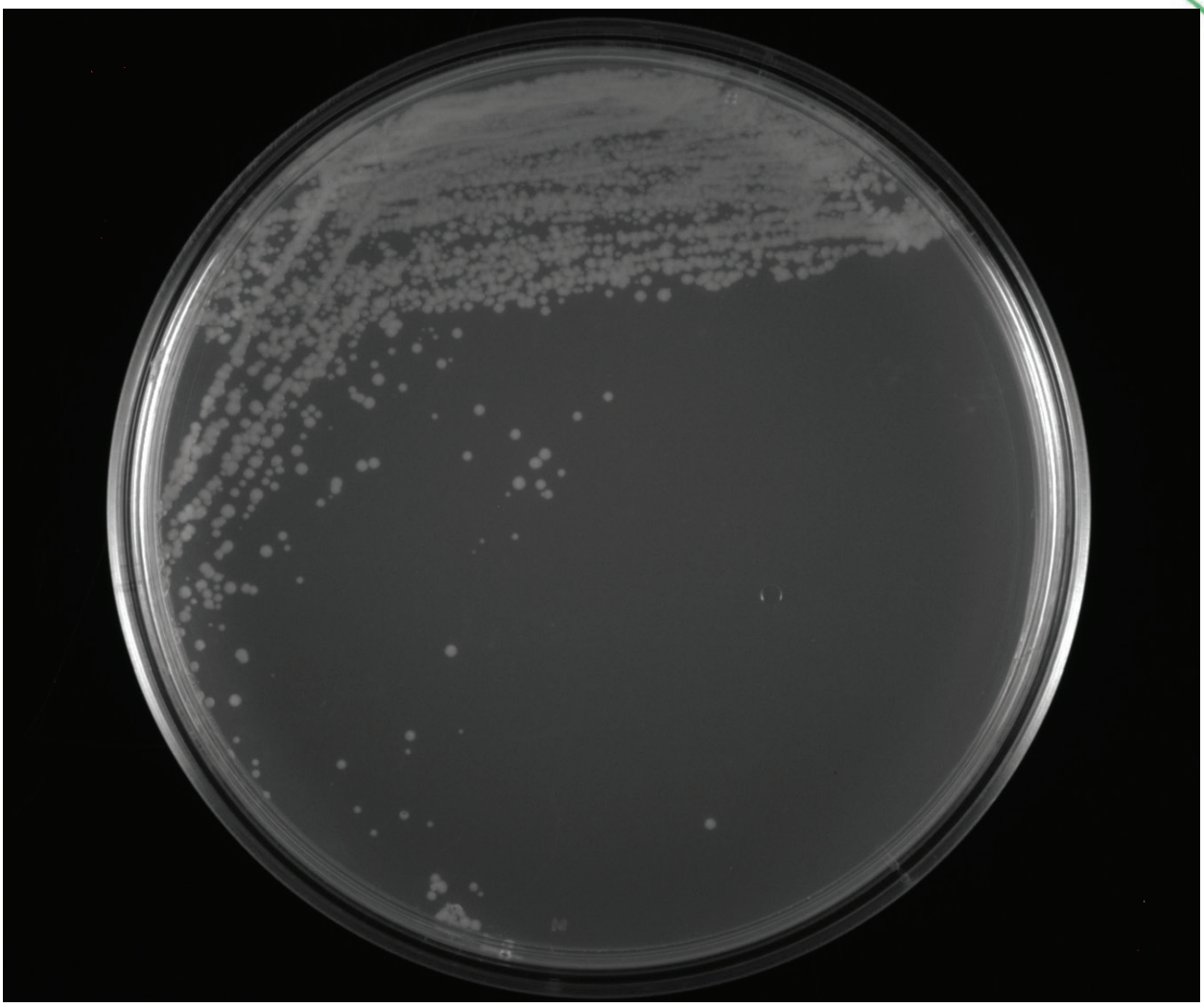


IMAGE COURTESY OF PETER HOYT

A Petri dish wih Elizabethkingia anophelis growing on it

such as altered gene expression or the acquisition of new toxins, enabled the bacteria to mutagenize rapidly. Further, Hoyt notes that Elizabethkingia are essentially "reservoirs of antibiotic resistance" for other bacteria, as most bacteria have the ability readily to take up DNA from other bacteria in their surrounding environment. The relatively ubiquitous presence of Elizabethkingia, coupled with their ability easily to rearrange their genome, provide the first clues regarding how E. anophelis caused the recent outbreak. Using high-throughput sequencing, the CDC was the first to confirm in patient samples that the E. anophelis strain was responsible for the outbreak occurring in the Midwest.

The Hoyt laboratory now is interested in understanding more about the evolutionary mechanisms of the bacteria that allow them to recombine so frequently. By analysing the sequences of the bacteria, the investigators found a high frequency of antibiotic-resistance genes as well as genes involved in creation of mobile genetic elements. These features could afford the bacteria increased opportunities for recombination and may have driven the observed cases from the outbreak.

Flores notes that no cases have been reported since last June and that the outbreak appears to have subsided.

Despite these advances in understanding Elizabethkingia, it still is not understood how or where the bacteria were able to infect their immunocompromised hosts. But the work in Hoyt's and other laboratories is helping researchers better understand how bacteria like Elizabethkingia drive deadly outbreaks.

The researchers will present their results at the 2017 ASBMB Annual Meeting at the 1:15 p.m. poster session on April 23 in McCormick Place in Chicago.

Hailey Gahlon (h.gahlon@imperial.ac.uk) is a Marie Curie postdoctoral research fellow at Imperial College London.

Accelerating evolution to break open a toxin

By John Arnst

Vomitoxin makes animals — surprise — vomit. The fungal toxin also causes gastroenteritis, anorexia and immunosuppression. The compound is produced by the fungi Fusarium graminearum and Fusarium culmorum, which can colonize wheat and similar crops. While regulations by the U. S. Department of Agriculture prohibit its presence in food produced for human consumption at one part per million, a lack of similar restrictions for animal produce results in weight loss and death among pigs and cattle. Contamination normally leads to the destruction of infected crops. To come up with a solution, researchers at San Diego State University are using directed evolution to transform an enzyme found in Pseudomonas aeruginosa to degrade vomitoxin, which is formally known as deoxynivalenol.

Cif, shorthand for cystic fibrosis conductance regulatory inhibitory factor, is a virulence factor secreted by P. aeruginosa. The bacterium is especially dangerous when it colonizes the lungs of people with cystic fibrosis. As its name would suggest, the Cif enzyme plays a key role in this process.

The Cif enzyme is a member of the epoxide hydrolase class of enzymes that break open the carbon–oxygen epoxide ring of many small organic molecules by converting the oxygen bonds into two hydroxyl groups with the addition of a water molecule.

Vomitoxin's structure contains an epoxide ring, which made the Cif enzyme an appealing choice for the researchers. "Our goal is to evolve this enzyme into a new enzyme that can bind and degrade the mycotoxin compound vomitoxin," says John Love. Love is the primary investigator on the work that will be presented at the meeting by his graduate student, Myung Soo Ko.

IMAGE COURTESY OF COMMONWEALTH SCIENTIFIC AND INDUSTRIAL RESEARCH ORGANISATION, AUSTRALIA

Blight in barley crops caused by Fusarium graminearum

"Another reason why we chose this enzyme is because its catalysis takes place in two steps," says Love. "It's a pretty standard mechanism."

In the first step, the enzyme forms a covalent bond with the substrate. Amino acids at the enzyme's active site then continue the catalysis and break bonds within the substrate, which, in this case, is the epoxide ring.

However, the Cif enzyme wasn't made to bind to vomitoxin's epoxide ring, so the researchers took to mutating its active site until it would.

In this process, "you randomize the DNA of the gene that codes for Cif," says Love. "That creates diversity, so you have thousands, if not billions, of enzymes that are pretty much the same but have different amino enzymes in the active sites."

That vast pool of mutated enzymes requires a filtering process, however, so the researchers developed a system known as bacterial surface display, or BSD. The system uses a combination of fused protein elements, anchoring transmembrane proteins and the fluorescent protein mCherry to drive the expression of a desired protein on the outer membrane of Escherichia coli. To express the Cif protein in this manner, the researchers cloned its gene into expression plasmids and transformed them into E. coli, where the protein was directed to the outer membrane by a leader sequence in the plasmid. Once the mutated enzymes were anchored there by the BSD protein elements, the researchers centrifuged the bacteria and conducted the catalytic analysis on the bacterial surface using the standard epoxide substrate epibromohydrin.

The researchers plan to evolve the enzyme to hydrolyze progressively larger epoxy substrates rather than seeking a mutation that immediately would cause the Cif enzyme to digest vomitoxin.

"Instead of hitting the home run, we're hoping that we're going to hit singles along the way," says Love. "At the end of the project, we'll have an enzyme that can successfully hydrolyze the epoxide deoxynivalenol."

The researchers will present their results at the 2017 ASBMB Annual Meeting at the 12:30 p.m. poster session on April 25 in McCormick Place in Chicago.

John Arnst (jarnst@asbmb.org) is ASBMB Today's science writer. Follow him on Twitter at twitter.com/arnstjohn.

An evening of science-themed improv

By Geoff Hunt

On April 24, comedian Mike Abdelsayed will lead a team of improv professionals from One Group Mind to put on "Nothing Academic: A Night of Science-Themed Improv" at The Comedy Clubhouse in Chicago. The improv comedy show, which is sponsored by the Public Outreach Committee of the American Society for Biochemistry and Molecular Biology, will present an example of science communication in action.

The POC constantly is looking for innovative ways to engage ASBMB members in communication and outreach activities, so the idea for doing a science-themed improv event at the 2017 ASBMB Annual Meeting made sense, given the location. "One perk of attending the ASBMB annual meeting is getting the chance to explore a new city!" says POC member Niki Woitowich, a Chicago resident. "Chicago is the birthplace of improvisational comedy, and this event allows ASBMB meeting attendees to get a feel for the Windy City through its rich cultural scene."

Another POC member, Teaster Baird Jr., adds that "using this medium makes science and scientists more accessible and real to the public."

To help the ASBMB put on the "Nothing Academic" event and connect with comedian Abdelsayed and his team, the POC reached out to the Chicago Council on Science and Technology, known as C_2ST. C_2ST is a nonprofit STEM education group for adults with a long track record of running events in the Chicago area. C_2ST Director of Programs and Public Relations Andrea Poet was immediately on board with the idea of a science-themed improv show. "We wanted to do something different than a traditional lecture, something that represented a fun way to communicate science," she says.

PHOTO COURTESY OF ONE GROUP MIND

Members of the One Group Mind improv comedy collective perform.

Fun is one thing, but how does improv actually relate to science? As Abdelsayed sees it, science has "become a far more collaborative effort, one that requires communication with each other, teamwork." That, he points out, "is what improvisation is. It's essentially teamwork on steroids."

For the POC, this improv event represents a perfect complement to the committee's more formal science-communication efforts, which include themed training workshops and the online training course "The Art of Science Communication."

So what can prospective attendees expect from the "Nothing Academic" event? According to Abdelsayed, improv players from The Comedy Clubhouse will use suggestions, based on science, from audience members to build a long-form improvisational piece around that topic. But, says Abdelsayed, given the unpredictable nature of improv, "we'll probably (also) have some interactive stuff and mix it up a bit."

The "Nothing Academic" event is part of a series of outreach activities organized by the POC at the ASBMB annual meeting that help bring science to the public. For Woitowich, these events represent an opportunity for scientists to take part in outreach activities. "We can have a significant impact on the cities we visit every year by providing programming both for our attendees as well as the local community," she says.

Baird takes it a step further. "Given today's political climate," he says, "I think it's even more important for us as scientists to get out in the general public to remind them, and ourselves, that we are citizens of humanity, just like them, who want to make our communities better."

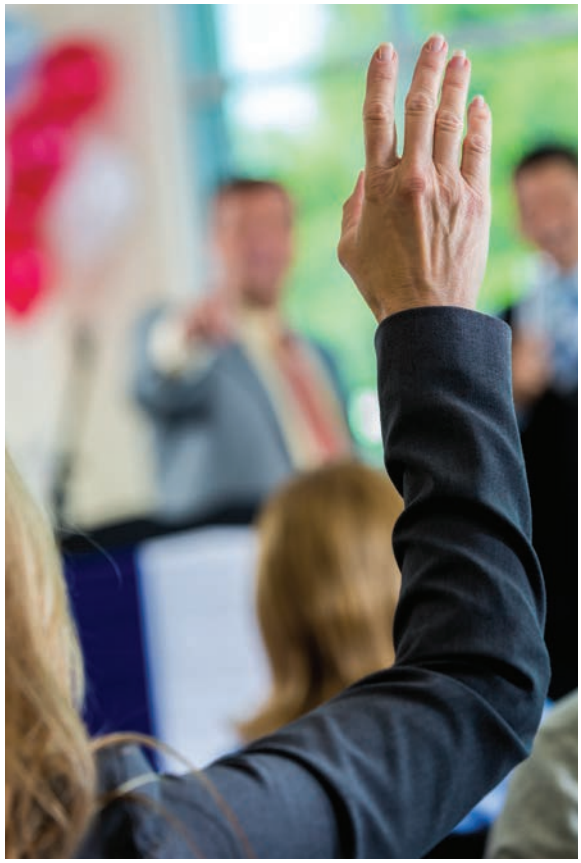
To purchase tickets for the event, visit www.ticketsource.us/event/169512.

Geoff Hunt (ghunt@asbmb.org) is the ASBMB's public outreach manager. Follow him on Twitter at twitter.com/thegeoffhunt.

Advocacy town hall

By Benjamin Corb

This year, the American Society for Biochemistry and Molecular Biology's Public Affairs Advisory Committee is trying something new at the 2017 ASBMB Annual Meeting in Chicago — an advocacy town hall. The past 12 months have seen many upheavals, mainly in the form of the 2016 election and its aftermath. The scientific enterprise has been caught up in the upheavals as well, with President Donald Trump's executive orders on immigration and his administration's handling of issues such as the Affordable Care Act, climate change and vaccine safety.



In light of these events, it is fair to assume that there is a renewed interest in science policy and the impact that Washington, D.C., is having on the scientific enterprise. To help those interested in understanding better how the government's decisions affect science, we have taken that old slogan, "You've got questions, we've got answers," to heart in designing an open-ended town hall.

The PAAC put together a panel of experts who are prepared to answer your questions and provide insights on the topics and issues that are of greatest importance to you. The PAAC chair, Wes Sundquist from the University of Utah, will update attendees on the ASBMB's efforts on sustaining the biomedical enterprise. Michael Lauer, the deputy director of extramural research at the National Institutes of Health, will answer grant policy questions. As the public affairs director for the ASBMB, I work regularly on the front lines of science policy as your voice to policy makers, so I too will be on the panel.

The town hall will start with brief presentations from Sundquist, Lauer and me, but the rest of the event will be driven by the questions you bring to us. Have questions over how a specific piece of legislation will affect your lab, or how the community is working to improve training for graduate students and postdoctoral researchers? Bring them! Have concerns over funding levels and pay lines? Bring them, too!

Besides your questions, we also will discuss how the ASBMB does advocacy on behalf of its members and what role you can play in these efforts. We will discuss our annual August advocacy campaign, which brings scientists to their elected state officials. We will describe the resources with which the ASBMB can provide you to help you be the best advocate you can be. For example, we can gather data to support your case and give you online training tools designed specifically with the scientist in mind.

With our event scheduled just days after the country-wide March for Science, we hope you will bring your energy and enthusiasm to this session and learn how we can help you to continue to have your voice heard in the U.S. Congress year-round. We want to build on the energy from the March for Science events and turn that energy into action.

Not able to attend the meeting? We'll have some people cover the event on Twitter as it unfolds, so even if you're not in Chicago, you'll be able to participate online. Search for the hashtag #PolicyTownHall. You will be able to follow the conversation and ask questions to panelists.

This is the first time the ASBMB has designed an interactive advocacy event like this during the annual meeting. We're hoping your enthusiastic participation will help us to ensure that this event becomes a staple at ASBMB annual meetings.

The advocacy town hall will be at 12:30 p.m., April 24 in room W184d in McCormick Place.

Benjamin Corb (bcorb@asbmb.org) is director of public affairs at the ASBMB. Follow him on Twitter at twitter.com/bwcorb.

Continue the conversation on Twitter!

By Allison Frick

You've just attended one of the new and fascinating spotlight sessions at the American Society for Biochemistry and Molecular Biology's annual meeting. You're inspired and want to find out what other attendees are saying about the session. What's one easy way to keep the conversation going and learn more about an area of biochemistry and molecular biology that interests you?

It's hashtags.

This year, we've assigned hashtags to specific topics to help attendees who share common interests connect on Twitter. When you send a tweet, be it a comment or a picture, simply include the relevant hashtag. That will add your tweet to the stream of tweets with the same hashtag. Depending on whether your Twitter account is set to private or public, including a hashtag will allow anyone who clicks on or searches for that hashtag to see your thoughts and join in the conversation.

On the right is a handy list of the hashtags. Remember to follow @ASBMB and use #ASBMB2017. We'll also be posting on Twitter information about lectures, workshops and spotlight sessions to keep you up to date on the meeting.

Allison Frick (africk@asbmb.org) is the ASBMB's print and digital media specialist. Follow her on Twitter at twitter.com/allisonfrick.

#antibiotics	Antibiotics
#bigtalks	Award lectures
#microbes	Bacteria, viruses, microbiome, infectious disease
#ASBMBed	Biochemistry education
#enzymes	Catalysis, enzyme mechanism
#cellbio	Cell and organelle dynamics
#cellsignal	Cell signaling, signal transduction, post-translational modification
#chembio	Chemical biology
#chromatin	Chromatin, epigenomics
#DNA	DNA replication, recombination and repair
#omics	Genomics, proteomics, metabolomics
#lipids	Lipids and membranes
#metabolism	Metabolism
#plants	Plant biochemistry and molecular biology
#profdev	Professional development
#proteins	Protein folding, aggregation, chaperones
#proteins	Protein synthesis, translation, proteolysis, degradation
#RNA	RNA, processing, transcription, noncoding RNA
#glyco	Sugars, glycomics, glycans, starches
#profdev	ASBMB workshops
#PolicyTownHall	Science policy

Annual Awards

ASBMB AWARD FOR EXEMPLARY CONTRIBUTIONS TO EDUCATION

Dolan recognized for 'transformation of teaching and learning'

By Adriana Bankston

Erin Dolan, the Georgia Athletic Association Professor for innovative science education at the University of Georgia, won the 2017 American Society for Biochemistry and Molecular Biology Award for Exemplary Contributions to Education. The award recognizes those who encourage effective teaching and learning of biochemistry and molecular biology.

Daniel Leahy at the University of Texas at Austin said in his nomination letter that Dolan's "commitment to evidence-based transformation of teaching and learning is visionary."

As the former executive director of the Texas Institute for Discovery Education in Science at UT Austin, Dolan led the Freshman Research Initiative, the nation's largest university undergraduate research program that gives first-year students the opportunity to engage in research with faculty and graduate students through a three-semester lab course. In addition to guiding the FRI, she established the Texas Institute for Discovery Education in Science, an institute for education innovation with the mission to enhance the college's leading role in science, technology, engineering and mathematics education. TIDES is focused on student programs that foster experiential and engaged learning, development programs for STEM faculty, and studies determining the effectiveness and impact of education programming.

Paula Lemons, who supported Dolan's nomination, is a fellow instructor of the Introduction to Biochemistry course Dolan teaches at UGA. Lemons noted that Dolan uses "learning objectives to guide student work, case studies and other in-class activities to get students' minds engaged and challenged, and constructed-response assessments that demand deep understanding and problem solving."

"I am honored and humbled to be recognized by my colleagues and the society with the award. It is exciting to see an influential organization like ASBMB reward efforts to promote teaching in ways that are consistent with how people learn."

— ERIN DOLAN

Dolan's contributions to teaching reflect her desire to help faculty become better teachers. She participated in the inaugural meeting of a national initiative to define threshold concepts in undergraduate biochemistry education.

She also took part in a think-tank meeting to promote course-based undergraduate research experiences in biochemistry and molecular biology. This resulted in a report published in CBE: Life Sciences Education in 2014. In this report, Dolan and her colleagues delineated what made course-based undergraduate research experiences meaningful. According to Sarah Elgin of Washington University in St. Louis, who wrote a nomination letter, this paper "provides a framework for thinking about assessment, about outcomes we might hope for and could measure. I believe this paper has had a very significant impact on the field."

Dolan also supports biology education research in her role as editor-in-chief of LSE. Kimberly Tanner of San Francisco State University, a founding editorial board member of LSE who also nominated Dolan for the award, writes of Dolan's "unwavering" focus on evidence-based understanding of science education. She also notes Dolan's ability to "raise the level of research in the field of biology education by coaching and not shaming, supporting and not dismissing, individuals aspiring to participate in the emerging field of discipline-based biology education research."

Dolan earned her Ph.D. at the University of California, San Francisco. She began teaching biochemistry at Virginia Tech and then moved to UGA. She then was the executive director of TIDES before returning to the UGA.

Dolan will receive her award during the 2017 ASBMB Annual Meeting in Chicago, where she will deliver an award lecture. The presentation will take place at 12:30 p.m. April 23 in room W184bc in McCormick Place.

Adriana Bankston (abankston81@gmail.com) is a former scientist with a passion for improving training and policies for junior scientists.

BERT AND NATALIE VALLEE AWARD IN BIOMEDICAL SCIENCE

Evans recognized for gene-expression research

By Aditi Dubey

Ronald M. Evans, a professor and the director of the Gene Expression Laboratory at the Salk Institute for Biological Studies in La Jolla, California, and a Howard Hughes Medical Institute investigator, has won the 2017 Bert and Natalie Vallee Award in Biomedical Science given by the American Society for Biochemistry and Molecular Biology. Evans won the award for his contributions to the field of nuclear hormone signaling and metabolism. His work has advanced significantly our understanding of the molecular processes that underlie obesity-related diseases, such as diabetes.

The award was established by the Vallee Foundation in 2012 to encourage creativity, originality and leadership in biomedical research. Both Bert and Natalie Vallee were prominent scientists who conveyed their passion for biomedical research through their work and teaching. The award honors scientists who have made outstanding international accomplishments in basic biomedical research.

Evans earned his B.A. from the University of California, Los Angeles, where he also later obtained his Ph.D. He went on to do postdoctoral work with James E. Darnell at The Rockefeller University, where he worked on mechanisms of mRNA transcription and translation. In 1978, Evans joined the Salk Institute, where he continued his research on regulation of gene expression by fat-soluble hormones as well as studying basic cellular metabolism of sugars and fats. His work has informed much of our current understanding of the role of nuclear receptors in both normal and disease physiology.

"I am honored to be selected as the recipient of the 2017 Bert and Natalie Vallee award in Biomedical Science. While research can become a world unto itself, the Vallee Award is a reminder that science and discovery has a very human side that touches us all."

— RONALD M. EVANS

Evans' notable achievements include the identification and characterization of the first nuclear hormone receptor, the human glucocorticoid receptor. His group subsequently identified more than 50 members of the nuclear receptor superfamily and their ligands. These include PPARγ and PPARδ, which are critical for fat metabolism, the thyroid-hormone receptor, the vitamin-D receptor and the retinoid-X receptor. In addition, his work has provided a basis for differential regulation of gene expression by nuclear hormone receptors: Evans' lab has demonstrated that subtle changes in cDNA of nuclear hormone receptors are sufficient to alter their respective ligands, co-activators and repressors, which in turn are capable of altering the set of downstream genes activated in conditions of normal physiology or disease.

"His achievements and contributions in research, albeit in a very different focus area, have inspired me and further taught me to think innovatively, insightfully and tirelessly," wrote Vivian Tang, a graduate student at the University of Western Australia, in her nomination letter.

Work done by Evans has led to the establishment of principles of DNA recognition and receptor heterodimer formation and to the discovery of the genetic code for hormone response. Several new drugs for the treatment of diseases such as cancer, obesity, respiratory distress syndrome and diabetes have been developed using his discoveries.

Evans has been honored with several prestigious awards, including the Albert Lasker Basic Medical Research Award, the Gairdner International Award, the Alfred P. Sloan Medal and the Wolf Prize in Medicine. Evans was elected to the National Academy of Sciences in 1989 and to the American Academy of Arts and Sciences in 1997. In 2005, he received the Grand Medaille, the highest honor of the French National Academy. Evans is also the March of Dimes chair in molecular and developmental biology and was awarded the March of Dimes prize in developmental biology in 2003.

Evans will receive his award during the 2018 ASBMB Annual Meeting in San Diego, where he will deliver an award lecture.

Aditi Dubey (dubeyad@nyu.edu) is a postdoctoral associate at New York University.

WALTER A. SHAW YOUNG INVESTIGATOR AWARD IN LIPID RESEARCH

Fairn recognized as 'one of the most promising lipid researchers'

By Adriana Bankston

Gregory Fairn, an assistant professor at the University of Toronto and staff scientist at St. Michael's Hospital in Toronto, won the 2017 Walter A. Shaw Young Investigator Award in Lipid Research for his discoveries in the field of lipid biology.

In his letter supporting Fairn's nomination for the award, David Byers at Dalhousie University said, "Greg Fairn has developed from one of the very best graduate students I have encountered to one of the most promising lipid researchers. He is rapidly becoming a leader in the lipid-research field not only through his scientific excellence and creativity but also by virtue of his positive attitude, collegiality and work ethic."

Fairn's career began in the lab of Christopher McMaster at Dalhousie University, where he investigated the regulation of lipid metabolism and vesicular transport in the trans-Golgi using yeast genetics. According to McMaster, who nominated Fairn for this award, Fairn "made a major breakthrough in our understanding of how cells target lipids and proteins to organelles within the cell." During his graduate studies, he published 11 papers, six of them as first author.

Continuing his work in the field of lipid biochemistry during his postdoctoral studies, Fairn examined negatively charged lipids and "uncovered a critical role of these lipids in the regulation of cell polarization, phagocytosis and macropinocytosis," said Vanina Zaremberg at the University of Calgary, who supported Fairn's nomination for this award. During this time, Fairn became proficient in conducting studies of lipid biology in mammalian cells using biophysical techniques in the laboratory of Sergio Grinstein from the Hospital for Sick Children and the University of Toronto. Grinstein also supported Fairn's nomination for the award. "While he learned virtually all we have to offer, I think we learned even more from him," said Grinstein in his letter of support.

"It is a great honor to be nominated for this award by colleagues and mentors who helped shape my career. Walt and Avanti Polar Lipids have been very active and supportive of lipid research over the years. I am truly humbled to receive the Walter Shaw Young Investigator Award. I'd like to thank ASBMB for the award, and the great mentors, colleagues, trainees and funding agencies for their work and support over the years."

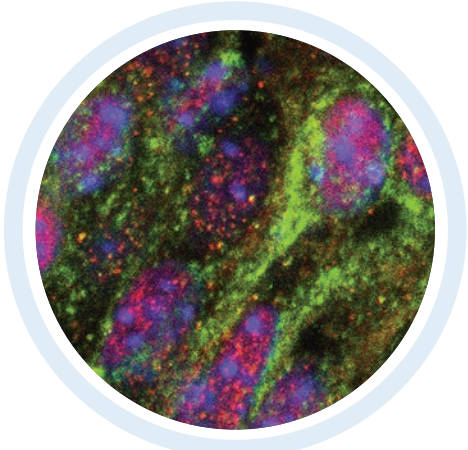
— GREGORY FAIRN

In his own laboratory at the University of Toronto since 2012, Fairn continues to "lead the lipid field with his pioneering discoveries on the interdependence of phosphatidylserine and cholesterol intracellular distribution and movement," according to Zaremberg. His laboratory recently generated a fluorescent cholesterol sensor suitable for live-cell imaging and electron microscopy. The biosensor has "enormous potential and will surely become a widely used tool in the near future," said Grinstein.

Fairn also has earned numerous awards attesting to his contributions to the field of lipid biochemistry, such as the Exceptional Trainee Award from the Hospital for Sick Children, a New Investigator Award from the Canadian Institutes for Health Research and an Early Research Award from the province of Ontario.

Fairn will receive his award during the 2017 ASBMB Annual Meeting in Chicago, where he will deliver an award lecture. The presentation will take place at 11:40 a.m. April 23 in room W183c in McCormick Place.

Adriana Bankston (abankston81@gmail.com) is a former scientist with a passion for improving training and policies for junior scientists.



ASBMB–MERCK AWARD

Frydman's protein-folding work defines 'the forefront'

By Adriana Bankston

Judith Frydman, a professor at Stanford University, has won the 2017 American Society for Biochemistry and Molecular Biology–Merck Award. The award recognizes outstanding contributions to research in biochemistry and molecular biology.

Frydman has made discoveries in chaperone-mediated protein folding and protein quality control. Her work uncovered basic principles of chaperone function during de novo protein synthesis as well as during quality control, when protein misfolding occurs. Correct folding of cellular proteins is a fundamental problem in biology and is essential to human health. "Her work encompasses an impressive string of path-breaking discoveries," said Peter Walter of the University of California, San Francisco, who nominated Frydman for this award.

Frydman's postdoctoral mentor, Ulrich Hartl at the Max Planck Institute of Biochemistry, recounts in his nomination letter how Frydman identified and characterized the eukaryotic cytosolic chaperonin complex TRiC/CCT system, including its structure and mechanism, in a series of publications from his laboratory. Frydman's identification of TRiC as a eukaryotic chaperonin similar to, but distinct from, bacterial GroEL was a "tour-de-force and elucidated the fact that protein folding begins co-translationally in eukaryotic systems," said the late Susan Lindquist of the Massachusetts Institute of Technology in her nomination letter of Frydman for this award. Hartl also said, "These studies are among the finest demonstrations of how one can dissect the mechanism of a complex macromolecular machine with a range of methods from biophysics, biochemistry and genetics."

"It is such an honor to receive the Merck award. Our work highlights how an incredibly elaborate network of chaperones controls and monitors every aspect of the life of proteins in the cell, from birth to death. The sheer elegance and complexity of this machinery, and its relevance for therapies for many misfolding diseases, make for an exciting challenge in years to come."

— JUDITH FRYDMAN

In her own laboratory at Stanford, Frydman is characterizing the process of protein quality control in eukaryotic cells. In a 2008 Nature paper, Frydman's group showed that eukaryotic cells have distinct quality-control compartments that channel misfolded proteins to different cellular fates. This work relies on the sequestration of misfolded proteins into spatially and functionally distinct compartments, including a compartment that serves as a reservoir for subsequent protein repair/ubiquitination followed by proteasomal degradation and another compartment that sequesters terminally aggregated proteins.

Frydman's work on protein homeostasis is also instrumental, as dysfunction of this machinery is associated with a growing number of human diseases. According to the late Lindquist, "her work holds important implications for a number of human diseases that result from misfolding of proteins, in particular neurodegenerative diseases."

Frydman has approached her studies with an "enormous intellectual rigor and dedication to quality, and with a remarkable sense for biologically significant questions," said Hartl. Walter further acknowledges that Frydman's work encompasses "an innovative, comprehensive and most elegant integration of molecular chaperone function with cellular physiology and pathologies." He adds that Frydman's "work continues to define the forefront of a rapidly growing field, and her contributions mark significant milestones of our collective progress."

Frydman has a Ph.D. in biochemistry from the University of Buenos Aires in Argentina. Frydman's honors include the Distinguished Young Scholars Award from the W.M. Keck Foundation and the Merit Award from the National Institute of General Medical Sciences. She has organized and chaired a number of conferences on protein folding and homeostasis.

Frydman will receive her award during the 2017 ASBMB Annual Meeting in Chicago, where she will deliver an award lecture. The presentation will take place at 8:45 a.m. April 26 in room W183ab in McCormick Place.

Adriana Bankston (abankston81@gmail.com) is a former scientist with a passion for improving training and policies for junior scientists.

HERBERT TABOR RESEARCH AWARD

Gottesman wins for post-transcriptional regulation work

By Mariana Figuera-Losada

Susan Gottesman of the National Cancer Institute has won the 2017 Herbert Tabor Research Award for her work on post-transcriptional regulation in bacteria. The Tabor award recognizes outstanding scientific accomplishments, excellence in biological chemistry and molecular biology, and significant contributions to the community of scientists. The award is named after the Journal of Biological Chemistry's former longtime editor at the National Institutes of Health. Gottesman's work has unveiled the role that energy-dependent proteolysis plays in protein turnover and how small regulatory RNAs influence regulatory circuits. Her research has shown that bacteria possess complex mechanisms of post-transcriptional gene regulation and inspired similar research in eukaryotes.

Early on, Gottesman and collaborators defined the regulatory mechanisms for energy-dependent proteolysis during stress. They used Escherichia coli to construct variants. These constructs allowed them to identify the Lon ATP-dependent protease as a regulator of bacterial capsule synthesis, cell division following DNA damage, and bacteriophage lambda development via degradation of regulatory proteins.

Collaborations with Michael Maurizi and Sue Wickner at the NCI allowed Gottesman to identify the ATP-dependent Clp proteases and the chaperone function of their ATPase domains. Recently, she and her collaborators have shown that regulated proteolysis controls the levels of the transcription factor RpoS in response to stress and various growth conditions. Degradation of RpoS requires ClpXP protease and an adaptor protein that delivers RpoS to ClpXP; under stress conditions, anti-adaptor proteins interfere with adaptor function. Several of these anti-adaptor proteins have been shown to be expressed differently to specific stresses.

"I am extremely honored to receive the Herbert Tabor Research Award from ASBMB and to have my name associated with Herb Tabor as well as the impressive list of previous winners. Herb Tabor is the ultimate example of someone who has, throughout his career, combined service to the scientific community with excellent science in a way that the rest of us can only hope to emulate."

— SUSAN GOTTESMAN

Gottesman and her team also found that the expression of RpoS protein is a highly regulated process that depends on novel small RNAs. DsrA is one of these small RNAs that activates the translation of RpoS at low temperatures. RprA and ArcZ are two other small RNAs that have been described that also function positively to regulate RpoS translation.

Lawrence E. Samelson at the NCI pointed out in his nomination letter that a number of parallels can be drawn between Tabor's career and that of Gottesman. Both developed their groundbreaking work at the NIH and demonstrated an unmatched commitment to the service of the scientific community. In their letters of support for Gottesman's nomination, Bonnie L. Bassler at Princeton University and Tina M. Henkin at Ohio State University highlighted the fact that Gottesman selflessly has encouraged the advancement of their careers despite not having been their official mentor. This shows Gottesman's commitment to being a "leader, a teacher and a mentor in the truest sense," wrote Bassler.

Gottesman has served on the boards of the American Society for Biochemistry and Molecular Biology, the Genetics Society, the American Academy of Microbiology and the National Academy of Science as well as the Howard Hughes Medical Institute's scientific advisory board. She was elected to the National Academy of Sciences in 1998 and the American Academy of Arts and Sciences the following year.

Gottesman earned her Ph.D. at Harvard University and moved to the NIH to pursue a postdoctoral fellowship. In 1974, she became a research associate at the Massachusetts Institute of Technology and, in 1976, returned to the NIH as a senior investigator. She later became chief of the biochemical genetics section at the NCI and co-chief of the laboratory of molecular biology.

Gottesman will receive her award during the 2017 ASBMB Annual Meeting in Chicago, where she will deliver an award lecture. The presentation will take place at 5:30 p.m. April 22 in the Skyline Ballroom W375c in McCormick Place.

Mariana Figuera–Losada (fmariana@hotmail.com) is an associate scientist at Albert Einstein College of Medicine.

AVANTI AWARD IN LIPIDS

Haucke's work has "high impact" in membrane biology

By Courtney Chandler

Volker Haucke, professor of molecular pharmacology and director at the Leibniz Institute for Molecular Pharmacology in Germany, has won the Avanti Award in Lipids from the American Society for Biochemistry and Molecular Biology. Haucke is being recognized for defining the roles of membrane lipid homeostasis in cellular transport.

Haucke's "findings are of key importance for cell physiology and pathophysiology," said Britta Brügger and Thomas Söllner at the Heidelberg University Biochemistry Center, who nominated Haucke for the award.

Haucke has made several contributions to defining lipid functions. Much of his research has focused on the role of lipids in exocytic and endocytic trafficking, specifically in neurotransmission. He has characterized specific lipid-binding endocytic adapters that recycle membrane proteins found in synaptic vesicles. These vesicles serve as stores for neurotransmitters until their release into nerve synapses. He further has defined the synaptic-vesicle membrane proteins as having roles in the coordinated processes of neurotransmitter release and synaptic vesicle regeneration.

Haucke's team also has investigated a group of phospholipids called phosphoinositides. The researchers described the cellular function of the phosphoinositide phosphatidylinositol-(3,4)-bisphosphate as a key player in clathrin-mediated endocytosis. Their work provided the first defined function for this lipid in cellular processes. It was an unexpected result that forced the lipid community to rethink what it knows about phosphoinositide involvement in this type of endocytosis.

"I feel truly honored to receive the Avanti Award in Lipids from the ASBMB for our work on phosphoinositides in membrane traffic and organelle identity. This is also an honor for the entire lab and its members, past and present. I am extremely grateful to a terrific group of people with whom I have had and have the privilege to work."

— VOLKER HAUCKE

In their nomination letter, Brügger and Söllner said Haucke "has contributed tremendously to our understanding of biological processes that are based on endocytosis, including the regulation of neurotransmission, demonstrating essential roles of lipids in organelle homeostasis."

Haucke's recent work focuses on how phosphoinositide metabolism affects endosomal processes. His research shows that spatial and temporal restrictions of phosphoinositide metabolism help to maintain membrane identities during exocytic and endocytic trafficking. Furthermore, he and his colleagues found that regulation of this metabolism is critical for cargo delivery to the cellular surface. This finding and subsequent research have led to a new model for intracellular membrane transport in which phosphoinositides play a key role in determining organelle identities.

Haucke's findings have therapeutic potential. "Targeting phosphoinositide-metabolizing enzymes might open new therapeutic avenues for the treatment of diseases such as cancer and a disorders," said Brügger and Söllner.

In her letter of support for Haucke's nomination, Lois Weisman at the University of Michigan described Haucke as "an outstanding researcher who has had a tremendous impact on current understanding of the roles of phosphoinositide lipids in membrane traffic."

Haucke, an elected member of the European Molecular Biology Organization, earned his Ph.D. in biochemistry from the University of Basel and did postdoctoral work at Yale University and the Howard Hughes Medical Institute. He was recruited as an independent group leader at the University of Göttingen, and then he moved to the Freie Universität Berlin to serve as full professor of biochemistry. In 2012, he was recruited to his current position. He is also a faculty member of the Freie Universität Berlin, where he holds a professorship for molecular pharmacology.

Haucke will receive his award during the 2017 ASBMB Annual Meeting in Chicago, where he will deliver an award lecture. The lecture will take place at 8:45 a.m. April 25 in room W183ab in McCormick Place.

Courtney Chandler (cochandl@umaryland.edu) is a graduate student at the University of Maryland, Baltimore.

RUTH KIRSCHSTEIN DIVERSITY IN SCIENCE AWARD

Robinson honored for helping disadvantaged high-school students

By Rajendrani Mukhopadhyay

Douglas N. Robinson, professor of cell biology at the Johns Hopkins University School of Medicine, won the 2017 Ruth Kirschstein Diversity in Science Award for developing the Summer Academic Research Experience program. The SARE program helps disadvantaged teenagers in inner-city Baltimore get experience in biomedical research as well as academic tutoring. The American Society for Biochemistry and Molecular Biology's Minority Affairs Committee selects the winners of this award.

Named after Ruth L. Kirschstein of the National Institutes of Health, the award honors outstanding scientists who are committed to helping underrepresented minorities enter the scientific enterprise and thrive within it. Kirschstein was renowned for science and public service. After working on the polio vaccine, Kirschstein became the first woman to direct an NIH institute, the National Institute of General Medical Sciences. Later, she served as deputy director and acting director of the NIH. Kirschstein was an advocate for training, particularly for underrepresented minorities.

Robinson studies how different cells take on their distinctive shapes. By understanding the fundamental principles that underlie cell morphology, Robinson's team aims to parse out how cell shape influences diseases such as pancreatic cancer, chronic obstructive pulmonary disease and degenerative motor neuron diseases.

But Robinson and his team also work to bring people into the laboratory who otherwise wouldn't have the privilege of doing research. About 34 percent of children in Baltimore grow up in poverty, which is almost three times higher than the national average. Robinson developed SARE nine years ago as a way for promising but disadvantaged students to get trained in academic and professional skills as well as to build a network of mentors.

"Science provides innovative solutions for health, environmental and technological challenges. Often overlooked is that science can and should provide career opportunities to everyone, regardless of their socioeconomic background. Dr. Kirschstein paved the way opening doors for all through science. I am deeply honored to be recognized by this award."

— DOUGLAS N. ROBINSON

Robinson got the inspiration for SARE when he and his wife, Lisa Naeger, began taking Sunday dinners to the Boys Hope house. The house, and the subsequent Girls Hope house, are supported by the Boys Hope Girls Hope organization, which provides at-risk children with homes, education, and financial and emotional support. At the dinners, Robinson and Naeger heard the teenagers express interest in research. After bringing two teenagers into his laboratory, Robinson's group decided to formalize the opportunity in the form of SARE.

Of the 37 scholars who have come through SARE to date, 23 have reached college age and matriculated into four-year colleges with partial or full scholarships. Half of the students have chosen science, engineering, math or health-related majors.

Peter Devreotes, the director of the department of cell biology at Hopkins, explained in his nomination letter that Robinson won a Health Career Opportunity Program grant through the Health Resources and Services Administration to expand the scope of SARE to the greater Baltimore area as well as the rest of the country. In the expanded format, the program also now serves disadvantaged undergraduates and postbaccalaureate students.

In his letter supporting Robinson's nomination, Bill Bement at the University of Wisconsin–Madison wrote, "Doug Robinson is one of an incredibly rare group: full-time researchers who nevertheless make major education contributions well beyond the expectations that come with a faculty position." Bement closed his letter by saying that Robinson was "an ideal candidate" for the award.

Robinson will receive his award during the 2017 ASBMB Annual Meeting in Chicago, where he will deliver an award lecture. The presentation will take place at 9:15 a.m. April 25 in room W183ab in McCormick Place.

Rajendrani Mukhopadhyay (rmukhopadhyay@asbmb.org) is the managing editor of ASBMB Today. Follow her on Twitter at twitter.com/rajmukhop.

DELANO AWARD FOR COMPUTATIONAL BIOSCIENCES

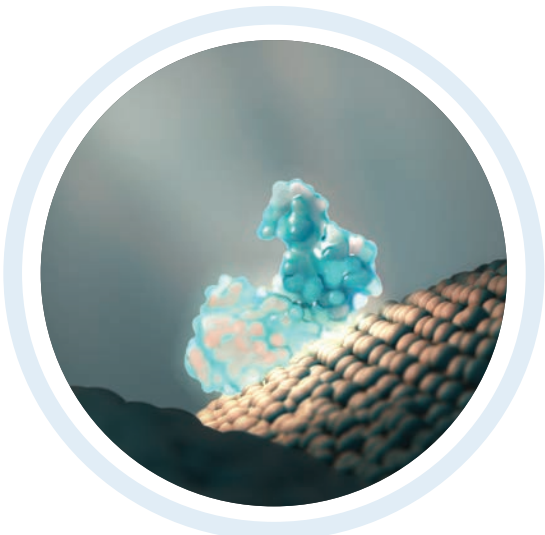
Shoichet wins award for informatics tools for drug discovery

By Dawn Hayward

Brian Shoichet, a professor of pharmaceutical chemistry at the University of California, San Francisco, won the 2017 American Society for Biochemistry and Molecular Biology DeLano Award for Computational Biosciences. Shoichet works on the development of docking methods and screening libraries and applies these tools in particular to G-protein–coupled receptors.

The Shoichet lab seeks to improve on traditional docking methods with new tricks of the trade. His lab has generated several distinct binding pockets that mimic typical protein cavities with which a promising small molecule could dock. This allows even failed predictions to help in the search for targets that match each small molecule tested.

In addition, the Shoichet lab works with libraries containing millions of small molecules, looking to enhance these libraries with the addition of bio-relevant molecules. This elevates molecular docking projects to the level of traditional high-throughput screening methods.



"My reaction to getting the award was excitement to have the work of the lab recognized, and melancholy remembering Warren Delano and his contributions. He came out of UCSF's graduate program and made important scientific contributions as well as his contributions to open science. He was an ever more central member of our community at the time of his untimely death."

— BRIAN SHOICHET

A key test of these methods has been on GPCRs. The Shoichet lab, in collaboration with Bryan Roth of the University of North Carolina, has characterized several orphan GPCRs, including GPR68. This work utilized another aspect of Shoichet's toolbox, the chemical probe, which identified GRP68's role in fear-based learning.

In his letter nominating Shoichet, UCSF colleague Matthew Jacobson wrote, "Brian is distinct in that he has extended the reach of computational modeling to study protein-ligand interactions with enormous impact on drug discovery and enzymology."

As the DeLano Award celebrates the development and dissemination of computational tools, Andrew Lee Hopkins of the University of Dundee noted in his letter of support for Shoichet's nomination that Shoichet indeed fits the bill. "In the spirit of the ASBMB DeLano Award, Professor Shoichet has made multiple informatics platforms of his research readily accessible to the scientific community to accelerate their own research," he wrote.

Shoichet earned his bachelor's degree in chemistry from the Massachusetts Institute of Technology and his Ph.D. in medicinal chemistry from UCSF. After postdoctoral fellowships at UCSF and the University of Oregon, Shoichet held a faculty position at Northwestern University before being recruited back to UCSF.

Shoichet won the PhRMA Foundation Career Award and the National Science Foundation Career Award. He also has served on the scientific advisory board for the National Institutes of Health RoadMap Chemical Libraries and Screening Initiative and the NIH Centers for Biomedical Computing.

Shoichet will receive his ASBMB award during the 2017 ASBMB Annual Meeting in Chicago, where he will deliver an award lecture. The presentation will take place at 9:15 a.m. April 26 in room W183ab in McCormick Place.

Dawn Hayward (dhaywar5@jhmi.edu) is a graduate student at the Johns Hopkins University School of Medicine.

ALICE AND C. C. WANG AWARD IN MOLECULAR PARASITOLOGY

Sibley recognized for research on the intracellular parasite Toxoplasma gondii

By Aditi Dubey

L. David Sibley, the Alan A. & Edith L. Wolff distinguished professor of molecular microbiology at Washington University School of Medicine in St. Louis, won the American Society of Biochemistry and Microbiology's Alice and C.C. Wang Award in Molecular Parasitology. Sibley is recognized for his work on mechanisms that help a pathogen adapt inside cells.

The Alice and C.C. Wang award recognizes scientists who have made seminal contributions to the field of molecular parasitology through novel and significant discoveries. The award honors the legacy of the renowned parasitologists C.C. and Alice Wang.

Sibley's group investigates the intracellular parasite Toxoplasma gondii. A common parasite associated with HIV, T. gondii is capable of infecting almost all warm-blooded animals, but is especially harmful to those with compromised immune systems. To most people, it is best known as the organism that causes toxoplasmosis, a condition that can be contracted through ingestion of oocysts shed by cats or consumption of undercooked meat harboring tissue cysts.

Sibley's achievements include elucidating how the organism invades the host and achieves virulence by outwitting the cellular pathways of the host cell. Sibley's work led to development of classical genetic maps for the parasite in order to dissect the genetic footprint and the molecular mechanism of its virulence. His work has advanced our understanding of processes such as cell motility and protein secretion and how these processes are regulated during host invasion by the parasite.

"I have known and admired Alice and C.C. Wang for their many contributions to the field of molecular parasitology over many years, and it is a real honor to be nominated for this award by my colleagues. I am humbled to be chosen among so many excellent candidates."

— L. DAVID SIBLEY

Not only has Sibley's research established T. gondii as a model system for molecular parasitology, but his work also has set a paradigm for understanding the biology of other pathogens, such as the malarial parasite.

Boris Striepen at the University of Georgia, wrote in his letter of support for Sibley's nomination, "David began these studies at a time when T. gondii was far from being a model system, in fact it was a rather obscure pathogen, studied by a handful of investigators around the world — he has maintained his position at the very cutting edge of that question for more than thirty years."

Sibley earned his doctorate at Louisiana State University, where he studied macrophage interactions with T. gondii. During his postdoctoral stint at Stanford University, he discovered that common strains consist of three abundant genotypes. After moving to his current institution, Sibley continued his work on T. gondii.

"It is his completely fearless approach, scientific excellence and rigor that has allowed David to use his expertise in molecular parasitology to cross disciplines and provide novel insights that are broadly relevant to understanding the outcome of host-pathogen interactions," said Christopher A. Hunter at the University of Pennsylvania's School of Veterinary Medicine, in his letter supporting Sibley's nomination.

Sibley is a recipient of the Burroughs–Wellcome Award in Molecular Parasitology, the Molecular, Cellular and Immunoparasitology Scientific Excellence Award from the American Society for Tropical Medicine and Hygiene and the Distinguished Investigator Award from Washington University School of Medicine.

"Sibley is an outstanding scientist regardless how he is measured, or who he is measured against," wrote Striepen. "He is one of the brightest stars of molecular parasitology and his accomplishments make him a natural candidate for the letter and spirit of the Alice and C.C. Wang Award."

Sibley's award lecture will take place during the 2017 ASBMB Annual Meeting in Chicago at 2:30 p.m. April 24 in W184a in McCormick Place.

Aditi Dubey (dubeyad@nyu.edu) is a postdoctoral associate at New York University.

EARL AND THRESSA STADTMAN DISTINGUISHED SCIENTIST AWARD

Taylor wins award for work on protein kinase A

By Dawn Hayward

Susan S. Taylor, professor of chemistry and biochemistry as well as pharmacology at the University of California, San Diego, has won the 2017 American Society for Biochemistry and Molecular Biology Earl and Thressa Stadtman Distinguished Scientist Award. Taylor has done pioneering structural studies of protein kinase A, revealing fundamental themes for all protein kinases.

In 1991, the Taylor group solved the structure of the catalytic subunit of PKA, which proved to be a prototype for nearly all other protein kinases. Her structural analyses uncovered detailed insights into the mechanisms of ATP and peptide binding and phosphate transfer as well as the roles of autophosphorylation and open versus closed conformations. These studies were complemented by her structural analyses for the type 1α and type IIβ regulatory subunits of PKA, which revealed how cAMP is recognized by two tandem binding sites linked allosterically.

The Taylor group then solved the structures of PKA holoenzyme. This work showed how the regulatory subunit inhibits the catalytic subunit by forming an R_2C_2 holoenzyme complex where two regulatory subunits block the activity of each catalytic subunit. Sequential and allosteric binding of cAMP to the regulatory subunit initiates conformational changes that lead to the dissociation of the catalytic activity of the C-subunit, which can then bind its substrate. P. Boon Chock of the National Heart, Lung and Blood Institute states that this molecular switch is a fundamental regulator of many biological events and defines a mechanism that is adapted in different ways by protein kinases.

Next, the Taylor group began comparing kinases and found another unifying theme: the hydrophobic spine that is present in all active protein kinases. This feature has proven invaluable for inhibitor development. In addition, Taylor collaborated with the late Roger Tsien of UCSD to measure cAMP levels and PKA activity in living cells. From this, she discovered a nuclear export signal in the protein kinase inhibitor.

"I was thrilled and deeply honored to receive the news from Natalie Ahn that I had been selected for the 2017 Earl and Thressa Stadtman Award. Although I have never worked in Dr. Earl Stadtman's lab, I have always considered him to be a close friend and would visit with him every time that I came to NIH. Earl and Thressa are both role models for future generations."

— SUSAN S. TAYLOR

With the later discovery of proteins known as AKAPs and the mechanism of allosteric activation of the RAF kinase, the Taylor group solidified themselves as leaders in the field. "Her research provides fundamental breakthroughs in our understanding of the catalytic action and regulation of protein kinases," states Chock.

The Stadtmans, for whom this award was named, exemplified scientific research, mentorship and science statesmanship. Mark A. Lemmon of the Yale University Cancer Biology Institute wrote in his letter of support for Taylor's nomination, "Susan Taylor was a frequent visitor to the Stadtmans' laboratories at (the National Institutes of Health), and indeed has described herself as 'a surrogate member of Earl's family.'"

Taylor received a bachelor's degree in chemistry from the University of Wisconsin–Madison and a Ph.D. in physiological chemistry from the Johns Hopkins University. She then held two postdoctoral positions at the Medical Research Council Laboratory of Molecular Biology in the U.K. and UCSD.

Among other awards, Taylor has won the ASBMB William C. Rose Award, an American Association for the Advancement of Science fellowship and the Federation of American Societies for Experimental Biology Excellence in Science Award. She also served as president of the ASBMB.

She was elected into the National Academy of Sciences, the National Academy of Medicine, and the American Academy of Arts and Sciences and served on the fellowships advisory panel for the Packard Foundation and the General Motors Cancer Research Foundation Awards Assembly.

Taylor will receive her award during the 2017 ASBMB Annual Meeting in Chicago, where she will deliver an award lecture. The presentation will take place at 9:15 a.m. April 23 in room W183ab in McCormick Place.

Dawn Hayward (dhaywar5@jhmi.edu) is a graduate student at the Johns Hopkins University School of Medicine.

ASBMB YOUNG INVESTIGATOR AWARD

Urban, a 'fearless young scientist' in protease research

By Courtney Chandler

Siniša (Sin) Urban, a professor of molecular biology and genetics at the Johns Hopkins University, received the American Society for Biochemistry and Molecular Biology's Young Investigator Award for his work on elucidating the mechanisms of intramembrane proteases.

Urban's research represents a relatively new field that has developed largely due to his work. "While many young faculty follow 'hot' trends, (Urban's) work is rigorous and extraordinarily innovative in an area that he himself created," Nobel laureate Carol Greider of the Johns Hopkins University said in her letter nominating Urban for the award.

Urban studies rhomboid proteins, a group of proteases present inside the lipid bilayer of cell membranes that cut other transmembrane proteins. In addition to characterizing the mechanisms of these unique enzymes, he also has identified roles for rhomboid proteases in protozoal pathogens. "Very few investigators work at the intersection of these difficult areas," explained Greider, "but what is clear from Dr. Urban's work is that it is an area of great current and future importance."

Despite being early in his career, Urban already has made numerous contributions to his field. Urban discovered and described the molecular function of the classical rhomboid gene during his graduate training in the Medical Research Council Laboratory of Molecular Biology in the U.K. For this discovery he has been awarded many prizes, including the Sandler Prize by the Genetics Society of America. Urban continued his work as a J.B. & Millicent Kaye Prize fellow in cancer studies in Cambridge before moving to Harvard University as a fellow for the Society of Fellows.

"The privilege of curiosity-driven research is its own reward, but I'm truly honored to have our work recognized in this way by our ASBMB colleagues and friends. This is especially inspiring for the younger members of our small and emerging intramembrane proteolysis field."

— SINIŠA URBAN

In 2006, he was recruited to the Johns Hopkins University, where he has expanded his work of revealing the molecular basis of rhomboid function and its role in disease. He has published papers in various journals, including Cell, Nature and Molecular Cell, that define the structure of rhomboid enzymes and describe a novel method to study enzyme kinetics in the membrane in real time. "He decided that achieving a real understanding of intramembrane enzymes required developing cutting edge biophysical approaches that were absent in his field," said Greider. For this work, Urban also was named a Blavatnik Foundation scholar and a fellow of the Packard Foundation.

These systems have allowed Urban better to investigate how intramembrane proteases function. He has proposed a mechanism that is driven by kinetics and not substrate binding affinity. This represents a paradigm shift in the understanding of this class of enzymes, which previously had been assumed to act in an affinity-specific manner.

Urban also investigates the role of rhomboid proteases in medically relevant parasites, such as those that cause malaria. Protozoal diseases are enormous health burdens and often lack adequate treatments. His research on the role of rhomboid proteases in disease progression represents a promising area for development of novel protozoal treatments.

Urban is a hands-on scientist and "spends half his time at the bench" according to Greider. He also "devotes time to teaching and mentoring, including serving as Director of Admissions for the Johns Hopkins Biochemistry, Cell, and Molecular Biology (BCMB) PhD program at the School of Medicine," said Greider, adding that Urban "will continue to contribute breakthroughs to our scientific community here and abroad."

Urban will receive his award during the 2017 ASBMB Annual Meeting in Chicago, where he will deliver an award lecture. The presentation will take place at 9:15 a.m. April 24 in room W183ab in McCormick Place.

Courtney Chandler (cochandl@umaryland.edu) is a graduate student at the University of Maryland, Baltimore.

WILLIAM C. ROSE AWARD

Wickner honored for contributions to cell biology

By Sapeck Agrawal

This year's recipient of the American Society for Biochemistry and Molecular Biology's William C. Rose Award is Bill Wickner, a professor of biochemistry and cell biology at Dartmouth Medical School.

Established more than three decades ago and named after a former president of the American Society of Biological Chemists, ASBMB's precursor, the Rose award recognizes outstanding contributions to biochemical and molecular biological research and a demonstrated commitment to the training of younger scientists.

"Wickner's biochemical studies are responsible for our first understanding of how proteins are transported across membranes in bacteria. His subsequent work has illuminated the processes of membrane fusion and inheritance, two fundamental problems in eukaryotic cell biology. He is a consummate biochemist who deserves to be recognized for his outstanding research contributions and extremely dedicated mentorship," said Suzanne Pfeffer of Stanford University in her nomination letter.

Wickner's scientific career, which spans over 40 years and has produced more than 200 publications, began with a bachelor's degree in chemistry from Yale University in 1967 and then an M.D. from Harvard Medical School. At Harvard, Wickner worked with "a pioneer in lipid biosynthesis, Eugene Kennedy," wrote Pfeffer.

Wickner conducted his postdoctoral work with yet another scientific legend, Arthur Kornberg, at Stanford University, where he co-discovered the role of an RNA primer in DNA replication along with Randy Schekman.

Wickner's next stop was a professorship at the University of California, Los Angeles, where his group made history by being the "first to reconstitute bacterial protein translocation into proteoliposomes using purified components. His lab, and that of Shoji Mizushima, showed that SecY, SecE and SecG form a basic unit of the translocon that can translocate a preprotein when supplemented with SecA and ATP. Importantly, Wickner also showed that protein translocation requires a membrane potential in bacterial cells," wrote Pfeffer.

"We tackled protein translocation and membrane fusion as reductionists, piggybacking on the genetics work of Beckwith, Yoh Wada, Emr, Stevens and Schekman, developing rapid quantitative assays with purified organelles and then reconstituting and exploring the biology with purified proteins and lipids. I'm deeply grateful to my brilliant lab mates and to my revered teachers, Gene Kennedy and Arthur Kornberg."

— BILL WICKNER

In 1993, Wickner moved to Dartmouth Medical School, where he served as chairman of the biochemistry department. His group researched mechanisms responsible for membrane fusion, this time in eukaryotic cells. According to Pfeffer, "one of Wickner's most important discoveries was that the so-called NSF (NEM-sensitive transport factor) protein functions to disassemble SNARE proteins after fusion to permit SNARE protein reutilization, rather than catalyzing membrane fusion per se. He carried out highly detailed biochemical analyses of the individual steps underlying the fusion of yeast vacuoles in vitro and in cells."

Wickner has won numerous honors and accolades, including election to the National Academy of Sciences in 1996, an American Cancer Society Faculty Research Award, a Guggenheim Fellowship, and a Merit Award from the National Institutes of Health. He also was elected to be a foreign associate of the European Molecular Biology Organization and a member of the American Academy of Arts and Sciences.

Embodying the true spirit of scientific education and collaboration and that of the William C. Rose Award, Wickner has been "an outstanding and prolific mentor" to a long list of budding and now successful scientists, which includes "46 postdoctoral fellows, 17 graduate students, and 13 technicians," Pfeffer said.

On his lab webpage, Wickner describes himself as "an over-aged postdoc" and a "lab rat" who simply loves "talking and doing science."

Wickner will receive his award during the 2017 ASBMB Annual Meeting in Chicago, where he will deliver an award lecture. The presentation will take place at 8:45 a.m. April 23 in room W183ab in McCormick Place.

Sapeck Agrawal (sapecks.srivastava@gmail.com) is a science writer.

MILDRED COHN AWARD IN BIOLOGICAL CHEMISTRY

Yang a "highly accomplished crystallographer"

By Courtney Chandler

Wei Yang, an investigator and section chief of structure and mechanism at the National Institutes of Health, has won the Mildred Cohn Award in Biological Chemistry from the American Society for Biochemistry and Molecular Biology. The Cohn award honors the first president of the ASBMB and recognizes scientists who have advanced our understanding of biological chemistry through physical methodologies. Yang received the award in recognition of her work on elucidating the structure and function of proteins involved in genome maintenance.

Philip Hanawalt of Stanford University wrote in support of Yang's nomination, saying he could "think of no person more deserving than Wei Yang for this recognition of a woman who has made substantial advances in understanding biological chemistry using innovative physical approaches."

Much of Yang's work has focused on structural characterization of proteins involved in DNA mismatch repair and translesion DNA synthesis. She has solved the crystal structures of bacterial MutL, MutS, MutH and DNA helicase II proteins in complex with their DNA substrates. This work, plus activity assays, serves as the foundation for understanding how the mismatch repair system recognizes and removes mismatched DNA to ensure high fidelity during DNA replication.

In collaboration with Roger Woodgate of the NIH, Yang's team determined the first crystal structure of a Y-family DNA polymerase complexed with a DNA lesion and engaging in bypass synthesis. Subsequently, in collaboration with Fumio Hanaoka in Japan, who discovered human Y-family DNA polymerase eta, or Pol eta, her team elucidated the molecular mechanism Pol eta uses to bypass ultraviolet-induced DNA lesions and avoid mutations and malignancy.

"I am thrilled and deeply humbled to receive the Mildred Cohn Award in Biological Chemistry. Dr. Cohn, an extraordinary scientist, mentor and former ASBMB president, was a pioneer and both a role model and inspiration to me and my generation of women scientists."

— WEI YANG

"She moves smoothly from one field to another," said Hanawalt in his letter, "always providing the insights that are derived from her understanding of fundamental crystallographic approaches."

In more recent work, Yang and her team used time-resolved crystallographic techniques to study the mechanism of DNA synthesis. This allowed them to construct the first detailed picture of phosphodiester bond formation by a human polymerase, which included a description of the transient recruitment of a magnesium ion and interactions needed for nucleotide addition.

"Dr. Yang is both an outstanding crystallographer and an outstanding biochemist," said Martin Gellert of the NIH in his letter of support for Yang's nomination. "This combination of talents has enabled her to obtain deep insights into several important biological systems in the general field of DNA repair and recombination."

Yang earned her Ph.D. from Columbia University in 1991. As a graduate student in Wayne Hendrickson's laboratory, Yang, along with Robert Crouch of the NIH, determined the first crystal structure of RNase H bound to its RNA/DNA substrate, thereby establishing how this protein removes the RNA primers made during DNA replication. She characterized the structure and function of the UvrD helicase. This work revealed a distinct role for UvrD helicase in mismatch repair in addition to its traditional role in repairing DNA lesions produced by ultraviolet light.

Yang went on to postdoctoral fellowships at both Columbia University and Yale University. In 1995, she was recruited to the NIH as a tenure-track investigator at the National Institute of Diabetes and Digestive and Kidney Diseases.

Yang is a member of both the National Academy of Sciences and the American Academy of Arts and Sciences. She has received the Dorothy Crowfoot Hodgkin Award from the Protein Society and the Bea Singer Young Investigator Award from the Gordon Research Conference on Mutagenesis and Carcinogenesis.

Yang's award lecture will take place at 8:45 a.m. April 24 in room W183ab in McCormick Place.

Courtney Chandler (cochandl@umaryland.edu) is a graduate student at the University of Maryland, Baltimore.

Focus on exposure

By Kaoru Sakabe

In past columns, I've made the point that figure preparation begins at data acquisition, but I haven't really explained my reasoning in depth. So here, I'll fill you in. Once you've snapped your picture or exposed your Western blot, that image becomes the version of record for your experiment. If the data you've collected is poor quality from the outset, your figure is already compromised.

One way to tell if you've nailed your image's acquisition parameters is to look at your image's histogram. Being able to interpret the histogram correctly can tell you if you can move forward with snapping the next picture of your mutant phenotype or if you need to tinker with the acquisition settings.

If you're a digital photography aficionado, you probably are very familiar with histograms and the information they contain. Here's a quick overview for those not yet accustomed to viewing them: A histogram of an image displays the distribution of pixels in the image, showing a graph of the number of pixels with a given intensity. For an eight-bit grayscale image, there are 256 possible intensities ranging from 0 (black) to 255 (white) for each pixel in the image. The histogram will not tell you how these pixels are distributed in space, just the distribution of the pixel intensity.

Ideally, you want the pixels to lie between the two extremes. This ensures that the fine details of your

images are captured. If the pixels are clustered at either end, you've likely oversaturated or underexposed your image. For example, aggressively adjusting the black levels of an immunofluorescence image to reduce the background eliminates hallmarks of a true experimental image. On the other hand, oversaturation leads to loss of fine details and makes it impossible to quantify the signal. Why? From the point of view of the detector, i.e., the camera or film, once it has recorded the maximum amount of signal, it cannot register any more. If you've hit the limit on either end of the histogram, the detector won't be able to tell you if a band or a cell feature is two times or 20 times more intense than a neighboring band or cell.

If you're acquiring images on a microscope or gel-documentation system, the hard part already is done for you, because these instruments typically show you the histogram of the image you've just acquired. If you are using film, take multiple exposures of your blot to make sure you are within the linear range of the signal so you can properly quantify it. Once you've scanned your film, you can use either Photoshop or ImageJ to look at the histogram of your image. A telltale blip at either end of the histogram will tell you that you need to adjust your acquisition settings or use a different exposure of your film (Figure 1).

The histogram is also useful in telling you if your image has been overadjusted during figure preparation. After you've adjusted the brightness or contrast settings of your image, make sure to check the histogram one final time. If the histogram has shifted too far to the left or to the right, you've likely truncated the pixels that were at the ends of the distribution, and your image is now overly adjusted (Figure 2). If your histogram shifts too far to either end, the resulting image may raise flags with reviewers or the journal, because it may look like you're trying to hide something. Remember, there's no need to hide your true experimental results!

Doing your due diligence at the image-acquisition phase will save you time as you prepare your figures for publication, which could be months or even years after you initially acquired your data. Going back and repeating an experiment because an immunofluorescent image was underexposed or a band was completely blown out can be frustrating, to say the least, so use these tips to make the most out of your data.

Doing your due diligence at the image-acquisition phase will save you time as you prepare your figures for publication.

Kaoru Sakabe (ksakabe@asbmb.org) is the data integrity manager at the ASBMB.

Figure 1. A spike on the histogram at 0 (red arrow) indicates that a Western blot was burned out (all black); a spike at 255 would have indicated that the blot was overexposed (all white).

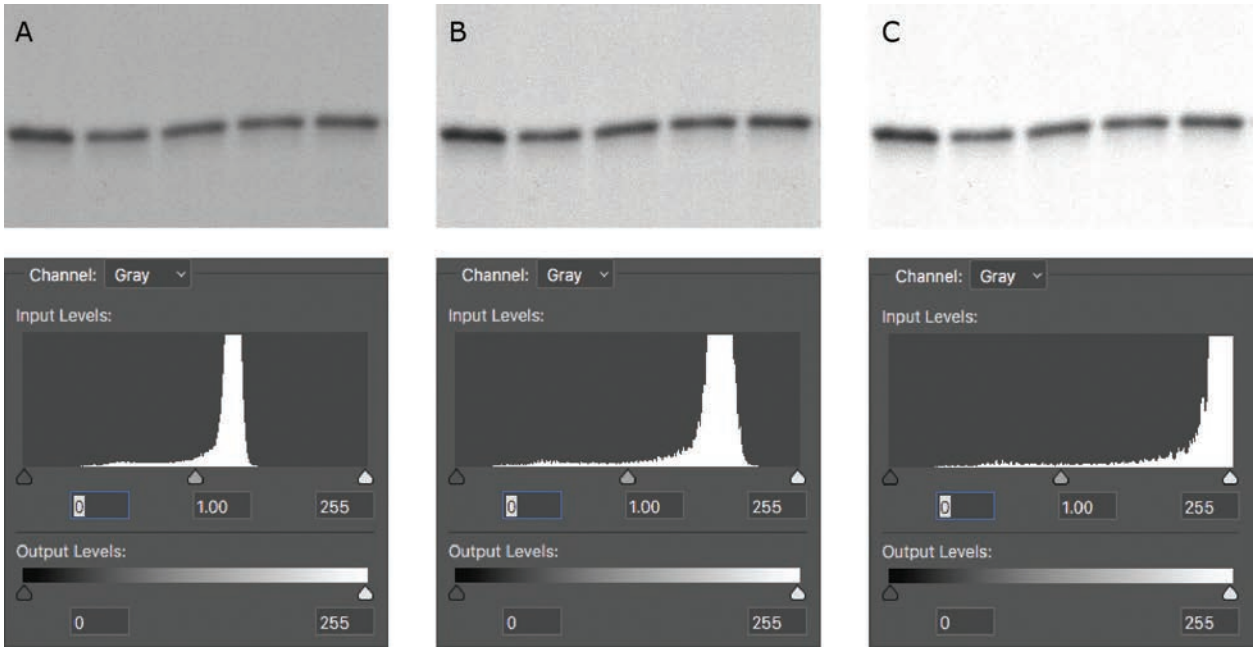


Figure 2. (A) The original capture of an immunoblot (B) Some of the levels were adjusted, but the pixels were still distributed between the two extremes. (C) The immunoblot was overly adjusted. The corresponding histogram shifted to the right, indicating that the majority of the pixels now were white.

Please come visit me and the editors of the Journal of Biological Chemistry at the 2017 ASBMB Annual Meeting in Chicago! We will be discussing how to publish in the journal. We encourage you to bring any questions you may have about preparing your manuscript, presenting your data, and making sure your reporting is transparent and reproducible. The session will be at 6:15 p.m. April 25 in room W184a, McCormick Place.

Making figures and slides for everyone, including the colorblind

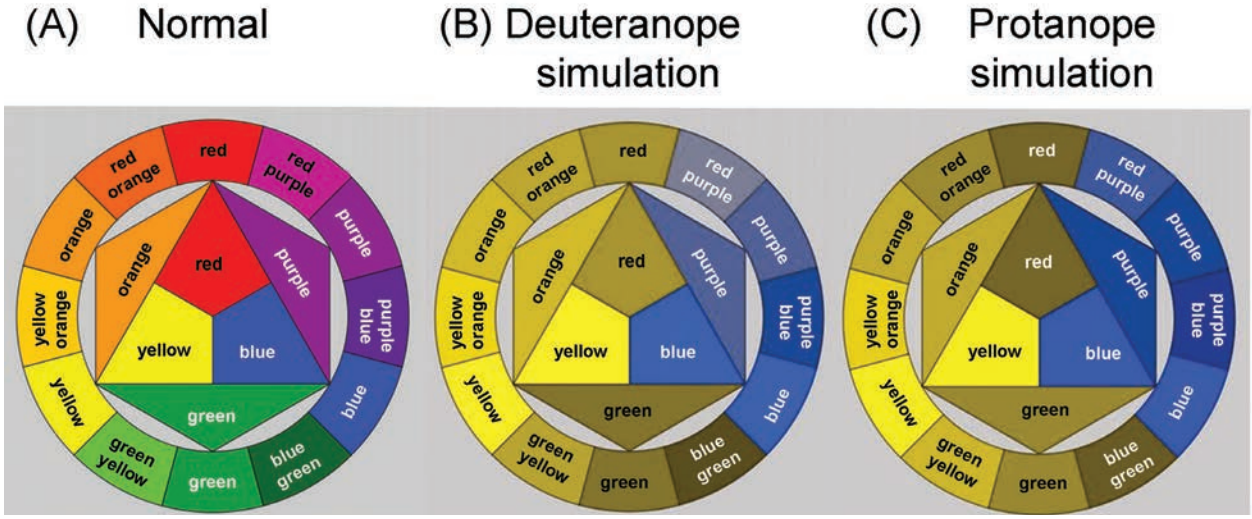
By Robert Roskoski Jr.

Color coding of objects is a useful and important way to convey information in biochemical studies. Examples include double staining in confocal micrographs and the use of a range of colors to denote variable gene expression. However, one in 12 males and one in 200 females are red-green colorblind. In an audience of 100 men and women, about four people may be colorblind. For effective communication, we must consider colorblind scientists when making figures and PowerPoint slides.

One in 12 males and one in 200 females are red-green colorblind. In an audience of 100 men and women, about four people may be colorblind.

Colorblindness is not the loss of perception of all colors (a condition called monochromacy). Rather, colorblindness makes it difficult for the person to distinguish between certain colors. The primary colors are red, yellow and blue, while the secondary colors are purple, green and orange (Figure 1). Visual color is sensed by three types of retinal cone cells corresponding to the primary colors of red, yellow and blue. Defective red cone cells result in a color blindness called protanopia (from Greek "prot" for the "first" type of cone), while defective green cone cells result in deuteranopia (from Greek "deuter" for the "second" type of cone). Both of these deficiencies are transmitted by X-chromosome-linked inheritance and result in red-green colorblindness. Note in Figure 1 that blue and yellow are perceived identically by normal and red-green colorblind individuals.

For both protanopes and deuteranopes, distinguishing red from green is more difficult than distinguishing yellow from green (Figure 1). Avoid having red and green lines cross or placing red and green objects close to each other (Figure 2). Although yellow has good color character for both the protanope and deuteranope, it may be too close to white for the normal



IMAGES COURTESY OF ROBERT ROSKOSKI JR.

Figure 1. A color wheel illustrating the colors as seen by (A) a normal individual, (B) a deuteranope or (C) a protanope

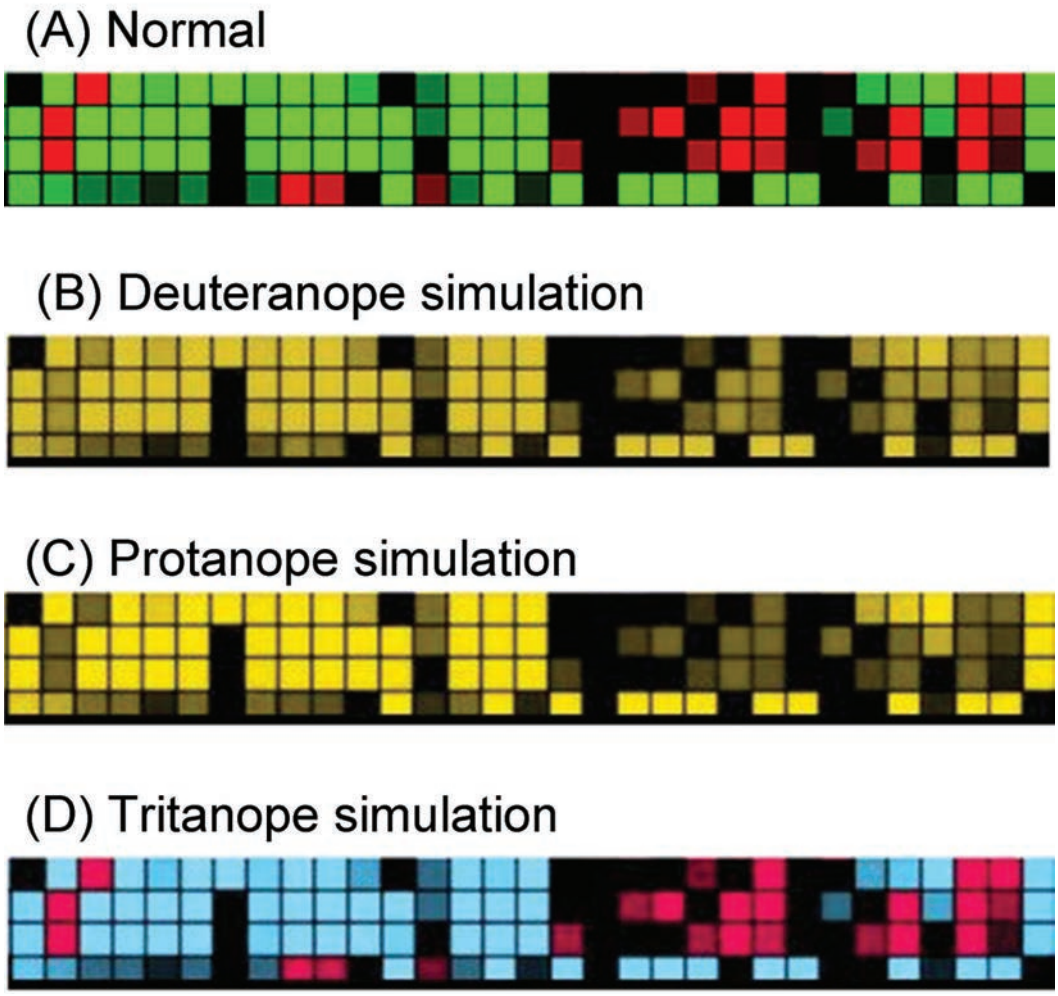


Figure 2. A gene-expression profile indicated by red, black and green matrices

eye to distinguish unless it has a black border or background. Black, white and gray are easily differentiated by nearly everyone.

Of all of the hues, blue is perceived uniformly as a color by all individuals. The color should be used first when generating colored images and slides, followed by yellow or green, depending upon the background. Red is perceived as black by protanopes (Figure 1), so red print over a black background may be invisible to them. Also, the use of dark-red text for emphasis when incorporated within a string of black text will not be perceived differently by protanopes. Moreover, dark-blue text may be perceived as black by everyone, so it may be better to use sky blue or Carolina blue for highlighting. With care, two different shades of blue may be distinguishable. It helps to examine such figures generated on a color printer to ensure that different shades of blue can be differentiated. If it makes no difference whether an object is red or black, it's all right to use red for the many people for whom red is a favorite color. Be aware, however, that the protanope will see it as black. White, gray and black are not colors, but they are distinguishable and useful.

You can check your colored figures for how the colorblind perceive them by using the freeware at vischeck.com. You can upload your figure and see how it appears to a colorblind person, or you can download the program and make analyses and revisions as necessary. This is the procedure for generating the figures for this piece. For additional advice on preparing color figures, visit jfly.iam.u-tokyo.ac.jp/html/color_blind.

A suitably made color figure may be worth more than a thousand words because of the additional information that color coding conveys. However, the coloring should be made so that it can be perceived and differentiated by all scientists, colorblind or not.

Robert Roskoski Jr. (rrj@brimr.org) is the scientific director of the Blue Ridge Institute for Medical Research.

Trump's travel ban

By Lana Saleh

On Jan. 27, President Donald J. Trump issued his first executive order on immigration that temporarily suspended the admission of refugees and barred citizens from seven countries that are predominantly Muslim — Iraq, Iran, Syria, Somalia, Sudan, Libya and Yemen — from entering the U.S. on any visa. I felt outraged and frustrated. I am Muslim. I am also a Palestinian refugee who immigrated to the U.S. I fear that the American dream is fading away for many young immigrants with the enforcement of executive orders of this kind.

My story

In August 1996, my mother received a call from the U.S. Embassy in Damascus, Syria. She was being notified that the I-130 application "Petition for Alien Relative" had been accepted by U.S. Immigration and Naturalization Service. The petition had been filed by her mother, my grandmother, who was a U.S. citizen. My mother was instructed to report to the embassy for an interview as a necessary last step for approval of her immigrant visa application.

The immigration process took a total of 10 years, from the initial filing in 1986 until acceptance in 1996. During that decade, several application forms were filled out, and legal documents, such as birth certificates, marital papers, bank records, employer records and education certificates, were obtained. Family photographs and even utility bills were presented. Medical examinations were performed. Fees were paid. All these things were done to satisfy the requirements of the I-130 application.

My mother, my two younger sisters and I set out for the interview at the U.S. Embassy in Damascus. Unfortunately, my eldest sister was not part of the immigration process, since she was no longer recognized as a child by the time our petition was approved. At the end of the interview, the consular officer granted us acceptance and a visa for travel to the U.S. within six months. I was one month from losing my status as a child according to the criteria of the INS. My parents scrambled to make all the travel arrangements so that my mother, my two younger sisters and I could immigrate to the U.S. before I turned 21.

Upon our arrival at the John F. Kennedy airport, we were escorted to the INS office, where my mother handed a sealed envelope given to us by the U.S. consular official in Syria to the official in the room. I felt my mother's sense of apprehension and relief as she extended the envelope she had clutched to her chest tightly during the 12-hour trip from Beirut to JFK, afraid that she would lose it or that it would disappear into thin air before the final interview, which would dictate the fate of a decadelong process and her children's future.

When the INS official finally stamped a temporary green-card visa seal on our passports, granting us entry to the U.S. as lawful permanent residents, my mother's eyes filled with tears. She realized her children would

have a more hopeful path than her own in a dreamland called America.

My mother and father's families were expelled from their homes in northern Palestine during the 1948 Palestinian exodus. My mother's family became refugees in Syria, and my father's family sought refuge in Lebanon. Both families knew extreme poverty and struggle. As children, my parents worked in farms and factories to help their families survive the hardships they faced after being forced to abandon their homes and start from scratch in foreign lands. My parents got married in 1970 and settled in Lebanon. But in 1975, the country began to witness 18 long years of war.

My maternal uncle, who is a medical doctor, was the first to immigrate to the U.S. Various family members, including us, followed. In 2001, my mother, my sisters and I took an oath of allegiance to the U.S. and became American citizens. Later that year, we filed I-130 petitions to bring my older sister and father to the U.S.

Worried about the future

My sisters and I now live in various parts of the U.S. with our own families. I am married to a Korean-American whose family immigrated to the U.S. when he was 2-years-old. My husband and I are both accomplished scientists with jobs in biotechnology. This country granted me the opportunity to live in a place free of war and turmoil and focus on obtaining a Ph.D. in biochemistry and co-authoring 26 peer-reviewed scientific articles. We have two beautiful daughters, who are half Palestinian-American and half Korean-American, which is a rare mix that one would find only in a society like the U.S. with such diverse cultural, racial and religious backgrounds. We believe that our family is an example of the American dream and that ours is the story shared by millions of immigrants who helped make this country into what it is today.

So with the enactment of President Trump's travel ban, I fear the loss of many talented scientists and engineers, who will be deprived of the hope for peace, freedom and opportunity to work in a land that always has extended the promises of peace, freedom and opportunity to the world.

The facts

On March 6, Trump revised the travel ban to remove Iraq from the list of countries. He claims the necessity of this travel ban on the grounds of protecting the country from a flood of dangerous terrorists. This claim has not been supported by facts. No terrorist attacks have been committed on U.S. soil by nationals of the seven affected countries since 1975, and the ban ignores the rigorous process of vetting applicants for visas by U.S. Citizenship and Immigration Services.

Trump's initiative also ignores the facts demonstrating the productivity and contributions of immigrants to the U.S. workforce. Analysis of the U.S. Census Bureau data between 2010 and 2013 by the Pew Charitable Trusts demonstrates that immigrants make up 13 percent of the population and 17 percent of the workforce. A report by the National Science Foundation states that 18 percent of all scientists and engineers in the U.S. in 2013 were originally immigrants. Of these foreign-born scientists, 63 percent were naturalized U.S. citizens, 22 percent were permanent residents and 15 percent were holders of temporary visas. Additionally, 57 percent of these immigrants were born in Asia; 6 percent were born in Africa. These are the two continents harboring the seven countries named in the first travel ban.

The NSF also reports that immigrant scientists and engineers were more likely to earn postbaccalaureate degrees in 2013 than were U.S. citizens who were born in the U.S., Puerto Rico or another U.S. territory or born abroad to U.S.-citizen parents. In fact, six of the 2016 American Nobel laureates are immigrants. The president of the Association of American Universities, Mary Sue Coleman, estimated 17,000 students from the seven countries affected by the first travel ban currently study at American universities.

If the travel ban is implemented, many sectors of the American economy are expected to suffer. In fact, many life scientists, including myself, view Trump's actions as destructive to the foundations of academic and scientific institutions. Trump's discriminatory travel order threatens the paradigm of open, free and timely global scientific exchange and decreases the effectiveness of development and innovation. The ability of foreign researchers in the U.S. to travel abroad to attend scientific meetings and conferences will be restricted, as will the ability of foreign scientists to attend scientific meetings or visit scientific institutions in the U.S. Such executive orders also greatly will impede the global recruitment of scientific talent and, ultimately, reduce American scientific competitiveness on the world stage. A failure to sustain an influx of international talent combined with a decrease in expenditure in life-science research and development, which the U.S. has been witnessing since 1999, implies a grim prospect for the future of science and engineering in this country. Our scientific prominence must not be bartered for short-term partisan gains.

To paraphrase the words of Beverly Gage in her Jan. 31 New York Times Magazine piece on the history of American resistance: It is time for all Americans to think about where they want history to go.

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The travel ban is why I can't be at the ASBMB annual meeting

By Adel Rezaei Moghadam

On Jan. 27, I was shocked to hear about the executive order that imposed a 90-day entry ban for travelers from seven countries, including Iran, into the U.S. I am Iranian. With the executive order, I had to give up my hope of attending the annual meeting of the American Society for Biochemistry and Molecular Biology being held this month in Chicago.

I was born in Ardabil, a city in Iran. I grew up in a family that had a great respect for science and seeking new knowledge. My parents inspired me to learn and improve. My education at primary and secondary school captivated me in many ways, and I found myself very passionate about health science. Therefore, I picked the field of experimental sciences as my major in high school, and I was very eager to pursue my educational goals at one of the best universities in my country.

At university, I began working as a research assistant in order to gain experience and to expand my interests in science. My research experiences led me to participate in many national and international conferences, workshops and training programs.

As I found myself curious and passionate about molecular biology, biochemistry and cell science, I wanted to do more research. I applied to the University of Manitoba in Canada and began in a master's program in January 2016. I chose Joseph Gordon and Saeid Ghavami as my co-supervisors, as they are world-class researchers in their respective fields of muscle development and cancer biology.

Prior to starting my master's degree, I collaborated with Ghavami, and we published several manuscripts and a book chapter. I was involved in various projects investigating therapeutic applications targeting apoptosis, autophagy and the unfolded protein response pathway in cancers. This gave me an opportunity to develop a wide range of skills and knowledge in the field of cancer biology and therapy.

I took a keen interest in Gordon's research program, which focuses on pediatric diseases involving muscle tissue and insulin-resistant cells. My proposed research combines Ghavami's expertise in cancer biology with Gordon's expertise in muscle development to study the regulation of programmed cell death in alveolar rhabdomyosarcoma for the potential of developing novel cancer therapies.

A few months ago, my co-supervisors encouraged me to submit an abstract to the 2017 ASBMB Annual Meeting. This meeting is a prestigious conference in the field of molecular biology. At this conference, I would have an enriching experience by attending biochemical and molecular biology presentations, networking with my peers and participating in career-development workshops. I was keen to learn about the latest innovations in cell science and cancer therapy. In addition, attending this conference would provide me with a great opportunity to interact with established scientists and graduate students and forge new collaborations.

After submitting my abstract, I was excited to later receive an ASBMB travel award. I felt honored to receive this support from the society.

As an international attendee to the meeting, I started preparing for my U.S. visa application, which involved traveling from Winnipeg to Vancouver to submit official documents. I was very disappointed when the executive order happened. I couldn't complete the application; there was nothing I could do. The effort I put toward attending this conference was gone.

However, I am very thankful to both my co-supervisors, who have supported me through this process; their encouragement has helped me. I also appreciate the sympathy and positivity from my lab mates in a situation that I have no control over. I have been overwhelmed by the support I have received from my family, friends, colleagues and ordinary Canadians.

There are many students and scientists affected by this travel ban. I think it is unfair for students and young trainees who cannot attend these events. I spent months planning for this conference that would benefit my training. It is a shame that I am put in this tough circumstance that I could not have foreseen.

I am filled with regret that I can't accept the ASBMB travel award and attend the meeting this year. However, I always will be grateful for receiving this honorable travel award, and I look forwarding to attending the meeting at a better time.

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Empowering immigrants in rural America

By L. Sofia Gonzalez

I stumbled upon United Speakers when I was a first-year transfer student at Truman State University. The university is in Kirksville, Missouri, about four hours away from St. Louis. The group's goal is to help the rural communities of Kirksville and nearby Milan through English-as-second-language classes, social activism and other needs. I feel blessed to have the opportunity to place my heart and soul into this group.

Why do I believe that outreach and service to immigrants is an important social issue? Why is this social issue relevant to scientists? Now, and particularly during the years I served as president and vice president of this student-run organization, I have seen my story and my parents' stories reflected in the individuals I've taught or served. I've had the experience of being an immigrant, which has driven me to provide language services.

I have lived with the fear that I didn't deserve to have a full education, internalizing what some teachers and professors had told me. After all, as I was told by these teachers and professors, why would a former undocumented child of modest economic means amount to much? I was fortunate to be young enough when my father petitioned permanent residency for us. Despite the 12-year wait, during some of which I returned to Mexico by myself, I was able to return and pursue my dreams.

Now, as someone who is at the threshold of graduate-school decisions, I know that I have come this far because of people and a country that invested in a stranger they did not know. I went through ESL classes starting in third grade and was helped so much by the few teachers who didn't feel burdened by a child who didn't speak a word of English for most of the year.

Once I learned English, I became my parents' translator. I spent parent-teacher conferences translating conversations for them and put in years of reading and understanding bills, letters, contracts and medical information for them. My responsibilities as a child prepared me well for United Speakers.

In the rolling hills of pork and corn agriculture of Kirksville and Milan, it is a surprise to find booming immigrant communities. The communities are made of new francophone African families as well as people from Mexico, El Salvador and Guatemala. The Hispanic community has been present for as long as the Smithfield pork company has been in these rural towns of 14,000 (Kirksville) and 2,000 people (Milan).

The heartland of America seems unprepared for these immigrant communities, but it is exactly for this reason that I grew to love the work so much and why United Speakers has built such a strong link to the communities in both Milan and Kirksville. My parents could rely on church organizations, nonprofits and wisdom from more stablished immigrants in St. Louis. But the immigrants in Kirksville and Milan have none of that. United Speakers serves as translators for court cases, helps the sole ESL teacher in the schools, offers the only adult ESL classes in both towns and works with the lone social activist in the severe cases of workers' abuse.

It has been uplifting to see new Truman students come to United Speakers and be overwhelmed by the work needed from them but yet give hours of their time every single week. As I often tell them, it is a different world from the safety of campus and the city suburbs we grew up in. The need, so painfully present in Kirksville and Milan, has driven me to start more ESL classes, hold ACT-prep classes for young immigrants interested in leaving the factory, invest in getting more help from the language faculty at Truman, and motivate friends and students who thought they were inadequate for teaching and outreach. If you are willing, you are prepared. There is no more to it.

I understand how highly improbable stories, with patience, extraordinary tenacity and just a bit of luck, become possible. It is because of this that I would argue that the scientific community needs to dive into the uncomfortable topic of immigration and political asylum. The potential that the scientific community, and the country as a whole, will find in immigrants is unsurpassable. New generations are laying their foundations in this nation for the very first time. That immense task needs to involve everyone, scientists included.

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Losing my doomsday clock to start graduate school

By Charles Brenner

When I was a sophomore at Wesleyan University, Ronald Reagan defeated Jimmy Carter to become the 40th president of the United States. It was 1980, and the Bulletin of Atomic Scientists moved its Doomsday Clock forward to seven minutes to midnight. In 1981, the clock was set to four minutes to midnight, and in 1984, the clock was set to 11:57 p.m.

During those years, I kept a clock in my bedroom that was set to the Doomsday Clock. My clock was a Russian pocket watch that my grandfather had given me. While the clock served an important notice of the state of the world, looking at that clock made me feel confined and limited in what I could accomplish.

I studied molecular biology at Wesleyan. When I graduated in 1983, I took my first job at Chiron Corp. With so much global uncertainty, I wasn't sure what to do with my life, and I could not commit to going to graduate school. It was obvious to me that there was a world of discoveries that could be made in biochemistry and molecular biology, but I wasn't entirely sure that there was going to be a world as we knew it.

I enjoyed the opportunity to visit a biotechnology center in a developing country in late 1985. It was there that I realized that my greatest contribution to the world might be through research and teaching.

Returning to the U.S., I decided that I would take the plunge and invest in my own education. I was working at the DNAX Research Institute when the Doomsday Clock was pushed back to 11:54 p.m. in 1988. Around that time, I lost my doomsday clock. While it had sentimental meaning as a gift from my grandfather, that clock was holding me back. I needed to suspend temporarily my disbelief in the future to create one for myself.

I was in graduate school at Stanford University when the Berlin Wall was demolished and the Soviet Union collapsed; the clock was pushed back to 11:50 p.m. in 1990 and 11:43 p.m. in 1991. There's no question that the thaw of the Cold War allowed me to experience more space in which to express my creativity.

My son was in in college when Donald Trump defeated Hillary Clinton to become the 45th president of the United States. The Bulletin pushed the clock forward to 11:57:30 p.m., which is the closest it has been to midnight in my lifetime. My son and most of the college students I teach don't yet know what their life's work will be and whether they will need advanced training. Many of them are uncertain about their future because of current events.

Just like Wesleyan students in the early 1980s, they want to be politically active. I support their efforts to agitate for speech, education, equality of opportunity, peace and freedom of expression. I remind them that a part of freedom of expression can be finding your own voice in the classroom and in the laboratory, thereby advancing the boundaries of what is known.

I am grateful that external forces in the late 1980s and early 1990s provided me with enough space to go to graduate school. But I could not have done so had I not lost my doomsday clock.

Charles Brenner (charlesbrenner@mac.com) is the Roy J. Carver Chair and head of biochemistry at the University of Iowa. Views expressed are his own.

Science in the post-truth age

By Jennifer DuBois

The results are in: Denmark is a prison.

In Hamlet's dark mood, Denmark felt like, and therefore was, a prison. As he famously explained, "There is nothing either good or bad, but thinking makes it so."

The statement speaks to the current zeitgeist. The Oxford Dictionaries named "post-truth" as its 2016 word of the year, defining it as "relating to or denoting circumstances in which objective facts are less influential in shaping public opinion than appeals to emotion and personal belief." The American Merriam–Webster dictionary acknowledged a related phenomenon earlier, giving its 2006 word of the year to comedian Stephen Colbert's "truthiness" — believing something that feels true, even if it is not supported by fact.

A climate of post-truth may not be such a big problem if you happen to be in marketing. However, for the rest of us, particularly those of us in the scientific community, the sway of feelings over fact-based arguments is troubling.

The line between fact and belief, even in matters of science, has grown more and more nebulous. A co-worker teaching an introductory chemistry course recently described an incident. While explaining the infrared spectrum of carbon dioxide to his class, he commented on the connection between carbon dioxide, infrared absorption and global climate change. After the lecture, a student in a state of some agitation expressed her surprise that the professor was allowed to air his personal opinions in a university lecture. The professor did a double take: Had climate change become the new evolution?

Part of the problem may stem from how scientists talk about science. Experimental science is rooted in the scientific method, which is itself a form of inductive reasoning based on systemic observations. There's the rub: Inductive reasoning, or "the inference of general laws from particular instances" (thank you again, Oxford Dictionaries), easily can be misused or mistrusted by those not practiced in the art. Members of the American Society for Biochemistry and Molecular Biology are familiar with the adage from enzymology in which we say that we never can prove that a mechanism is true; we only can falsify particular steps.

This falsification criterion, in turn, comes from a philosophy of science that sets a very high bar for truth. An observation, however often made, is just that: an observation. Bertrand Russell illustrated the shortcomings of inductive reasoning with his story of a turkey. This turkey woke every morning expecting to be fed as he always had been without fail, only to have his head cut off on Christmas Day. Albert Einstein is reputed to have summed up the problem of induction with the adage "No amount of experimentation can ever prove me right; a single experiment can prove me wrong."

Observations, even repeatedly made, can appear to lie. And scientists, rather than proclaiming climate change and its anthropogenic causes as fact, instead describe a series of observations with which that conclusion is consistent.

The general public is right to be skeptical, just as our graduate students absolutely must be, if we are to have any hope for scientific progress. Copernicus was right to question the obvious conclusion that the sun circles the Earth, just as medieval mariners suspected the world was not flat. However, their questions were not grounded in feelings, preferences or personal agendas but rather a rising tide of countermanding observations.

Abraham Lincoln is said to have observed: "You can fool all the people some of the time, and some of the people all the time, but you cannot fool all the people all the time." By letting the air out of the emotional appeals — the marketing — we may hope to cut down the number of people in the middle category. Science education and outreach have a vital role to play in demonstrating how to use inductive reasoning — the scientific method — to construct a credible argument. Embedded in that notion is that every argument based on observation has limitations and the reason for those qualifications and wiggle words that we insert at the conclusion of every scientific paper.

Truth may be for the saints and philosophers. Experimental science and those who invoke it are limited to theories: proofs beyond a reasonable doubt. Such proofs cannot be communicated adequately in a brisk 140 characters of text, and they rarely leave one with a satisfying, truth-y feeling. Science education and outreach must become more engaging and compelling than ever if an informed scientific viewpoint is going to compete — for the public's minds if not their hearts — in 2017.

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Finding the right fit

By Rachel Fairbank

The summer before my senior year of high school, I worked in a developmental genetics lab at Cornell University, where I screened for mutations suppressing the sma-9(cc604) mutation in the nematode Caenorhabditis elegans. My adviser was a brand-new faculty member and optimistic enough to give a chance to a lost and confused high-school student. After the summer ended, I continued to work in the lab. Its culture of learning and questioning filled a need in me.

As these experiments progressed, I remained lost and confused about my life. Research offered escape from a difficult family situation and challenged me on an intellectual level. Although close, research was not the perfect fit. My search had not yet ended.

As a college freshman, I chose music as a major. At age 21, I changed my major to biology. Throughout this time, I continued working in the genetics lab. Before I knew it, I'd put in seven years, a period during which I watched the hard work of an entire lab grind toward a pathway describing cell-fate specification decisions in a C. elegans cell lineage.

Eventually, I graduated with a bachelor's degree in biology and headed to the wilds of Texas for a Ph.D. program in developmental biology at Baylor College of Medicine. At age 26, crippled with anxiety resulting from an accident, I dropped out of graduate school, leaving research behind. At 29, I entered graduate school again, this time in creative writing at the University of Houston.

At 30, I finally found my vocation: science writing.

In a little while, I'm set to graduate with an MFA in creative writing. During these years, as I wrote my first full draft of a book and started my first science-writing job, I've had the time to reflect on my decisions. Some days, when I think about all of the false starts and odd paths I've traveled in my life, I wonder how I could have been so lost, so confused, so aimless. On the worst of these days, I cave in to regret about all the time I've lost.

Of all my regrets, the biggest one is this: I spent too much of my life wanting to be someone else. Like Cinderella's ugly stepsisters chopping off their toes to fit into a golden slipper, I chopped off pieces of myself in the hopes of fitting into a profession when instead I should have focused on finding what fit me.

One side of me longed for creativity. Another side of me longed for logic. As a music major, I zeroed in on science. As a science major, I indulged my creative impulse with music and writing. Back and forth I went. Science. Art. Science. Art.

Forcing myself into a mold was exhausting. In a never-ending battle of fear and doubt, I questioned my abilities and the future. In my second year of the developmental biology program, I was hit by a car walking to school. In the aftermath of recovery, as I returned to school, I was forced to confront the reality that I hadn't found my vocation yet. As a researcher, I was nervous and absent-minded, with a habit of going off on tangential literature searches.

Soon enough, I had to sit down and think about who I was and what I loved to do. I loved the rush of analyzing results. I loved learning about new discoveries. I loved thinking about the big picture.

Most of all, I loved hearing stories about science. My favorite classes were the ones where we learned the stories behind the discoveries. Barbara McClintock toiling in obscurity with her rows of corn as she discovered transposable elements. Hilde Mangold performing the tiny, delicate experiments demonstrating the process of embryonic induction only to die in a gas explosion before her results were published.

Even the scientists around me contained a treasure trove of stories. My adviser at Cornell did her postdoctoral fellowship in Andy Fire's lab during the years when he performed the experiments demonstrating gene silencing could be triggered by tiny snippets of double-stranded RNA. The lab next door to us had published the paper that laid the foundation for this discovery. During my final semester of college, my favorite class, which I renamed "scientist story time," featured a tag-team of professors that turned each subject into a story about the scientists behind the discovery.

Eventually, I stumbled onto the idea of science writing and found my way to a writing program where I learned to lean into my storytelling impulse. Once that happened, the puzzle pieces fell into place. The doubt and anxiety disappeared, replaced by the confidence that I'd finally found the career that fit.

Rachel Fairbank (rcfairbank@gmail.com) is a science writer and graduate student in creative writing.

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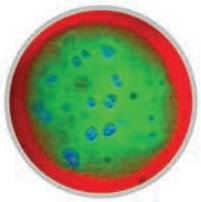

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