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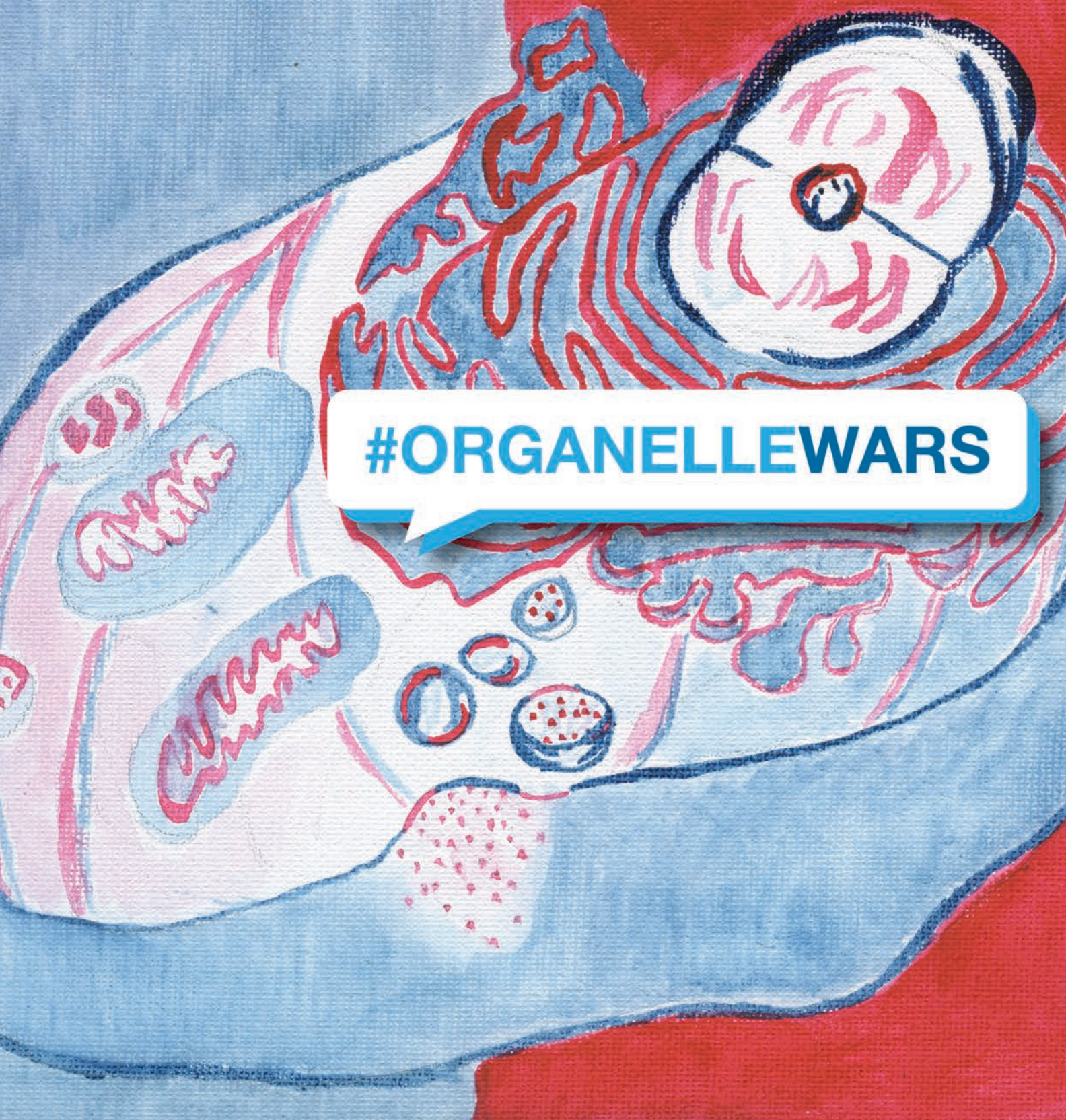
ASBMB TODAY

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



the journal of biological chemistry

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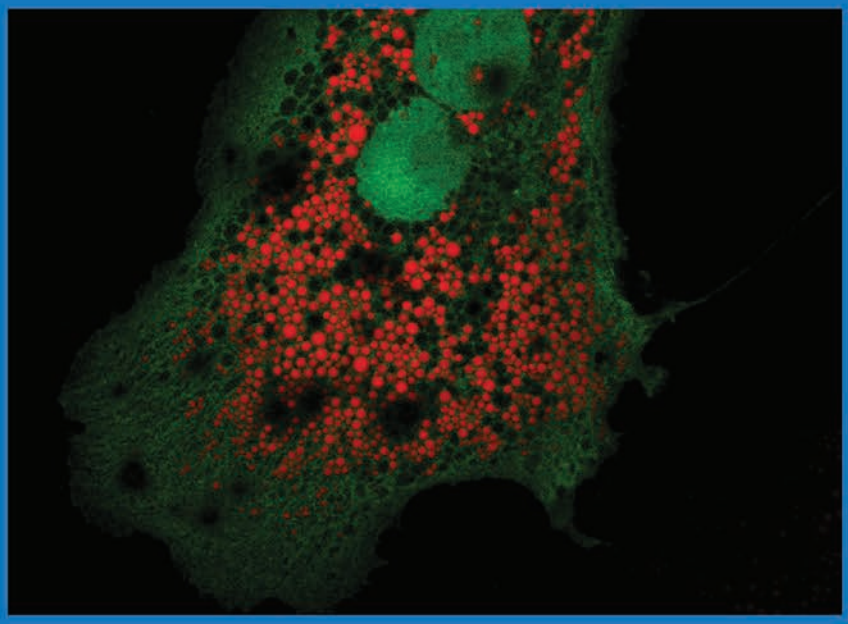
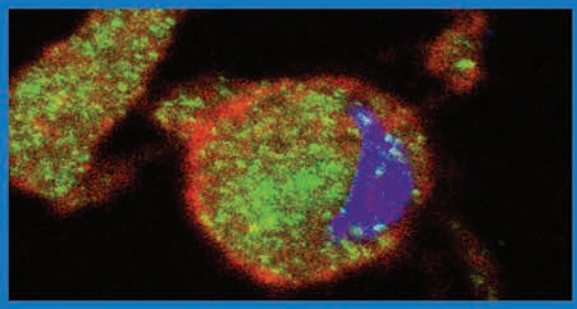
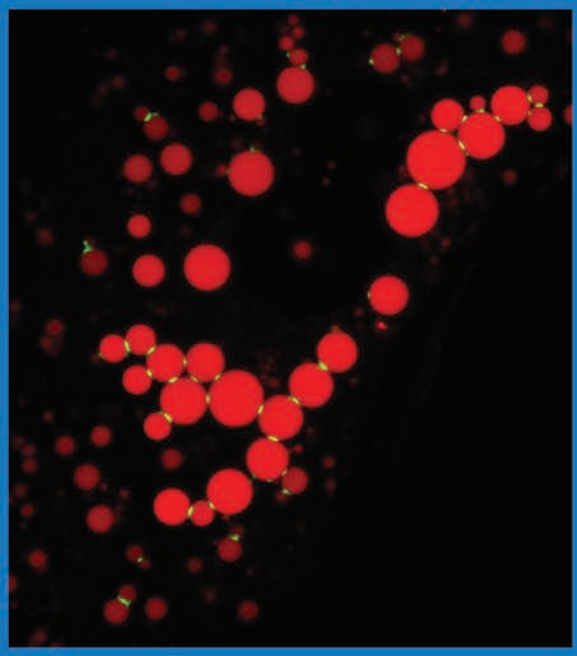
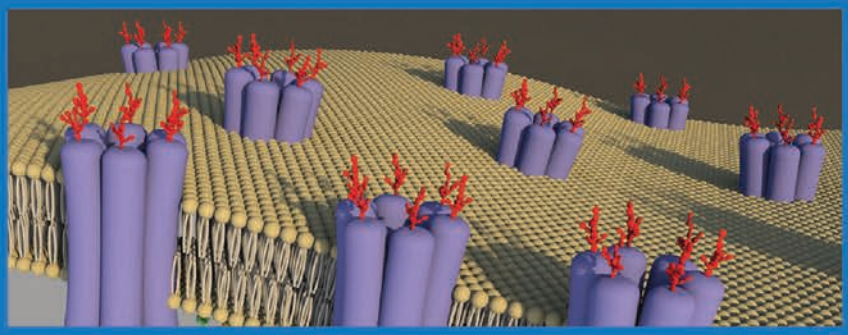
Highlights of 2016









The editors at the Journal of Biological Chemistry are pleased to present a collection of papers as our "Highlights of 2016."

Thumb through the copy that is included with this issue of ASBMB Today or visit www.jbc.org/site/highlights/2016/.

ASBMB

American Society for Biochemistry and Molecular Biology

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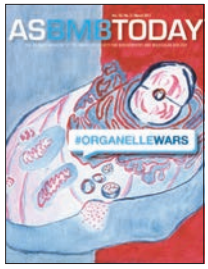

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ASBMB TODAY
THE MEMBER MAGAZINE OF THE AMERICAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY

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Biochemists, unite!

By Natalie Ahn

The American Society of Biological Chemists, which later became the American Society for Biochemistry and Molecular Biology, was founded more than 100 years ago. The founders' aim was to embrace the biological sciences from the chemical point of view and separate biochemistry from physiology as a distinct scientific pursuit. Our fields of biochemistry and molecular biology defined the molecular and chemical mechanisms that govern biological processes, fostering one of the most productive areas of scientific investigation.

Today, the ASBMB continues to stand for the discovery and understanding the molecular mechanisms of life at deeper levels. We stand for education: We provide each new generation of scientists and educators with mentorship and resources to teach critical thinking, problem solving and the other skills of great scientists. We are prominent national advocates: We reach out on behalf of our members and the larger community of scientists to inform governmental and nonscientific groups about the consequences of policy decisions on science. We also inform scientists about policies that affect our research and teaching. And, most importantly, we foster community by creating a home for people to find collaborations, share ideas and promote careers.

What does it mean to be a biochemist or molecular biologist as our fields increasingly become the foundation for discovery across the wide spectrum of biological and medical sciences? Binks Wattenberg, Enrique De La Cruz and Dan Raben begin a discussion to answer this question for biochemists in their Perspectives essay on page 37. Keeping our community alive enables each of us to flourish.

You can help make us even stronger. Not a member of the ASBMB? Please join our community today. Already a member? Enlist a colleague to join too. It's easy: Go to www.asbmb.org/membership/ or email membership@asbmb.org.

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

Let's talk about reproducibility

By Benjamin Corb

In 2015, Leonard Freedman, the founder and president of the Global Biological Standards Initiative, and colleagues published a report that claimed $28 billion worth of biomedical research is irreproducible. The amount accounts for more than 50 percent of all preclinical research grants.

This analysis, published in PLOS Biology (1), shook scientists and science advocates alike. Articles and blog posts critical of the study have been published in the 18 months since Freedman's analysis was published, including by us and others, that were critical of his study, citing concerns over Freedman's potential conflicts of interest (2).

The inability of scientists to reproduce other scientists' findings is an issue that the biomedical research enterprise should be concerned about, especially given that so much of the research is funded by the federal government, which is to say taxpayers. Being good stewards of taxpayer money must mean conducting sound science. Sound science requires reproducibility. But is the amount of research irreproducibility in the life sciences really so clear-cut that we can chastise an entire community? The fact is that we need more data before we can draw firm conclusions.

Researchers at the Center for Open Science recently began their own analysis on reproducibility, specifically in cancer research projects (3). Their preliminary results are complicated. Project researchers are examining 29 papers published in Science, Nature and Cell since 2012, repeating the same experiments and asking whether or not they can reproduce the findings. One unique aspect of this reproducibility effort is its transparency. Unlike previous efforts that have not shared which papers they attempted to reproduce, this group is publishing their findings from beginning to end — the papers they reviewed, the procedures and methods they followed, and their entire results are all available for review and scrutiny. Sean Morrison, a senior editor of eLife, told Nature, "For people keeping score at home, right now it's kind of two out of three that appear to have been reproduced" (4).

Conversations about reproducibility can be different among scientists because the importance of the issue may vary significantly across different disciplines. Some efforts, like one led by the Federation of American Societies for Experimental Biology, set the issue of reproducibility as the need for scientists to be transparent, careful, diligent about recordkeeping, and mindful of reducing potential pitfalls, such as confirmation bias and poor study design, while conducting research. Scientists rightfully feel proud of the enormous contributions made by biomedical research, but they can get defensive about their work, which can complicate the issue. Moreover, even the definition of what constitutes reproducibility is murky, because science, particularly biological science, is complex. Small environmental differences from one lab to another or unclear methodology explanations can affect the ability of one researcher to reproduce the results of another.

Nevertheless, rather than overreacting to criticisms regarding reproducibility, let's commit to the goal of making our scientific work careful, scholarly and impactful. If we articulate this goal clearly, then the enterprise will be better protected against any future criticisms, and perhaps then we'll show the criticisms to be irreproducible.

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2. http://policy.asbmb.org/2015/06/22/research-reproducibility-rises-to-the-top/.
3. Nosek, B. & Errington, T. *eLife* **6**, e23383 (2017).
4. Baker, M. & Dolgin, E. *Nature* **541**, 269 (2017).

Benjamin Corb (bcorb@asbmb.org) is director of public affairs at the American Society for Biochemistry and Molecular Biology. Follow him on Twitter at twitter.com/bwcorb.

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Six ASBMB members named fellows of the National Academy of Inventors

Francis Barany of Weill Cornell University, Barbara D. Boyan of Virginia Commonwealth University, Paul L. Modrich of Duke University, Nicholas Muzyczka of the University of Florida, Ronald T. Raines of the University of Wisconsin–Madison and Bruce W. Stillman of Cold Spring Harbor Laboratory have been nominated as 2016 fellows of the National Academy of Inventors.

NAI fellows are named inventors on U.S. patents who are nominated by their peers for their outstanding contributions as innovators. Fellows are recognized for having a significant effect on society through innovations in patents and licensing as well as discovery and technology.

These six members, along with the other 2016 NAI fellows, will be inducted as part of the Sixth Annual Conference of the National Academy of Inventors in April.


BARANY


BOYAN


MODRICH


MUZYCZKA


RAINES


STILLMAN

Serio to be dean at UMass Amherst


SERIO

Tricia R. Serio, professor and head of the department of molecular and cellular biology at the University of Arizona, will become the dean of the College of Natural Sciences at the University of Massachusetts Amherst in August.

Serio takes over the department from Steven Goodwin, who is stepping down after leading the college and one of its predecessors, the College of Natural Resources and the Environment, since 2002.

Serio's research is focused in the field of protein folding, where she seeks to comprehend the mechanisms by which protein conformations can act as elements of inheritance and infectivity.

Serio brings leadership and a distinguished academic career to her new role.

Hartl and Schulman named visiting professors


HARTL


SCHULMAN

F. Ulrich Hartl, director of the department of cellular biochemistry at the Max Planck Institute of Biochemistry, and Brenda Schulman, the Joseph Simone vhair in basic research at St. Jude Children's Research Hospital, have been named as 2017 Vallee visiting professors.

Hartl's research explores protein folding and quality control. Recently, he has focused on understanding the molecular mechanisms underlying neurodegenerative disorders.

Schulman's research focuses on the structural basis for post-translational modification by ubiquitin and ubiquitinlike proteins.

The Vallee Visiting Professorship program pairs renowned scientists with leading biomedical research institutions as a means of promoting intellectual exchange, collaboration and discovery.

In memoriam: P. Michael Conn

P. Michael Conn, senior vice president for research, associate provost, and the Robert C. Kimbrough professor of internal medicine with a joint appointment in the department of cell biology and biochemistry at Texas Tech University Health Sciences Center, died in November.

Conn identified an underlying biological principle that has significantly altered the understanding of cellular mutations that result in human disease. His work has shown the potential for the development of new therapeutic treatments.

A highly accomplished researcher, Conn won, among numerous awards and honors, the J. J. Abel Award of the American Society for Pharmacol-

ogy and Experimental Therapeutics as well as the Miguel Aleman Prize, Mexico's national science medal.

Conn authored and co-authored more than 350 publications and wrote and edited more than 200 books. He served as president of the Endocrine Society, during which time he founded the forerunner to the Hormone Health Network, a group that serves as a public education resource on hormone-related issues.

In memoriam: Eugene Roberts

Eugene Roberts, distinguished scientist emeritus at the City of Hope Medical Center, died of pneumonia in November. He was 96.

ROBERTS

Originally born Evgeny Rabinowitch in Krasnodar, Russia, Roberts moved to Detroit in 1922. He obtained his B.S. from Wayne University in 1940 and received his M.S. and Ph.D. at the University of Michigan in 1941 and 1943, respectively.

Shortly after his graduate work, Roberts was employed to work on the Manhattan Project, serving as an assistant head of the project's inhalation research program. After the war, Roberts joined Washington University in St. Louis, Missouri, in the division of cancer research.

An important figure in the neuroscience community, Roberts discovered the presence of abnormally large quantities of γ-aminobutyric acid in the brain in 1949. In 1954, he went on to work at the City of Hope Medical Center in Duarte, California, where he later created an interdisciplinary division of neurosciences, the first of its kind.

He is survived by his wife, Ruth Roberts; his son, Paul; his daughters, Judith and Miriam; and his five grandchildren.

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

Upcoming ASBMB events and deadlines

APR

April 5 – 6: ASBMB Hill Day
April 22 – 26: ASBMB annual meeting, Chicago

MAY

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

JUNE

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

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Susan Lindquist (1949 – 2016)

By Jeffery W. Kelly & Richard I. Morimoto

Susan Lindquist was one of those very rare, incredibly special individuals who inspired many scientists, whether through her seminars, an insightful question at a research talk or poster session, or her prolific and innovative contributions to the scientific literature. Sue also wrote frequently about our roles and responsibilities as academic scientists and the importance of being supportive and reliable mentors. She was an outspoken advocate for women and minority scientists. Her passion, out-of-the-box thinking, and enthusiasm for science were contagious. Sue's life was cut short by cancer on Oct. 27. For those who knew her, it will come as no surprise that she fought this horrible disease to the very end through a combination of grit and scholarship, which is how she lived her honorable life.

PHOTO COURTESTY OF CEAL CAPISTRANO/WHITEHEAD INSTITUTE

Susan Lindquist

I, Jeff Kelly, first came to know of Sue Lindquist through her amazing contributions to the scientific literature. I recall meeting her for the first time at the University of Chicago. She was charming, insightful, incredibly intelligent and, in many respects, larger than life! After a decade of friendship, we co-founded FoldRx Pharmaceuticals. Through the challenges typical of successful biotech ventures, Sue remained a wonderful friend and colleague until her untimely death. One of these challenges we encountered in FoldRx was not having enough money to pursue Sue's dream of using yeast-based neurodegenerative disease models to discover small-molecule drugs — a dream that finally is being realized in another company, Yumanity (see below). Through sacrificing her own scientific agenda, Sue enabled FoldRx to commercialize Tafamidis, a drug now sold by Pfizer, to ameliorate the transthyretin amyloidoses.

I, Rick Morimoto, interacted with Sue Lindquist as far back as when I was a first-year graduate student at the University of Chicago. It was a chance meeting with Sue when she had joined Hewson Swift's laboratory as a postdoctoral fellow and was writing her Ph.D. thesis (she had done her graduate work with Matt Meselson of Harvard University) that led me to the wonders of the heat shock response and the lifelong pleasure of knowing Sue. Her infectious enthusiasm was unmatched, and this led me, upon completion of my doctoral work at Chicago, to join Meselson's lab as a postdoctoral fellow. Upon returning to Chicago and joining the faculty of the biochemistry and molecular biology department at Northwestern University, our laboratories re-engaged, exchanged students and reagents, and had numerous joint meetings. It was a wonderful time. A few years later, together with Betty Craig at the University of Wisconsin–Madison, we expanded our mega-group meetings to form the Midwest Stress Response and Chaperone Meeting, which continues in its 22nd year with the same spirit of support of young investigators and great science.

Sue started her academic career as a professor in the department of molecular biology at the University of Chicago, and then she moved to the Massachusetts Institute of Technology as a professor of biology until her death. Lindquist served as director of

PHOTO COURTESTY OF EDWARD BUCKBEE

Lindquist with her husband, Edward Buckbee, and her daughers, Alana (left) and Nora (right), when she received the Naitonal Medal of Science in 2010.

the Whitehead Institute from 2001 to 2004, becoming one of the first women in the nation to lead a major independent biomedical research organization. She was also among the first women to serve on the board of directors of a major pharmaceutical company, Johnson & Johnson. Besides being a Whitehead Institute member, Sue was an associate member of the Broad Institute of MIT and Harvard, an associate member of the David H. Koch Institute for Integrative Cancer Research at MIT, and a longtime investigator of the Howard Hughes Medical Institute. She received numerous awards over the course of her career, including the National Medal of Science, the highest scientific honor bestowed by the United States (awarded by President Barack Obama). She was an elected member of the National Academy of Sciences, the National Academy of Medicine, the American Philosophical Society, the American Academy of Arts and Sciences, and the British Royal Society.

Sue is perhaps best known for her prion research. She provided strong evidence for a new paradigm in genetics — that inheritance could result from self-perpetuating protein aggregate structures. She discovered genetic modifiers of this protein-only form of inheritance, such as the disaggregase Hsp104. These insights led to discoveries that contributed significantly to understanding devastating neurological maladies such as Alzheimer's, Parkinson's and Huntington's diseases. She was also very well known for the concept that molecular chaperones, such as Hsp90, functioned as capacitors of morphological evolution by regulating the influence of other mutated proteins. For example, Sue showed that Hsp90 could protect cell-signaling pathways from being compromised by mutations in other proteins. If Hsp90 function was lowered — by environmental stress, for example — mutated proteins would fold differently and new traits would appear. She also pioneered how stress-responsive signaling pathways like the heat shock response regulated this capacity. Sue was considered one of the experts on cellular protein folding, and she studied how chaperones like Hsp90 could influence kinase function as it relates to cancer and other diseases.

Many of Sue's most insightful discoveries were enabled by her devotion to her favorite organism, Saccharomyces cerevisiae, and her fearlessness to jump to mice, iPS-derived human cells or whatever organism was most appropriate to demonstrate the relevance of her findings in yeast.

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This approach is exemplified by Sue's discovery that three copies of the α-synuclein gene compromised vesicular trafficking, which now is accepted as the basis for some of the Parkinson's disease phenotypes — a discovery that was made in yeast. This concept of making discoveries in yeast that translate to neurodegenerative diseases underlies the strategy for drug discovery at Yumanity Therapeutics, a company Sue co-founded with Tony Coles in 2015.

Ulrich Hartl, a Max Planck Investigator and longtime friend of Sue Lindquist, captured the feelings of many by telling us, "We are not only losing an eminent leader of the field but also a warm-hearted and generous colleague and an advocate for young scientists. She will live on in our memories and through her fundamental contributions to science."

Sue loved the two-steps-forward-one-step-back process of scientific discovery as much as she loved dancing. She was an eternal optimist. By seeing through hazy data to make discoveries like few others could, she trained numerous scientists, providing the inspiration that would convince someone to pursue science for a lifetime.

We close with a fond memory. Sue, like many gifted people, was a bit absentminded when it came to practical things. One memorable event unfolded at the 2006 Protein Folding in the Cell meeting in Saxtons River, Vermont. One of us, Morimoto, had picked up Sue at her home in Boston, and together with Betty Craig and Carol Gross of the University of California, San Francisco, enjoyed the two-and-a-half-hour drive to Vermont accompanied by laughter and discussions among longtime friends. Arriving at Saxtons River, we discovered that Sue had left her computer at home. Thinking that we would catch some of the Boston colleagues coming up to Saxtons River, we quickly made a number of calls, but to no avail, as they were already en route. At this point, with a fair amount of commotion, it was discovered that Patricia Clark of Notre Dame University had grown up nearby in New Hampshire and that her dad flew a helicopter and had one in his yard. In quick course, it was arranged for the computer to be delivered to Hanscom Field near Boston and brought to the meeting by Patricia's dad. The next morning, just when the meeting participants were gathered outside for a coffee break, we heard the characteristic sound of a helicopter as it got closer and closer and finally landed in the adjacent open field. Even though Sue had hoped that this step in the process would be discreet, all the participants watched in amazement as Sue retrieved her computer. This contributed further to her larger-than-life persona.

Rest in peace, dear friend — you are sadly missed.

PHOTO COURTESY OF EDWARD BUCKBEE

Lindquist at the U.K.'s Royal Society in 2015 when she became a member.


Jeffery W. Kelly (jwk@scripps.edu) is the Lita Annenberg Hazen Professor of Chemistry in the department of chemistry and the chairman of the department of molecular medicine at the Scripps Research Institute. Richard I. Morimoto (r-morimoto@northwestern.edu) is the Bill and Gayle Cook Professor of Biology in the department of molecular biosciences at Northwestern University and the director of the Rice Institute for Biomedical Research.

Thressa C. Stadtman (1920 – 2016)

By P. Boon Chock & Rodney L. Levine

Thressa "Terry" C. Stadtman died peacefully at her home on December 11 at age 96. She is remembered as a pioneer in anaerobic electron transport, vitamin B12 metabolism and selenium biochemistry.

Stadtman and her husband, Earl, created a superb mentoring environment at the National Institutes of Health in which many outstanding scientists were trained. The Stadtmans' rigor in scientific inquiry and their superbly successful mentoring is affectionately known as "the Stadtman Way" (see https://history.nih.gov/exhibits/stadtman). Their Journal Club, initially feared by newly arrived trainees and then enthusiastically embraced, was the furnace where scientific rigor was forged. No detail or question was too trivial to be brushed aside. The Stadtman Way also included generous sharing of credit in publications with more junior scientists. Camaraderie, lively and friendly discussions, and lab parties led to lifelong friendships among those lucky enough to be shown the Stadtman Way.

PHOTO COURTESTY OF BUHM SOON PARK

Thressa C. Stadtman

Terry was born in 1920 in Sterling, New York, and studied bacteriology at Cornell University, where she received her B.S. in 1940 and her M.S. in 1942. She earned her Ph.D. from the University of California, Berkeley, under the guidance of Horace Barker, who discovered the active form of B12. For her thesis work, she investigated the mechanism of methane fermentation by the activities of two anaerobic microorganisms, Clostridium sticklandii and Methanococcus vannielii, which she isolated from the mud that she scooped up from the San Francisco Bay. While working in Barker's lab, she met his technician and graduate student Earl Stadtman. They were married in 1943.

After graduation in 1949, Terry and Earl moved to Boston, where she did postdoctoral training with Christian Anfinsen at Harvard Medical School while Earl worked with Fritz Lipmann at the Massachusetts General Hospital. When Anfinsen was offered a position as the chief of the Laboratory of Cellular Physiology and Metabolism at the NIH in the newly formed National Heart Institute, he invited Terry to move with him to continue her work on bacterial cholesterol oxidase. At that time, the NIH was one of the few institutions that would hire a married couple as independent investigators. Earl was able to tag along as the accompanying spouse because Anfinsen also offered him a position. They moved to the NIH in 1950. Terry retired from the NIH in 2009, 59 years after her arrival.

The Nobel laureate Michael Brown at the University of Texas Southwestern Medical Center, who did a postdoctoral stint at NIH, says, "What I remember most about Terry was her enthusiasm for science … Terry was outgoing and always eager to discuss data, whether they were her own or that of others. Her interest in biology was as pure as that of anyone I ever knew. Terry loved scientific beauty without any concern for its utility. She was a scientist's scientist."

Terry's initial scientific goal at the NIH was to elucidate principles of biochemistry by studying the metabolism of anaerobic bacteria, organisms that seem capable of almost any imaginable chemical reaction. While chemists had to use harsh solvents and high temperatures to make many reactions

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happen, these microbes easily achieved the same reactions with extraordinarily high yields at ambient temperatures in aqueous solutions! Using extracts from M. vannielii and C. sticklandii, Terry studied the mechanisms of amino acid fermentation and methane production from carbon dioxide. Her investigations gave insights into anaerobic electron transport and vitamin B12 metabolism. In the course of these studies, Terry and her colleagues discovered four of the vitamin B12-dependent enzyme systems. She also established that the free form of B12 can function as a methyl-group carrier and that its deoxyadenosyl coenzyme forms serve as hydrogen carriers. Her findings provided the basis of our understanding of methane biosynthesis.

PHOTO PROVIDED BY P. BOON CHOCK & RODNEY LEVINE

Stadtman with her husband, Earl, at their Ph.D. graduation

Terry's pioneering work on selenium biochemistry began with her 1972 discovery that protein A, a low-molecular-weight subunit of the Clostridial glycine reductase, is a selenium-containing protein. In 1976, Terry and her colleagues became the first to demonstrate that the selenium was present as selenocysteine. They showed that selenocysteine plays an essential role in the catalytic activity of many selenoenzymes. Terry went on to establish that selenium is an essential constituent of several other enzymes. In addition to its incorporation as a selenocysteine residue, the selenium can be coordinated with molybdenum in a molybdopterin cofactor. She extended her findings to eukaryotes with her demonstration that a thioredoxin reductase in human lung adenocarcinoma contained selenocysteine. Her studies then established that the codon for selenocysteine in thioredoxin reductase was UGA, which was normally the terminator codon. By collaborating with August Böck of Munich, Dolph Hatfield of the National Cancer Institute and others, she soon was established that selenocysteine is inserted co-translationally rather than as a post-translational modification. Hence, selenocysteine came to be known as the 21st amino acid, sharing its codon with the termination process.

In 1984, Terry and colleagues showed that the selenium-containing nucleoside in several bacterial seleno-tRNAs was 5-[(methylamino)methyl]-2-selenouridine. Collaborating with Robert Balaban at the NIH, she established that seleno-phosphate is the immediate selenium donor for the biosynthesis of selenium-containing biomacromolecules, selenocysteine in proteins and 5-methylaminomethyl-2-selenouridine in seleno-tRNAs. A series of publications from 1996 until her retirement elucidated the mechanism by which selenophosphate synthetase catalyzes the synthesis of selenophosphate. It's not surprising that Terry became known as the "mother of selenium biochemistry."

Terry was elected to the National Academy of Sciences in 1981 and the American Academy of Arts and Sciences in 1982. Among her awards, Terry received the William C. Rose Award of the ASBMB in 1986, the Klaus Schwarz Medal from the International Association of Bioinorganic Scientists in 1988, and the inaugural

L'Oreal Lifetime Achievement Award for Women in Science from L'Oreal–UNESCO in 2000. The organism Methanospaera stadtmaniae is named in her honor. The American Society for Biochemistry and Molecular Biology gives out the Earl and Thressa Stadtman Distinguished Scientist Award every other year to an established scientist for his or her outstanding achievement in basic research. The award alternates with the Earl and Thressa Stadtman Young Scholar Award, which goes to a scientist with 10 years or less of experience as an independent investigator. The awards were established by friends and colleagues of the Stadtmans to preserve their legacies as scientists and mentors.

Terry, with the wholehearted support of Earl, championed and supported women in science. Terry was able to attend Cornell only because of scholarship support and working as a waitress for four hours a day. This is why Terry generously endowed the Stadtman Scholarship Fund for undergraduates and the Stadtman Fellowship Fund for graduate students at Cornell. The funds provide support for women who are majoring in the sciences. Being optimistic about the future for women in science, she specified that when women no longer face roadblocks to careers in science, Cornell may redirect the Stadtman funds to support other groups who still face impediments in becoming scientists.

PHOTO PROVIDED BY RODNEY LEVINE

Stadtman tried her hand at viticulture.

Earl and Terry lived in a home on six acres adjacent to Rock Creek Park in Washington, D.C., one of the places President Abraham Lincoln sometimes took carriage rides to try to unwind a little during the Civil War. After Earl's death in 2008, Terry deeded their property to expand the park. In accepting the gift, the park commissioners gave that section of the park the legal name the Stadtman Preserve. It features a mature forest, steep slopes, floodplain, wetlands, a stream, and nearly a thousand azaleas and rhododendrons planted by Earl.

Terry also tried her hand at applied biochemistry. In the early 1990s, she purchased a first-growth vineyard in the Burgundy region of France known by the name "Les Chouacheux." She, Earl and a microbiologist friend from Berkeley, Terry Leighton, then launched a classic Stadtman experiment aimed at answering the question, "If we make a pinot noir wine with Burgundy grapes but with Napa Valley yeast and fermentation techniques, will it taste like a French Burgundy or a California Pinot Noir?" Their answer to the question turned out to be, "We're not sure, but it's a really good wine!"


P. Boon Chock (BChock@nih.gov) and Rodney L. Levine (Rlevine@nih.gov) are members of the laboratory of biochemistry of the National Heart, Lung and Blood Institute, where Terry Stadtman's laboratory was located during her 59 years at the National Institutes of Health.

Cox wins Tabor award for work on extracellular matrix

By Mariana Figuera–Losada

Thomas Cox at the Garvan Institute of Medical Research in Australia won a Journal of Biological Chemistry/Herb Tabor Young Investigator Award at the 2016 Matrix Biology Society of Australia and New Zealand Conference. Amanda Fosang of the Murdoch Childrens Research Institute in Australia, who is a JBC associate editor, presented the award to Cox.

Cox obtained his Ph.D. in 2008 at the University of Durham in the U.K. He continued his training as a cancer-cell biologist at the Institute of Cancer Research in London and then in the laboratory of Janine Erler at the University of Copenhagen. He started his own group at the Garvan Institute of Medical Research in late 2016. He now is the matrix and metastasis group leader.

The Tabor award recognized Cox's research on the effects of extracellular matrix remodeling on cancer progression, metastasis and therapy response. He has shown that ECM remodeling is induced by post-translational modifications that alter its biochemical and biomechanical properties and that lysyl oxidase and lysyl oxidaselike family members play an important role in this process. As a long-term goal, Cox intents to establish a novel therapeutic approach to treat solid cancers by targeting ECM dynamics.

PHOTO COURTESY OF THOMAS COX

Thomas Cox

"Being awarded the Journal of Biological Chemistry/Herbert Tabor Young Investigator Award is a tremendous honor for me," says Cox. "Over the years, I have seen many fantastic junior researchers awarded this prestigious prize and feel very privileged to be one of the 2016 recipients."

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

Hidden information in mRNA

By Dawn Hayward

Just before the translation step of protein synthesis, the cell protects its messenger RNA with a 5′ methylguanosine triphosphate cap and a 3′ polyadenosine tail. Other modifications can occur within the message itself. One of these, called m^6A_m, appears on adenosine nucleotides at the start of transcripts and has a methyl group on both its sugar and nitrogenous base. This modification was discovered in the 1970s but received little attention, and its role was unknown.

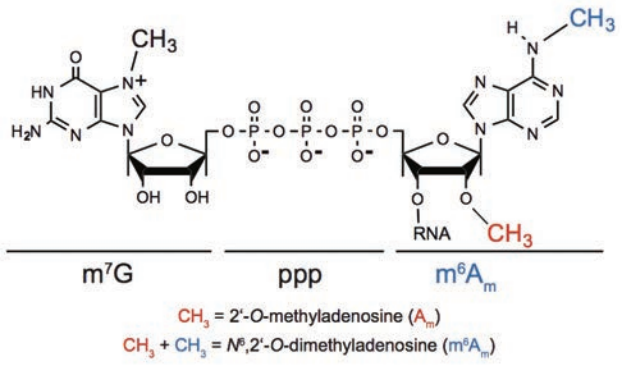

IMAGE PROVIDED BY SAMIE JAFFREY

Modifications of the extended mRNA cap.

In a paper published Dec. 21 in the journal Nature, Samie Jaffrey of the Weill Cornell Medical College and colleagues explored m^6A_m in depth. They determined which enzyme specifically removes this modification and the profound effects this mark has on mRNA stability.

Jaffrey says that the m^6A_m modification largely was overlooked after its initial discovery. The m^6A_m modification was present at less than 10 percent of the level of the m^6A modification, which also is found in mRNA. The adenosine is modified only at its nitrogenous base and occurs internally rather than at the beginning of the transcript. In addition, FTO, the fat mass and obesity-associated protein, was identified as the "eraser" of the m^6A_m mark. The m^6A_m modification fell by the wayside.

But by using analytical chemistry techniques, the Jaffrey group discovered that FTO actually prefers the m^6A_m modification over m^6A. FTO showed a higher catalytic efficiency toward m^6A_m. In cells, FTO overexpression decreased m^6A_m levels specifically, and its knockdown increased the ratio between m^6A_m and A_m.

Next the Jaffrey group looked at this modification's role. While the cap and tail are known both to protect and aid in translation initiation, modifications to the nucleotides themselves have been studied less. Jaffrey calls these marks "hidden information sitting inside RNA molecules" and sought to uncover their significance.

To figure out what m^6A_m is doing, the group analyzed previously compiled translation efficiency data. This data reveal how well a large group of mRNA transcripts is translated at a given point, explains Jaffrey. The investigators sorted transcripts based on the first nucleotide identity, A_m, C_m, G_m or U_m, and used specialized mapping techniques to identify those beginning with m^6A_m. From this analysis, m^6A_m transcripts showed longer half-life and higher expression levels, an indication of stability.

Next, the group collaborated with Mergerditch Kiledjian of Rutgers University to study the decapping of mRNA transcripts. The 5′ methylguanosine triphosphate cap at the beginning of messenger RNA can get "decapped" by DCP2, the putative enzyme in a large complex. By using a radioactivity assay where the release of methylguanosine diphosphate was detected if DCP2 succeeded in decapping the mRNA, researchers found that transcripts beginning with the m^6A_m modification were not as easily decapped and destabilized.

Finally, the Jaffrey group explored the phenomenon of microRNA-mediated degradation of mRNA transcripts. This degradation is important, as some transcripts are mysteriously resistant to its effects.

The group again looked at previously compiled translation efficiency data, this time in DICER knockdown cells, a key component of the degradation machinery. Transcripts beginning with m^6A_m did not change significantly, while others increased, an indication of lowered susceptibility.

Jaffrey marvels at how the stability of these mRNAs was encoded: "It was actually in the nucleotides, something that was invisible to standard methods," he notes. Next steps from this work include studying how levels of this modification are affected in disease states as well as identifying the methyl-transferase that adds this mark.

For the time being, m^6A_m is back in the spotlight.

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PI-PLC β1 in differentiation and disease

By Lucio Cocco

Evidence from several laboratories has highlighted the presence of autonomous nuclear inositol lipid metabolism. The evidence suggests that lipid molecules are important components of signaling pathways operating within the nucleus. The findings are important, given the fact that nuclear signaling activity controls cell growth and differentiation.

Among the nuclear enzymes involved in this system, inositide-specific phospholipase C, or PI-PLC, β1 is one of the most extensively studied enzymes. Besides the studies on its signaling activity, clinically oriented studies have shown that a mono-allelic deletion of the PI-PLCβ1 gene is associated with the evolution of myelodysplastic syndromes, or MDS, into acute myeloid leukemia. Studies also have showed that increased PI-PLCβ1 gene expression, due to reduced methylation and reactivation of its promoter, associates with responsiveness to demethylating agents, such as azacitidine, in MDS. Extensive clinical and molecular evaluation have aimed to establish a predictive role of increased PI-PLCβ1 gene expression during the first three cycles of treatment with the demethylating drug azacitidine. The data obtained hint at PI-PLCβ1 expression as a useful tool for the early identification of a subgroup of patients with a higher probability of response to azacitidine. These data suggest also a possible involvement of nuclear PI-PLCβ1 in the early stages of hemopoiesis and specifically in the control of cell-cycle progression in progenitor hemopoietic cells. Nuclear PI-PLCβ1 also is involved in myogenic differentiation.

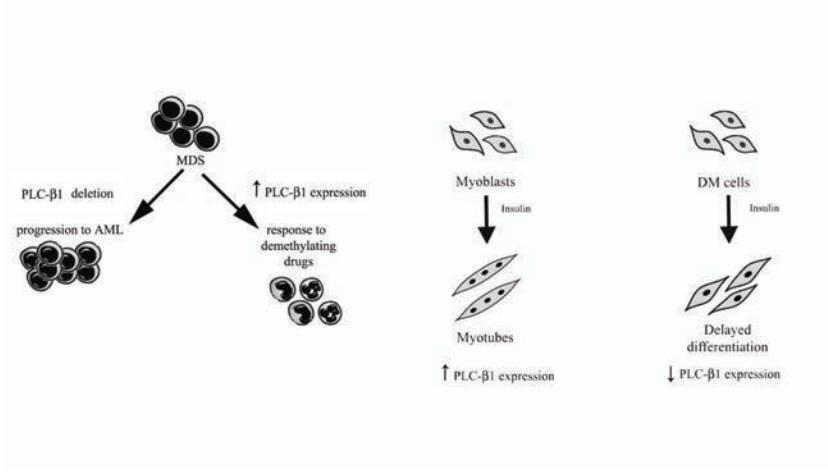

IMAGE PROVIDED BY LUCIO COCCO

Inositide-specific phospholipase C plays a role in MDS and a form of muscular dystrophy.

Indeed, nuclear PI-PLCβ1 plays a crucial role in the initiation of the genetic program responsible for muscle differentiation: the enzyme activates the cyclin D3 promoter during the differentiation of myoblasts to myotubes. This indicates that PI-PLCβ1 is essential for cyclin D3 promoter activation and gene transcription through c-jun/AP1.

Myotonic dystrophy is the most prevalent form of muscular dystrophy in adults. DM type 1 and type 2 are dominantly inherited multisystem disorders. DM1 is triggered by the pathological expansion of a CTG triplet repeat in the gene coding for DMPK, the dystrophia myotonica-protein kinase. A CCTG tetranucleotide repeat expansion in the ZNF9 gene, which encodes a CCHC-type zinc-finger protein, causes DM2. Unlike in normal myotubes, the level of expression of PI-PLCβ1 in DM1 and DM2 cells already is elevated in proliferating cells. Treatment with insulin induces a dramatic decrease in the amount of PI-PLCβ1. During differentiation, cyclin D3 and myogenin are elevated in normal myotubes. When DM1 and DM2 cells are induced to differentiate, they do not show any increase in these proteins. Forced expression of PI-PLCβ1 in DM1 and DM2 cells increases the expression of differentiation markers myogenin and cyclin D3 and enhances fusion of DM myoblasts. These results highlight again that nuclear PI-PLCβ1 expression is a key player in myoblast differentiation, functioning as a positive regulator in the correction of delayed differentiation of human skeletal muscle.

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A factory runs riot

By Catherine Goodman

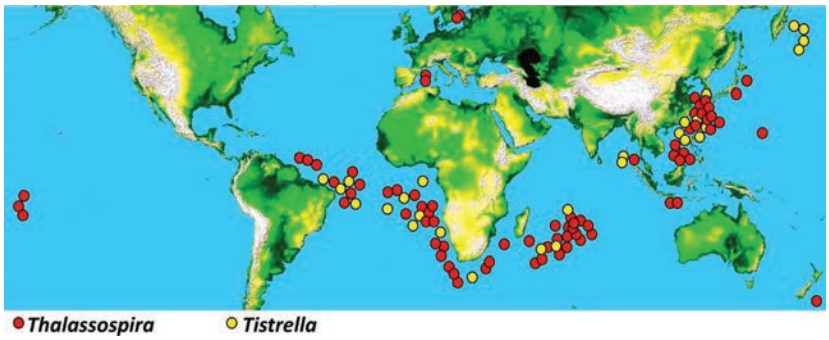
Bacteria are at war. Their foes? Other microorganisms. Their weapons? Among other things, they deploy small chemicals called natural products. These chemicals often are co-opted by humans for drugs, making it important to keep identifying natural products and the pathways by which they're made. While these biosynthetic pathways often have minor departures from rules developed over many examples, a recent paper in the **Journal of Biological Chemistry** reports an extreme case of rule-breaking that explains how a structurally diverse group of natural products, called thalassospiramides, is made.

Two of the common pathways that bacteria use to make natural products rely on a series of enzymes linked together in long chains. The chained enzymes function much like factory workers at an assembly line: When a molecule arrives at their station, they add a new piece or adjust an existing piece and send it along. In this way, many copies of the same thing are made efficiently.

However, the bacterial foe doesn't just sit around, waiting to be killed; the bacteria evolve to resist specific natural products. So many assembly lines either swap workers with other pathways or get the workers to make tweaks to create multiple, slightly different molecules based on the same overall blueprint.

A team led by Pei-Yuan Qian at Hong Kong University of Science and Technology and Bradley Moore at the University of California, San Diego previously had teamed up to study the assembly line that makes thalassospiramides. These compounds inhibit an enzyme called calpain protease, which is important in neurological disorders, cancer and other medical conditions.

In earlier work, Qian, Moore and colleagues reported 14 thalassospiramides from four types of ocean-dwelling bacteria. They also wanted to determine the blueprint for the assembly line because, as Qian recalls, "We wondered how and why bacteria from different genera produce similar compounds."



Locations where the bacterial strains were collected.

Surprisingly, the assembly line was sending some workers home for the day, asking other workers to perform their jobs two or three times, and bringing in workers from elsewhere in the factory. As first author and then-postdoctoral fellow Avena Ross recalls, "The huge diversity of molecules seemed to be coming from a single, quite simple assembly line that behaved in a highly unusual manner."

Since their first study only examined four bacteria, teams led by Qian, Moore and Ross — now a faculty member at Queen's University — suspected there might be more to learn. So they looked through the Marine Culture Collection of China, including samples from the Baltic and Bering seas, the coasts of Madagascar and New Zealand, and everywhere in between, to assemble 130 different strains of bacteria, leading to 21 new thalassospiramides.

By looking at the DNA sequences for a subset of the bacteria they collected, the authors found seven copies of the thalassospiramide assembly line, only four of which were operational. By pairing compound structures with assembly lines, the authors could see how the four functional lines were hiring, firing and reusing enzymes even more than anticipated based on the previous report. Moreover, they discovered that attempts to outsource an intact assembly line to a different bacterial factory might have caused the seemingly functional but unproductive lines, as some of the individual parts needed for the assembly line workers weren't available in the new factory.

While some of these individual mechanisms of hiring, firing and reusing enzymes to create compound diversity have been seen in other natural product assembly lines, the new mechanisms as well as their combination are surprising. Moore notes that the assembly line "is able to break so many of the conventional 'rules.'"

Ross believes more surprises await as they determine how the rule-breaking occurs at the level of individual enzymes and reactions. Perhaps, in times of war, rules are meant to be broken.

Catherine Goodman (cgoodman@asbmb.org) is the JBC's scientific editor. Follow her on Twitter at twitter.com/cate_goodman.

Predicting when blood goes bad

By John Arnst

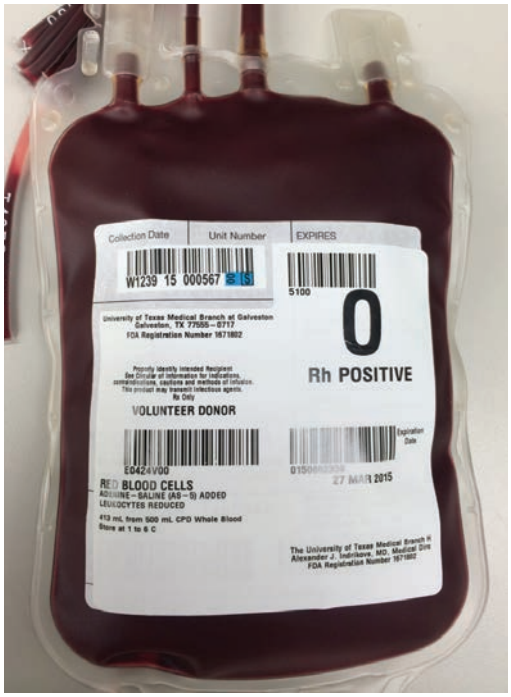
Despite the best efforts of blood banks and networks, some blood bags end up spoiling before they can make it to patients in need. A blood bag's spoilage depends on the red blood cells' ability to avoid hemolysis caused by low ATP levels. The breaking of red blood cells can release byproducts, such as iron and hemoglobin, that become toxic in a free-floating form by intensifying bacterial infections and interfering with the nitric oxide signaling that is key for vasodilation.

In a paper published in the journal **Molecular & Cellular Proteomics**, researchers at the University of Wisconsin–Madison have generated a model for predicting post-storage ATP levels in blood based on the concentrations of five key metabolic factors. They found these factors exhibited high heritability and were inherited as a block rather than individually.

"We can use this model to identify blood that can be potentially stored for longer or shorter periods as well as potentially identify other factors to make all blood store longer," says Erin M. M. Weisenhorn. Weisenhorn, the first author on the paper, is a graduate student in Joshua J. Coon's group.

To generate this model, the researchers performed a comprehensive metabolomics and proteomics study to examine heritability of metabolites associated with ATP consumption in the red blood cells in 18 pairs of twins. Heritability is the proportion of phenotypic variability due to genetic factors.

The researchers wanted to examine the proteins in the red blood cells' membranes because of their high abundance there. To do this, they



centrifuged the hemoglobin out of whole red blood cells, as it makes up about 98 percent of the cells' protein content. The researchers digested the remaining proteins with trypsin and subjected them to a novel proteomics and metabolomics approach. The approach involved two sets of liquid chromatography with tandem mass spectrometry, separately optimized for acidic and basic metabolites, combined with a standard analysis by gas chromatography and mass spectrometry. They quantified 328 separate metabolites from the red blood cells, including those from the glycolysis and pentose phosphate pathway.

The researchers then identified 119 membrane proteins and 148 metabolite concentrations that had a heritability rate of more than 30 percent, which previously had been established as a benchmark level of heritability in studies with the same group of twins.

The researchers additionally found that there was a 60 to 90 percent chance of heritability for the entire set of metabolites related to the pathways. "We observe that you don't just sort of randomly inherit high levels of 2, 3-diphosphoglycerate or pyruvate, but rather that the entire block of metabolites from the intermediate part of the pathway are all inherited at a higher or lower level," says Thomas J. Raife, a co-author at the UW's department of pathology and laboratory medicine. "This means a phenotype."

The researchers proposed phenotypes for both high and low levels of ATP after six weeks of storage. The phenotypes correlated to five key parameters, which include pH and concentrations of phosphofructokinase, an essential enzyme in glycolysis, as well as the proteins Band 3, BPGM and CA1. As a trio, the latter proteins correlated negatively with post-storage ATP conditions, making lower concentrations desirable for blood viability.

This phenotypic existence of varying levels of glycolysis also could have implications for a variety of metabolic diseases, says Weisenhorn. One such correlation is the Warburg effect, in which a cancer is associated with extremely elevated levels of glycolysis. "You can potentially imagine if someone inherits higher levels of these glycolytic proteins or metabolites and has naturally higher flux through this pathway, then they might be more predisposed towards cancer."

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Targeting cholesterol for ALS treatment

By Monika Deshpande

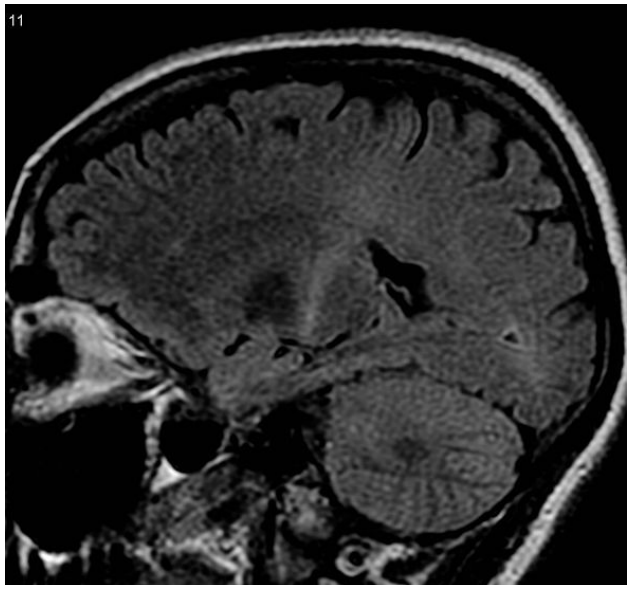

IMAGE COURTESY OF FRANK GAILLARD/WIKIMEDIA

The brain of an ALS patient.

In 2014, amyotrophic lateral sclerosis came into the limelight with the Ice Bucket Challenge. ALS, also known as Lou Gehrig's disease, is a debilitating and fatal disorder that attacks the nerve cells. There is no cure for ALS. In a recent paper in the **Journal of Lipid Research**, researchers showed for the first time that patients with ALS have higher levels of cholesterol in the fluid surrounding the brain than people without the disease. The researchers propose that a potential therapeutic approach for ALS treatment could be to use drugs that reduce the levels of cholesterol in the brain.

ALS typically starts with twitching of muscles and ultimately leads to paralysis and respiratory failure. About 20,000 Americans have this disease at any given time. Riluzole, the only drug for ALS approved by the U.S. Food and Drug Administration, has been on the market since 1995 and shows only modest slowing of progression in a fraction of patients. There is no current treatment that stops or reverses ALS.

In the JLR study, led by William J. Griffiths of the Swansea University Medical School and Martin Turner of the University of Oxford in the U.K., the researchers investigated whether targeting cholesterol was an option for ALS treatment. Earlier studies indicated that an increase in cholesterol caused oxidative stress that led to neuronal death in ALS. Secondly, a gene critical for cholesterol metabolism called CYP27A1 was identified as a susceptibility gene that increased a person's likelihood of contracting ALS. Some studies indicated that statins, drugs that reduce cholesterol, exacerbated ALS. However, a 2013 study definitively showed that the detrimental effects of statins were abolished when adjusted for age of onset and body mass index. "In the light of these studies, and considering that about 25 percent of the body's cholesterol is present in brain, it seemed like cholesterol might be a potential target for ALS studies," says Griffiths.

The investigators used serum, the clear liquid separated from clotted blood, from 35 ALS patients and 24 healthy individuals, and cerebrospinal fluid, the colorless fluid surrounding the brain and spinal cord, from 20 ALS patients and 15 healthy individuals for the study. They measured cholesterol and its metabolites by mass spectrometry.

Their analysis of the serum showed no significant differences in cholesterol or most of its metabolites between ALS patients and controls. However, Griffiths and colleagues observed the most interesting results in the cerebrospinal fluid. The level of cholesterol, specifically nonesterified cholesterol, was higher in the cerebrospinal fluid of the ALS patients.

Griffiths and his team explained this observation by pointing to the greater number of neurons that die during ALS. The dying neurons release more cholesterol from their membranes, and the metabolic pathways are unable to remove this excess cholesterol.

Alternatively, the investigators proposed, cholesterol metabolism through a pathway called LXRβ signaling could be defective in ALS patients. Support for this hypothesis comes from mice that are deficient in the LXRβ gene. These mice, similar to ALS patients, have neuronal inflammation and high cholesterol levels in the spinal cord. Furthermore, other metabolites of cholesterol that are part of the LXRβ signaling pathway also were reduced in the cerebrospinal fluid of ALS patients.

"We think that people with ALS are unable to dispose of the nonesterified cholesterol from brain efficiently, leading to the presentation of the disease," explains Griffiths. Griffiths is optimistic that "not only will our work provide a method to diagnose ALS but be useful to stratify people for more efficient clinical trials and could also provide a route to development of a new drug to treat ALS."

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Solving the faculty diversity problem

A model shows it's possible to diversify the workforce within a single tenure cycle. The solution has to do with hiring decisions

By Angela Hopp

Last fall, a paper in the journal eLife called into question the wisdom that the key to diversifying the faculty hiring pool is focusing on building the talent pool of underrepresented scientists. The paper showed that diversity within the Ph.D. pool has increased dramatically. The problem is that scientists from underrepresented backgrounds aren't being hired at the rate needed to establish parity in basic science departments (1).

"My goal is to use data to demonstrate clearly and quantitatively that there's a robust talent pool of scientists from underrepresented backgrounds. We can stop having conversations based on the idea that scientists of color don't exist," says Kenneth Gibbs Jr., the lead author of the paper.

As a program director at the National Institute of General Medical Sciences in the Division of Training, Workforce Development and Diversity, Gibbs isn't in the business of advising faculty hiring committees or department administrators. But the model he and his colleagues developed spells out for hiring managers what they have to do, collectively, to establish a national faculty body that reflects the talent pool and comes closer to reflecting the American population.

"If two-thirds of the medical schools hired and retained one faculty member from an underrepresented background every year for six years, the system would reach parity with the Ph.D. pool in one tenure cycle," explains Gibbs.

Bottom line: Diversifying the faculty is not unsolvable. As Gibbs puts it, "the mathematical reality is that it takes around 100 assistant professor hires annually to reach parity with the Ph.D. pool. It's just arithmetic."

Gibbs earned his bachelor's degree in biochemistry at the University of Maryland, Baltimore County, and his Ph.D. in immunology at Stanford University. He became a science policy fellow for the American Association for the Advancement of Science, completed a postdoctoral fellowship at the National Cancer Institute, and then joined the NIGMS.

For the paper, Gibbs teamed up with Jacob Basson, a biostatistician at the NIGMS; Imam M. Xierali, who manages the American Association of Medical Colleges' diversity programs; and David Broniatowski, an assistant professor who directs the Decision Making and Systems Architecture Laboratory at the George Washington University.

ASBMB Today's executive editor, Angela Hopp, talked to Gibbs about his interest in workforce diversity, his team's findings and obstacles to achieving parity. This interview has been

edited for length, style and clarity.

You wrote once about how the metaphor of the STEM pipeline doesn't stand. Why don't you like it?

I appreciate where people are trying to come from. There is a clear educational pathway to scientific independence. (Common wisdom is that) the only way you get more scientists of color in the end is to stuff more at the beginning. That would lead you to believe that workforce diversity challenges are mainly about a lack of diversity early on, but empirically that doesn't work out. Moreover, even if we had a clear pipeline, current conversations about this topic often ignore the shearing forces that are inside the pipe. When training environments and climates do not support all trainees, including minority trainees, that is a shearing force that we need to change.

Also, I really don't like the notion of leaking, as it takes agency away from people. I did not leak out of any path. I actively made decisions about my career. I work in science policy, as do many contemporaries who are from well-represented racial backgrounds. When my white male colleagues make a choice similar to mine, it's not referred to as "leaking," but when people of color or women choose a career outside of academia, we're called "leaks." I don't like that. Nobody leaked. Everybody is making choices. To really get at workforce issues, including diversity, we need to understand those choices and address the root causes of those choices.

Conversations about a "leaky pipeline" take our attention away from what we have: a talent pool of thousands of scientists of color. Let's acknowledge that reality and, as a community, work to ensure we are utilizing this talent pool.

When and how did you get started in this line of work?

I was an AAAS policy fellowship for two years at the National Science Foundation in the Directorate for Education and Human Resources in the Division of Human Resource Development. I got interested in understanding what was happening with me and my peers as it related to our career development.

I was in a number of different meetings where I was hearing talk about young scientists broadly and young scientists of color particularly that did not seem to match my own life experience. I also recognized, as a scientist, that questions such as "Why do Ph.D. scientists make the career decisions they do?" and "How do the decisions differ across demographics?" can be addressed empirically.

You were hearing people say what in those meetings?

One was that (scientists of color) didn't exist. That didn't align with my experience. I look in the mirror every day and see a black man scientist. I see hundreds of them on Facebook.

Two, there was a conflation of race and poverty. I grew up middle class. I'm an "n" of one and recognize that a fair number of peers had greater proximity to economic struggle, but that's definitely not the case for all underrepresented scientists.

There was also the idea that we were all at minority-serving institutions. These are great institutions. However, I had many underrepresented colleagues at places like Stanford, Harvard, Hopkins and Duke.

Specifically, I had three black women friends who completed their Ph.D.s at elite East Coast institutions and who had first-author papers in (the Proceedings of the National

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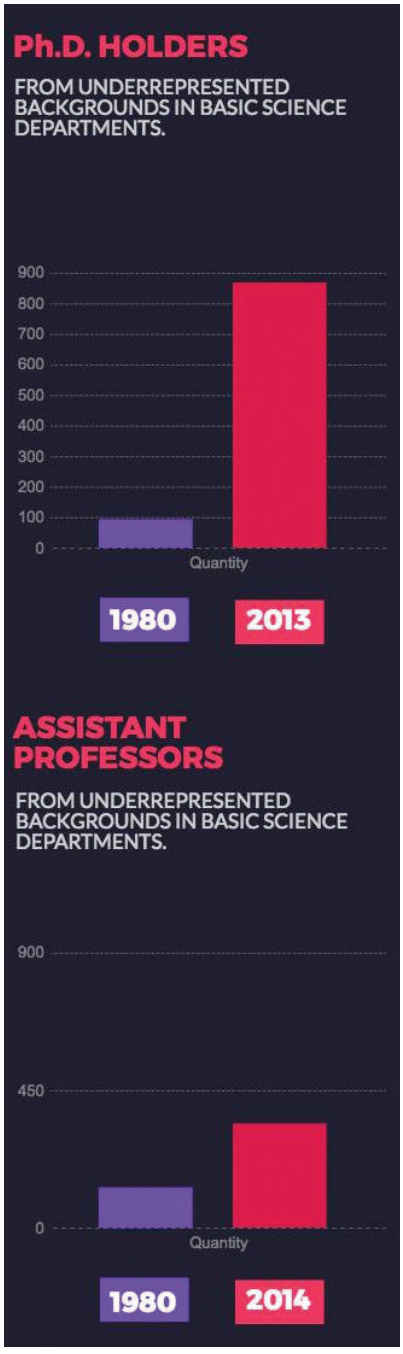
PHOTO COURTESTY OF KENNETH GIBBS

Kenneth Gibbs Jr.



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Academy of Sciences), Nature and Science. However, their graduate school experiences were so challenging and, frankly, hostile that at the end two of them said, "I'm completely out of science," and the other one said, "I will do research, but I'll never stay in academia." This is a loss for all of us, because these excellent scientists are accomplishing at the highest levels of our field, and they don't feel there's a place for them within our system.

Then there was one of my white male colleagues, probably one of the smartest people I've met in science, who chose to start a business instead of pursuing academia. I began to wonder: Why is anybody doing what they're doing? Are the reasons that my white male colleagues are moving out of science the same reasons as those of these black women that I know? Why are these postdocs staying for six to eight years to try to get a faculty position?

I recognized that I could not change everything, but we can illuminate what is happening. Change happens when people start thinking about the world differently. I decided I'm going to research it.

Tell me about your own education and career path.

I am a black American whose family has been here for centuries, meaning slavery and Jim Crow are part of our history. My grandfathers had fourth- and eighth-grade educations, and my grandmothers both had high school diplomas. My mother and father were the first people in their families to graduate from college, and that set up a worldview for me in which education was very important.

I grew up in Durham, North Carolina, the Research Triangle Park. I did lots of internships in high school and got really hooked on the idea of scientific research and what scientific research can do to be helpful to humanity as a whole, so I pursued a career as a scientist.

I went to the University of Maryland, Baltimore County, as a Meyerhoff scholar. I also had funding from the (Maximizing Access to Research Careers) program at NIGMS, and the Howard Hughes Medical Institute.

I got a biochemistry and molecular biology undergrad degree and did my Ph.D. in immunology at Stanford University, where I focused on intersections of stem cell biology, signaling biology and cancer biology, at which point I had my existential crisis.

You weren't sure if you wanted to stay at the bench?

Remember, I started doing research as a high-schooler. My first job was working in a lab. I worked in labs at the (University of North Carolina) and at Duke (University) and in the 10th and 11th grades. So I'd been doing research from the age of 15, and I finished my Ph.D. when I was 26.

I wanted to take a step back and see if this was what I wanted to be doing. I can do it, and I enjoy it. But is it a thing that I really want to be doing?

This was before the distress in the Ph.D. workforce was en vogue to talk about. I completed grad school from 2005 to 2010, so this was before it was widely known. But observationally, I could see something weird was happening around people getting jobs. People were doing postdocs for seven years, with Cell papers, and not getting jobs.

At that point you took the AAAS fellowship, which set you off on this policy route.

The more I explored, the more I kept coming back to these really compelling workforce questions.

I paired with a colleague of mine, Kimberly Griffin, who is an education

researcher at the University of Maryland. My goal has been to quantify the qualitative aspects of our enterprise, because scientists respond to numbers, not quotes. We started writing papers and got some support from the AAAS's education unit and the Burroughs Wellcome Fund, and we've written a series of papers on biomedical workforce development (2–4).

Given that you felt the pipeline metaphor was inadequate, did that play into your decision with this eLife paper?

Yes, exactly. I was thinking about it as a system instead of a pipeline. Where do people move in and out, and what are points of leverage that can help achieve certain outcomes?

From an NIH perspective — again, not a policy perspective, but just a factual reality — most of our money goes to medical colleges. And so biomedical workforce diversity is linked to faculty diversity in those specific contexts. I also figured examining these environments could help illuminate what's happening in other contexts, because many scientific environments have these challenges. We focused on medical schools because of access to high-quality longitudinal data.

Which brings us to your findings.

We wanted to know what actually has been happening over the past 30-plus years — from 1980 to 2014 — as it relates to (1) Ph.D. production among scientists from underrepresented and well-represented backgrounds and (2) progression into assistant professor positions in basic science departments. We then used these data to build a conceptual model of the workforce and used the model to test the impact of different intervention strategies in the short and long term.

We used the NSF Survey of Earned Doctorates, which (the Federation of American Societies for Experimental Biology) very nicely compiles every year on Ph.D. production (5), and the AAMC faculty roster for faculty data (6). In 1980, there were 93 Ph.D. graduates from underrepresented backgrounds, and that had grown to 868 by 2013. And in 1980, there were 132 assistant professors from underrepresented backgrounds in basic science departments, and that grew to 341 by 2014.

We see similar trends for scientists from well-represented backgrounds — that is, both populations grew. But then you see that from 1980, the underrepresented student population has grown 9.3-fold, whereas the professor population has grown 2.6-fold. The rate of growth for underrepresented Ph.D. (holders) is much greater than it is for underrepresented faculty.

Already, you could see that the pool is not depleted of underrepresented scientists.

What we see is that the pool of scientists from underrepresented backgrounds has grown almost eightfold in the past 30 years, compared with two-fold growth for the well-represented candidates. The sizes of the pools are different by an order of magnitude, but, all things being equal, as the pools grow, you'd anticipate comparable entry into these faculty positions. That's not what we see.

What we see is that for scientists from well-represented backgrounds, as the pool grows, more people are hired. But there's statistically no relationship between the size of the talent pool for underrepresented scientists and the number of assistant professors hired.

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The number of underrepresented candidates has grown but academe is hiring a smaller portion than it used to.

The nine-fold growth in the number of underrepresented Ph.D.s and the almost eight-fold growth in the underrepresented talent pool suggests that the collective action of the community to focus on and build the underrepresented talent pool has had an effect. This rate of growth is much larger than the growth of those populations in the country since 1980.

What we're seeing, though, is a lack of connection between the talent pool and academic hiring. Between 2005 and 2013, there were close to 6,000 underrepresented scientists who got Ph.D.s in the biomedical sciences, and between 2005 and 2014 there were six fewer underrepresented assistant professors. There's a disconnect that needs to be addressed.

Now explain the model.

The logic of the model is this: You have some number of people who aspire to these positions. They go into the market. And then people are hired based on the number of slots available.

In our model, there is no bias in hiring. People are hired directly proportionately to their representation on the market. So, if there are 90 well-represented folks and 10 underrepresented folks on the market and there are 10 slots, nine go to the well-represented folks and one goes to an underrepresented person.

Historically, there was some number of people who would have become faculty no matter what. There were minority professors in the 1970s when things were really terrible. There's some number that always is there.

We wanted to know how many additional people were over and above what would be expected with overall system growth through time. Using real data, in 2014, 5.8 percent of the assistant professors were underrepresented minorities. And that would be consistent with a 0.25 percent transition rate of URM scientists. That is, even though we're doing a lot to grow the URM talent pool, it looks like very few of these people are transitioning into the faculty pool.

You also tested some interventions, right?

We used the model to test the impact of three interventions in the short and long term. These interventions were: focusing on building the talent pool, increasing the number of faculty positions available, and increasing the transition rate of underrepresented Ph.Ds. onto the faculty market and their subsequent hiring.

All the models showed the same thing. If we only focus on growing the URM talent and let it grow exponentially, by 2080 we would have 73 percent underrepresented minority Ph.D.s but only 8.9 percent underrepresented minority faculty. The low transition rate means that even without active discrimination, the system operates in such a manner that the URM talent pool is disconnected from academic hiring.

To see if it was an issue of the number of jobs, we added 100 new assistant professor positions annually starting in 2015. Even with exponential growth in the underrepresented Ph.D. pool, there's still no impact.

Finally, we examined the impact of increasing the transition rate — that is, getting more URM postdocs onto the market. This dramatically increased faculty diversity, because as more URMs enter the market, more are hired. You only get diversity in the faculty if you get people into the mar-

ket and hire them. Changing the pool or the labor market are not sufficient to increase diversity. We have to think about transitions and hiring.

Bottom line?

Change is possible now. The reality is that there are about 1,000 assistant professors hired every year across the country. If you hire around 1,000 people a year, and you want 10 percent to be people from underrepresented backgrounds, which would match the Ph.D. pool, then that would mean hiring 100 underrepresented minority assistant professors a year.

We graduate close to 900 (underrepresented) Ph.D.s a year.

All of this is to say that we sometimes talk about issues of diversity as if they cannot be fixed. This can be solved with around 100 people.

We assumed (in the past) that growing the talent pool would be sufficient to right the system. This (paper) calls that into question. It speaks to the need, I think, to recognize that this is happening and then that there can be systemic architecture issues going on that are outside of the motives of any individual person's heart. Those issues need to be addressed so that this disconnect doesn't continue.

How hopeful are you that institutions will do this?

I believe that it can be done. We live in an interesting cultural moment. Science is not immune from the broader societal challenges as they relate to issues of race, and lots of institutions are examining if their practices are keeping up with broader changes in the world. It is possible, though it will take changing the way we've always done things.

Ultimately the goal is to enhance scientific excellence through diversity. If large sections of the workforce are disconnected from academic science, scientific excellence suffers. This paper focused on scientists from underrepresented backgrounds, but many of these issues also apply to women from well-represented backgrounds.

You are careful not to call your projections as "quotas."

Correct. It's a mathematical reality that it takes around 100 scientists from underrepresented backgrounds each year to reach parity with the Ph.D. pool.

You're not making, then, recommendations.

I am describing reality with the hope that we can have a broader conversation in the scientific community about how we ensure that we can effectively utilize the diverse talent pool that we have before us.

Medical schools could commit to doing this, if they really wanted to.

I think it's important for all actors in the system — individual scientists, institutions, funding agencies, scientific societies — to think about how they can individually and synergistically work to address the issues that have been illuminated by this paper.

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Meet Anne-Claude Gingras

One of the two new deputy editors for Molecular & Cellular Proteomics, she is a signaling soothsayer and candid Québécoise

By John Arnst

Anne-Claude Gingras is a senior investigator at the Lunenfeld–Tanenbaum Research Institute in Canada. Gingras started her lab at the institute in late 2005 and has won several awards for her research involving signal transduction and mass spectrometry. In addition to her role as a senior investigator, she is a director of the institute's proteomics group. This past fall, she and Steven A. Carr at the Broad Institute became deputy editors at the journal Molecular & Cellular Proteomics, where the current editor-in-chief is Al Burlingame at the University of California, San Francisco. Gingras spoke with John Arnst, ASBMB Today's science writer. The interview has been edited for clarity and length.

PHOTO COURTESY OF ANNE-CLAUDE GINGRAS

Anne-Claude Gingras

What are the projects that your group is working on?

My research is mostly focused on understanding how proteins essentially associate with one another to perform their activities and how these associations are perturbed by changes in the signals that the cells would receive. I'm also a full

professor at the University of Toronto.

What was your research training?

I did my Ph.D. at McGill University in the department of biochemistry. I worked on translation initiation. I was characterizing how a certain translation inhibitor called 4E-BP1 was able to receive signals from the insulin and growth receptor pathways to regulate translation initiation. This process is involved in different cancers and neurodegenerative diseases, and it's also targeted by viruses.

I graduated in 2001, and I started my postdoc in 2002. I wanted to do more of a technical postdoc, as I had a pretty good basis in fundamental biochemistry. I decided go to the lab of Ruedi Aebersold, who, at the time, was in Seattle. Ruedi was a rising star in proteomics. Now he's at the top of the world. He had just published a proteomics method called ICAT that enabled researchers to compare the abundance of peptides in two samples. What I wanted to do was to continue to work on the characterization of signaling pathways that impinge on translation but bring a proteomics component to it. I started in Ruedi's lab in 2002, and I worked there for just over three years.

What made you choose science as a career?

I did my bachelor's (degree) in Quebec City at the University Laval. It was in biochemistry. I worked in the lab one summer before the school year, and I realized I wanted to do research as a career. The mentor whom I had in the lab, André Darveau, was fantastic and spent a lot of time training me. He's now the dean of the science and engineering faculty at Laval University.

After that, I realized that I probably should learn English. I thought McGill was a good compromise, because in Montreal, you can still live in French and work in English. I did my Ph.D. with Nahum Sonenberg. Nahum's lab had discovered the internal ribosomal entry site that many viruses use to initiate the translation of their own proteins. He had also discovered and cloned eIF4E, the protein that binds to the cap structure of the cellular mRNAs.

At the time when I joined, the lab had just cloned the first two inhibitors of translation that block the action of eIF4E. Arnim Pause, the student who cloned it, was leaving for his postdoc. I was lucky. I walked in the lab, and they said, "OK, new girl, your job is to finish the experiments for that paper that we're going to be submitting." They submitted it to Nature a few weeks after I started in the lab. When the paper came back, I had to help with the revisions, so I got to be on a Nature paper within a few months. It was really cool, and it was super motivating. Of course that didn't happen every week after that.

One of the things that I learned from Nahum is if you have somebody in your lab who's good, you need to let them come up with their own ideas and try them, because any given person will only have a limited number of good ideas. Nahum would leave the students and postdocs to essentially come up with crazy ideas and test them.

We had a case where we needed to map phosphorylation sites. There was this new professor at the time in Vancouver, who was Ruedi. (Author's note: Ruedi Aebersold was at the University of British Columbia from 1989 to 1993.) We sent him some of our samples to do Edman degradation, and that's how I got connected (with Ruedi). We then collaborated on several projects, eventually using mass spectrometry to study phosphorylation of 4E-BP1 and other proteins. That got me very interested in proteomics, and I knew I really

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liked Ruedi personally, so the decision to do my postdoc there was quite natural. I never applied for any other postdoc position, never went to an interview or anything like that, so it's quite unusual in that way. I felt like that was the right thing for me to do at the time.

Do you have any words of wisdom or a favorite motto for scientists in training?

There is one thing that I tell them: Be nice to everyone, because this is a very small world. If somebody needs help, try to guide them the best way you can, whether it's somebody in another lab or elsewhere. It's going to be these people coming back in your life later on.

I think when people join a lab after doing a Ph.D., sometimes they've been in a place where they were told to be selfish. It's such a collaborative world right now. The sooner you learn to be nice to people and work well with people and to share everything — the credit for intellectual contribution and reagents — everybody wins.

How's the new role at MCP going so far?

Having Steve and me joining at the same time is really good, because it enables us to re-energize and rethink the structure of the journal. Al has been really open to work as a trio of editor-in-chief and deputy editors, so it's been great so far. I think it's going to keep getting better.

What was your involvement with MCP prior to this?

I was one of the most used reviewers on the editorial board. I was dealing with a lot of the papers that had to do with protein interactions, cell biology and so on. And, of course, I was an author. I have several papers in MCP.

What was your reaction when they asked you to take over?

It caught me a little bit by surprise. My initial reaction was that I didn't want to have any more work, but then I thought, "Yeah, I have some ideas of how to improve the journal. This is the best journal in the field, and it's important for proteomics that people actually get involved and agree to step up and contribute."

Do you have any hobbies, or advice for balancing life in the lab with life outside it?

My significant other is also a scientist running his lab. If anything, he works more than me. We don't have a super balanced life. We do things like hiking, but we don't have kids, so that makes it easier for me to commit to something else. That's something that I fully realize is hard for some of my lab members with families.

I give a lot of courses, seminars and conference presentations abroad, so I travel several times a month outside of Toronto.

What's the most interesting place you've traveled to in the last year?

Just a couple of weeks ago, I was in southern Chile in this nice area that's north of Patagonia, with this beautiful volcano and lake. It was spring there, and it was beautiful. It's a nice opportunity when you're a scientist to travel to a lot of cool places, but it's something that most people who have a normal job don't get to do.

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Meet Steve Carr

One of the two new deputy editors for Molecular & Cellular Proteomics, he advises "to be in a field that is at the intersection of at least two different areas"

By John Arnst

Steven A. Carr, director of the Proteomics Platform at the Broad Institute of MIT and Harvard, became a deputy editor at the journal Molecular & Cellular Proteomics along with Anne-Claude Gingras at the Lunenfeld–Tanenbaum Research Institute in Toronto this past fall. Carr has been an associate editor with the journal since its inception in 2002. The journal's editor-in-chief is Al Burlingame of the University of California, San Francisco. Carr spoke with John Arnst, ASBMB Today's science writer. The interview has been edited for clarity and length.

Tell us about the work you're doing.

The research in my lab focuses on developing and applying technologies to quantify proteins, their modifications, and their interaction partners in stages of health, disease and other perturbation conditions. We do that work to try to understand the function of the proteins and their response and resistance to drugs.

My group also has a major focus on discovery and quantitative verification of biomarkers for major diseases, such as cancer, cardiovascular disease and infectious disease. We put a lot of time and effort into pushing new technologies in that area as well.

What was your background and research training?

I did my undergraduate work at a college in upstate New York called Union College. I was a chemistry major, and I thought I was going to be an organic chemist, so I went to (Massachusetts Institute of Technology) for graduate school and began work in a laboratory doing organic chemistry with Klaus Biemann.

I then did a postdoc with Vernon Reinhold at Harvard Medical School. I realized that mass spectrometry's primary use at the time was for analyzing post-translational modifications. The most difficult of those at the time was glycosylation. It remains probably one of the more difficult areas of post-translational modification to make any headway in.

I left Harvard Medical School to go to what was, at the time, Smith, Kline & French laboratories down in Philadelphia. They were just beginning to acquire very significant amounts of money from their H2 antagonist, Tagamet, which became, I believe, the first billion-dollar drug on the market. They were building a huge facility out in King of Prussia, Pennsylvania. They were an old, sleepy pharmaceutical company, but they hit on Tagamet, and they basically decided to completely revitalize and reorganize the company.

Hepatitis B vaccine was the very

PHOTO COURTESY OF STEVEN CARR

Steven Carr

first thing that I worked on when I went there, and it was really a thrilling process. I went back and forth from Philadelphia to Ricksonfort, Belgium, where our vaccine affiliate was located, and was involved in all of the initial characterization work of that vaccine product. What was very interesting was that protein was extremely hydrophobic.

Mass spectrometry was ideally suited for this. We covered a very high percentage of the protein in our regulatory filing, and it really helped the company get the vaccine on the market when we were in hot competition with Merck. It turned out to be a big product for the company, partially because the World Health Organization came around and said that all children have to be vaccinated with hepatitis B vaccine. That helped both Merck and Smith Kline.

That was maybe an unexpected and unusual win for a drug company at that time, because it was a very fast timeline from the start through to completion. That never happened again for me, so in many ways it was setting me up for failure further on. Many disappointments in projects occurred after that point and taught me that research is really hard and failure is the most common thing that you're going to have to learn to deal with. And keep your chin up.

Is there any advice you would give to young scientists?

There are two pieces of advice. One is that it's really important, if you can manage it, to be in a field that is at the intersection of at least two different areas. Mass spectrometry was kind of its own specialization, but applying mass spectrometry in biotechnology was that junction. It just makes you a lot more valuable (as a researcher) and provides the most interesting problems to work on. The other piece of advice is that life's long and work is a huge part of what you do, so you better care

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about what you do; otherwise, change what it is you're doing.

What made you want to become a scientist?

I credit my parents. Neither of them had a high-school education. They were forced as a result of the Depression to leave school and go to work. They impressed on me that I had to get an education. We ran a television repair shop out of my parents' house where I lived in Putnam Valley, New York. I did work in that shop alongside my father and learned a fair amount about electronics. He was entirely self-taught. Those were the sort of things that rubbed off on me — the general curiosity about how things work and why and this push toward education. Chemistry was originally the thing I wanted to do more than anything, partly because I had chemistry sets when I was a kid and did a lot of fooling around in the basement with them.

When I went off to college, there were a couple of really good professors at Union College that took a special interest in me. They helped me understand that I could actually apply to graduate school, I could go beyond just getting a college education, I didn't have to go out immediately and try to get a job after college.

How is the new role at MCP going so far?

I think it's going well. I feel empowered a bit more to help make decisions and move the journal forward into the future. There's a number of things that are still under discussion that I think are going to make MCP even more attractive to the community of proteomics scientists as a place to publish. Those will get rolled out in the not-too-distant future.

What was your reaction like when you were asked to be a deputy editor?

I was honored, of course, and felt empowered to speak up at an even greater level than I had previously. It's a pleasure working with Al and Anne-Claude. I think that we're a terrific team, and we've gotten off to a great start.

Between your lab work and your MCP duties, what do you do outside of the lab?

I have done a lot of woodworking in my time. I haven't been able to do as much lately, but I still consider that a hobby. I am a scuba diver. My son and I, whenever we have the chance, go off to some exotic location and go diving together. We've gone to Bonair several times (Author's note: Bonair is in the southern Caribbean). We've gone to Hawaii. The South Pacific, specifically Palau, is on the bucket list. That area is fantastic not just because the coral is still reasonably intact in those areas but because there are a lot of wrecks, unfortunately, from World War II. The wreck diving there is really, really good.

Do you have any advice for balancing your life in the lab with life outside of it?

Balance is definitely the wrong word. I think it's more like a teeter-totter. You sit on the work end, you hit the ground and you're sitting there for a while, laboring away at work, and suddenly you realize that, wait a minute, that other part of your life, which is the other end of the teeter-totter, is up in the air and you haven't dealt with it. You have to rebalance. I go traveling with my wife, and I go for long hikes with her. That's how I try to provide some balance.

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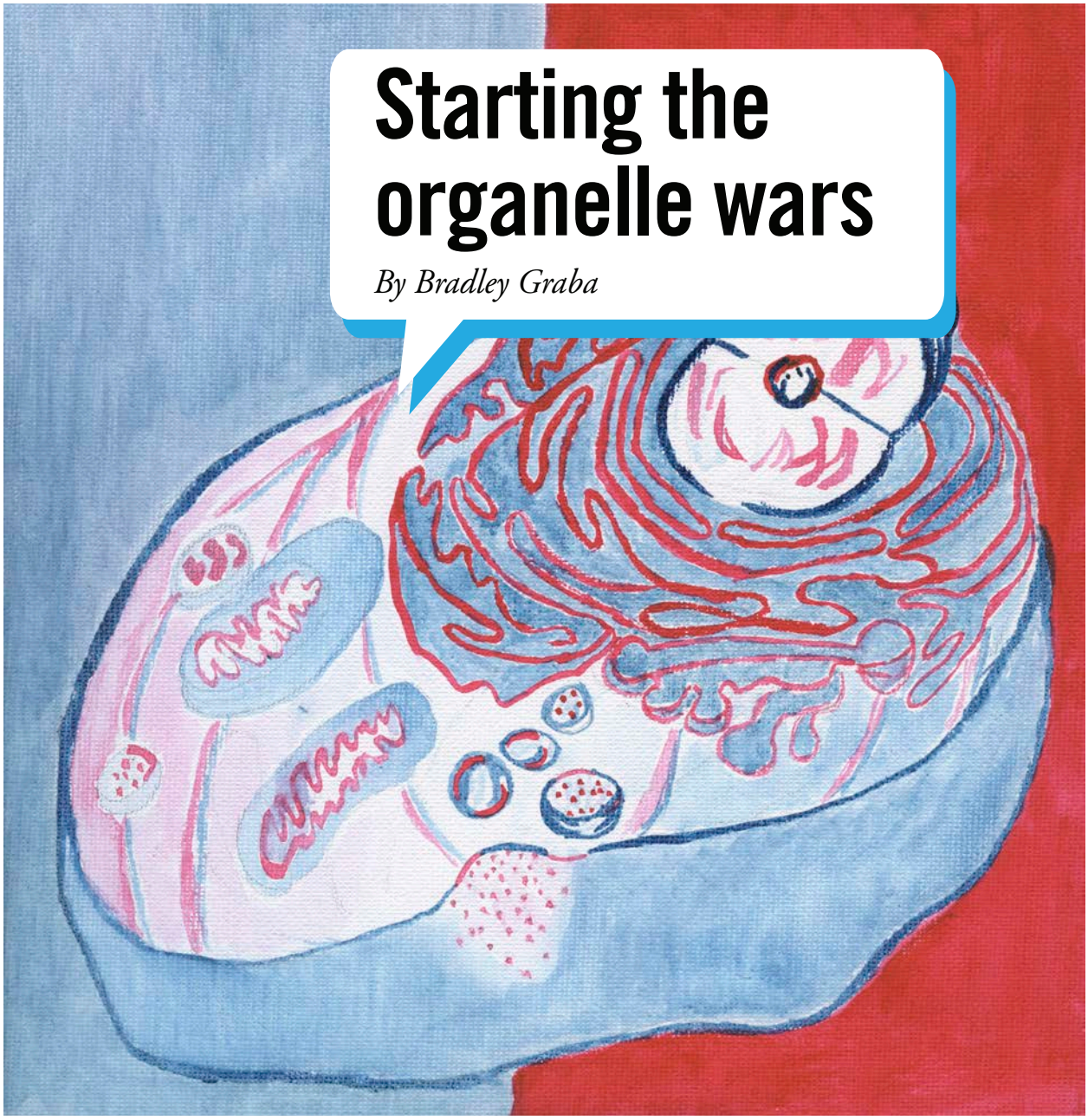

Starting the organelle wars

By Bradley Graba

IMAGE COURTESY OF ANDIE EVANS, A SENIOR AT WILLIAM FREMD HIGH SCHOOL

In 2011, I was looking for a way to invigorate teaching cellular organelles to high school freshmen.

I am lucky enough to teach at William Fremd High School in Palatine, Illinois, which is about 45 minutes northwest of Chicago. For me, teaching is a family affair. My mom was a third-grade teacher for most of her career, my dad was a junior-high science teacher and department chair, and my brother is a physics teacher and head of the math and science department at Crystal Lake South High School in the Chicago suburbs.

The community teachers and students are proud of our school, where 97.1 percent of our students graduate and 84.3 percent of our students are college-bound. The students have an average ACT score of 25 and, most importantly, they are motivated, respectful kids who are fun to work with. The overwhelming majority of our parents are supportive and involved in their kids' education. I myself graduated from Fremd in 1994 and then was hired back to teach there in 1998 after graduating from Illinois

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Wesleyan University with a bachelor's degree in biology with a minor in secondary education.

I was struggling to teach the unit on cellular organelles, because the unit can be a tough one to get students excited about. I had tried more cell-organelle projects than I cared to count over 13 years. So, in 2011, like most resourceful teachers, I went to the internet to find inspiration. There, I came across a project by Marna Chamberlain at Piedmont High School in California that piqued my interest.

Chamberlain had her students complete a project that involved campaigning for a cell organelle to be elected the most important organelle. I immediately fell in love with the idea of having my students run a campaign. The wonderful part of the project was that students not only had to promote their own organelle but also had to run a smear campaign against five other organelles. This requirement served the purpose of making sure that the students learned about more than just their own organelle.

One of the other requirements of the project was that the students create extra campaign materials, such as fliers, shirts and bumper stickers. Here, I added my own twist to the project. I encouraged the students to take their campaign to social media by creating Twitter accounts for their organelles. Since each account was in the name of the organelle and not in the name of the students, I did not hear any complaints from students or parents about creating the account. In addition, because the use of social media fell in the extras category, students could fulfill the requirement for that category of the project without using Twitter if they or their parents were not comfortable with the idea. The reason for adding the twist was pretty simple. My students were always on their phones and on Twitter anyway, so why not take the project to where my students were spending a lot of their time?

The use of Twitter put the project over the top. In 2011, Twitter wasn't as powerful as it is today. My students followed each other's accounts, and I followed them all to monitor the accounts. Everything stayed within the small circle of students in my class.

The next year was an entirely different story. It was an election year with President Barack Obama running for re-election against Mitt Romney, so the class was already buzzing with a little more excitement than usual when it came to campaigning. On the first day of the project, one of my groups mentioned that someone they didn't know was tweeting at them. Being relatively new to Twitter at the time, I was concerned as to who might be interacting with my 14-year-old students. It turned out that the person tweeting them was a researcher from England who specializes in studying the Golgi apparatus. Her name is Anne Osterrieder from the Oxford Brookes University, and she had been searching for information about the Golgi apparatus on Twitter when she came across my student's Twitter account.

At that point, the project exploded. Osterrieder and her colleagues began tweeting with my students' organelle accounts, prodding them with higher-level questions, holding them accountable for spreading misinformation, and engaging them in a way that my students had not expected from

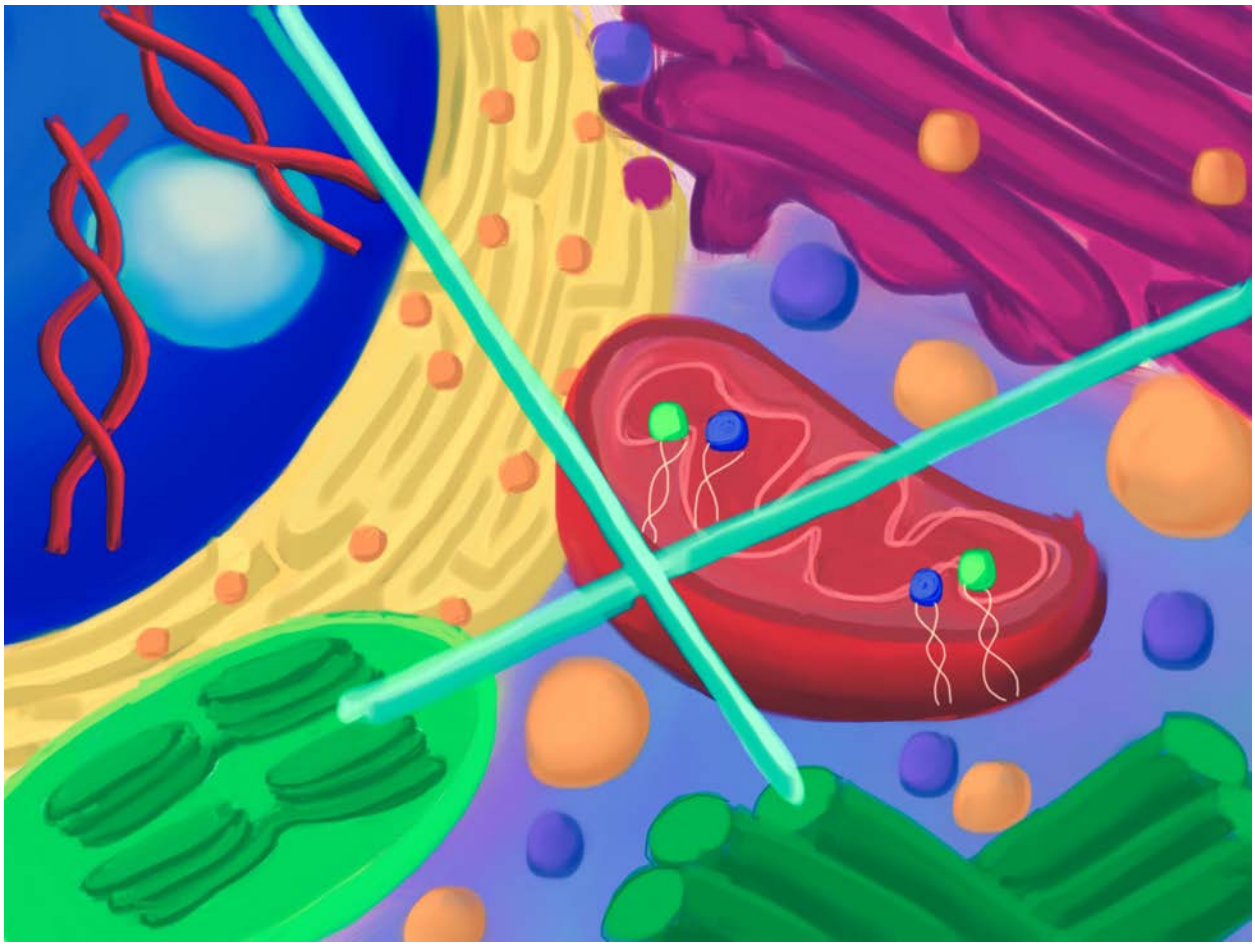

IMAGE COURTESY OF ADHITH PALLA, A JUNIOR AT WILLIAM FREMD HIGH SCHOOL

a freshman biology project in high school.

Soon we had scientists from around England, France and the U. S. tweeting with us. One of those scientists, John Runions, also of Oxford Brookes University, has his own BBC radio show under the persona of "Dr. Molecule" and discussed our project on his show. Runions also is the person who developed the #organellewars hashtag.

One of the main outcomes of the project was that my students realized that what they tweeted was going to be fact-checked. They began using Google Scholar and citing their sources, because they had a live and knowledgeable audience watching their every move. The quality of information being produced by my students about their organelles and the ones that they were smearing improved as the project continued, as did their knowledge of the structure and function of all of the cell's organelles.

Over the past four years, one of the big changes I have made is that I have required that smears of other organelles not be related to diseases that are caused by problems with the organelle. I found that students spent time researching the names of diseases that were caused by organelles but were not focusing on the specific role of the organelle in the disease. Now the smear campaigns must relate to the structure and function of each organelle, not the diseases it causes. The smear campaigns definitely have become more useful in terms of student learning. This year, it also was interesting to see the number of made-up statistics students would add to their tweets about the number of people supporting them as well as the use of monikers. The trends of the 2016 election definitely made their way into our classroom election.

The #organellewars project has changed the way I approach teaching high-school students about cellular organelles. It allows my students to interact with scientists who are experts in cell biology. It holds them accountable for learning about their organelles, and it injects an element of fun and excitement into my classroom that had been missing with other cell-organelle projects.

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Pixel perfect

By Kaoru Sakabe

When the results started rolling in and a story began emerging, my thesis adviser usually instructed us to start assembling figures for a paper. At first, it was daunting to see blank figure panels nestled between the data we had. But the process made it easier to identify missing experiments and to see the logical progression of the story as the holes started to fill in.

With so much attention focused on building a scientific argument, the last thing on my mind while assembling figures was the final figure resolution. However, forgetting to keep resolution in mind from the start can cause problems later on. To avoid any potential issues down the road, I offer a few tips.

Let's start off with some basics. When reading submission guidelines for journals, they often throw around terms, such as "minimum resolution," "dpi," "ppi" and "vector graphics," which all seem irrelevant when you are eager to write up your manuscript. So what is a pixel, the first "p" in "ppi"? A pixel, derived from "picture element," refers to the most basic unit composing an image. Each pixel contains information telling the computer what color or shade of gray to display.

The film you have scanned, the immunofluorescent image you've snapped or the Western blot image you've exported from an imaging system — that image is composed of many pixels arranged in an x, y grid such that the final image will show coimmunoprecipitation of your protein of interest or mislocalization of your protein upon treatment with an inhibitor.

The resolution of the image refers to the density of pixels. It is the number of pixels that make up your image. The greater the resolution, the more information an image contains and the clearer your image will be. This quantity is expressed as pixels per inch, or ppi. For publication purposes, most journals will require that you submit your final figures with a minimum resolution of 300 ppi. You often will see dots per inch, or dpi, used interchangeably with ppi, but dpi actually refers to printer output, or how many dots of ink are found per inch of a printed document. Since we are talking about digital images, ppi is the more relevant term to use.

The last bit of information you need to know is that your image data can be either raster or vector data (Figure 1). Raster data is simply an image made up using pixels as building blocks as discussed above. Vector data, on the other hand, is not composed of pixels but rather is a set of instructions that tells the computer to display lines and curves. This type of data is useful for graphs or models, since it remains smooth no matter how much you zoom in. Conversely, raster data

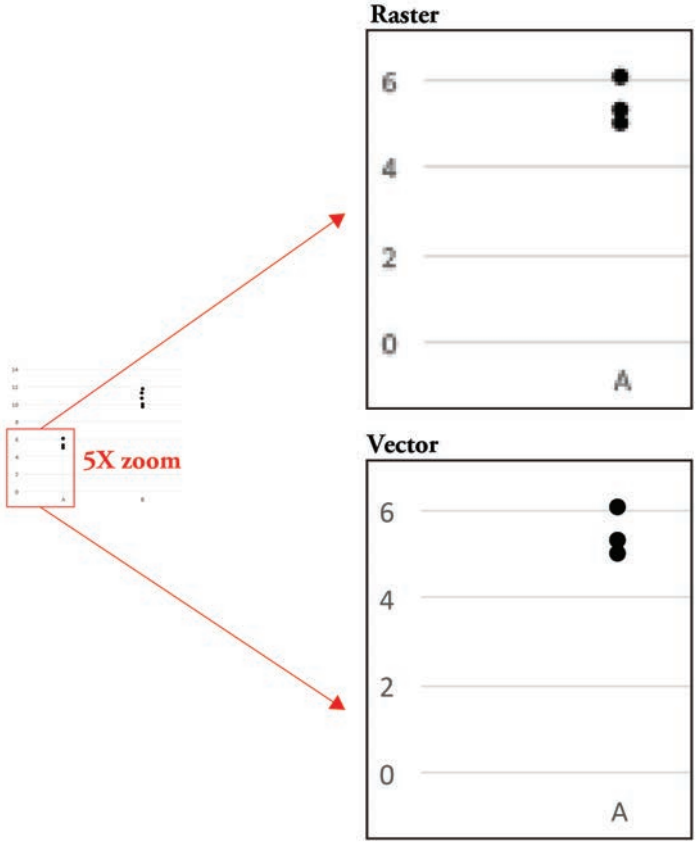

Figure 1. Raster data vs. vector data. Raster data becomes pixelated as you zoom in, whereas vector data remains clear.

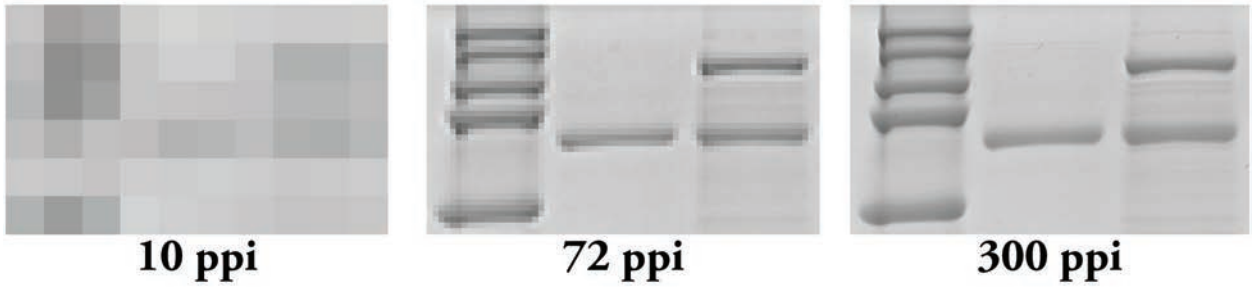

Figure 2. Resolution matters! Even if you scan your film at 300 ppi, if you use PowerPoint, you effectively are changing it to a 72 ppi image.

becomes pixelated as you enlarge the image, making the gridlike pattern of pixels obvious.

OK, we've got the basics. Now how do you apply this information to create awesome figures?

Tip 1: Remember that figure preparation begins at data acquisition. Make sure you are acquiring your image at the proper resolution. Whether you are scanning a film or exporting a file from an imaging system, keep the minimum resolution of 300 ppi in mind. It's never fun when you realize that you have to find a particular film to rescan at the proper resolution months (or even years) after you performed the experiment, or worse, conduct the experiment again if you can't find it. For graphs, make sure you are exporting the data in nonraster format, such as *.pdf, *.eps or *.svg. Exporting in these types of formats will prevent your graphs from looking pixelated and keep text legible no matter how you resize it later.

Tip 2: Use appropriate software when laying out your figures. PowerPoint may be user-friendly, but it is meant to work at screen resolution, which is only 72 ppi. When you export images from this program, you end up with a 72 ppi image that needs to be converted into a 300 ppi one (Figure 2). Conversion to a higher resolution image can result in an image that is too small for publication or one that is extremely pixelated. Additionally, depending on how you upscale, or increase the number of pixels in your image, your software program may introduce pixels into your image, thereby creating artifacts. Adobe Illustrator usually is recommended for figure assembly, but Inkscape and CorelDraw are good alternatives. These programs are meant to combine raster and vector data into a single figure and can do so without affecting the pixels found in raster data.

Tip 3: Set your canvas size to the physical dimensions provided by the journal. Most journals provide two or three size options: single-column width, double-column width and occasionally 1.5-column width. Once you insert your graphics into the figure using the appropriate software, you usually don't have to worry about image resolution; however, be careful when increasing the size of a raster image. If you insert a 300 ppi image and decide you want to double its size, the resulting resolution of that image will be 150 ppi and likely will look less clear than the original.

Keeping track of image resolution shouldn't be a hassle. By incorporating these suggestions into your workflow, you can rest assured you have done your due diligence.

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Effective communication: dream or reality?

By Geoff Hunt

"Science communication." It is a phrase that is both celebrated for its importance and reviled for its ambiguity. Scientists are encouraged to communicate often and effectively to help gain support for their research. At the same time, there is extensive debate among science-communication researchers and professionals about what communication is, what the goals are for different communication efforts and what approaches are most effective.

So it was with great fanfare and anticipation that the National Academies of Sciences released a report in December titled "Communicating Science Effectively." Stakeholders hoped the report would provide clarity on best practices in science communication and illustrate a path forward for the field.

Unfortunately, little within the report points toward immediately actionable suggestions. Instead, the main takeaway is that more research is needed into several questions related to science communication: How do social factors influence trust in science? How can (and should) science affect policy debates? What is the best way to communicate scientific controversies?

Looking to obtain more information about the report's intention and expected outcomes, several leaders within the science-communication field gathered in January for a public hearing with the report authors in Washington, D.C. The CEO emeritus of the American Association for the Advancement of Science, Alan Leshner, chaired the committee that wrote the report. At the hearing, Leshner stressed the timeliness of the report, emphasizing how effective communication is necessary to ensure that science continues to play a vital role in current events. Committee vice chairman Dietram Scheufele, professor at the University of Wisconsin–Madison, pointed out that the report was particularly relevant for the new generation of scientists who are more comfortable with communicating to broad audiences than previous generations but who still need direction on how to do so effectively.

Despite the optimism exuded by the authors, several within the community remain unconvinced. Rick Borchelt from the Department of Energy Office of Science asked during the hearing what the report added to the field of science communication, citing publications going back at least 15 years that made similar recommendations about doing more research into effective communication approaches. Other commentators also wondered aloud what agency or institution would fund the ambitious research agenda. To generate momentum from the report, the NAS announced that it will host the "Science of Science Communication" colloquium in November to explore these issues further.

But what can organizations and individuals do to help advance the cause of science communication? While nowhere near as ambitious as the research agenda laid about in the NAS report, one small step that we hope members of the American Society for Biochemistry and Molecular Biology will take is to start publishing the broader impacts of their work. Any researcher applying for funding from the National Science Foundation has to include a description of how he or she will demonstrate the broader impacts of the proposed research on society at large. Unfortunately, researchers rarely publish these types of results. When they do, the publications usually are confined to education-focused journals, thereby bifurcating the scientific research and science-education communities.

The ASBMB Public Outreach Committee wants to bridge this bifurcation to help connect scientists and science communicators. We are looking to collect and disseminate descriptions of broader impact activities that stem from research publications. Our motivation for this effort is straightforward: to provide ASBMB members with outlets that will help them communicate their science. If you have published an NSF-funded research paper within the past 12 months and want to share your broader impacts story, send us a message at outreach@asbmb.org.

The more we can generate buy-in and support within the research community for science communication, the stronger the argument becomes that communicating science effectively needs to be a standard part of the scientific process.

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

Is biochemistry a tool or a discipline?

By Binks W. Wattenberg, Enrique M. De La Cruz & Daniel M. Raben

Spoiler alert! The answer to the question posed in the title is clear: It's both.

But the distinction is important. When asking this question to new graduate students or some seasoned investigators, it's not unusual to hear them declare that biochemistry is "just a tool." Indeed, biochemists have developed methods and reagents that are essential for other disciplines. But biochemistry is more than just a collection of techniques. As a discipline, biochemistry is characterized by the mechanistic insights and predictive power that it produces.

The confusion inspired by the question is partly due to the fact that, as a distinct discipline, biochemistry seems on the verge of becoming a victim of its own success. This success is largely due to technical approaches and intellectual discoveries that have cracked questions of fundamental and clinical importance in biomedical research. Neuroscientists, immunologists, cancer biologists, structural biologists and physiologists, among others, all take advantage of these biochemical approaches and discoveries. However, this prompts the question as to who exactly is a biochemist and, relatedly, whether it makes sense to have a discipline devoted to biochemistry.

For example, what is the difference between a neuroscientist or pharmacologist using biochemical tools and a biochemist working in a neuronal system or studying a pharmacological problem? This question highlights the fact that scientists often identify themselves with the system they are studying. Take the individuals who exploit

proteomic and lipidomic techniques and enzymatic assays to examine the interactions of drugs or toxins with specific cellular components. They would identify themselves, validly, as pharmacologists despite the biochemical underpinnings of their studies.

Strictly speaking, a biochemist is someone who studies the underlying chemistry of biological processes and systems. Traditionally, biochemistry encompasses the study of the chemistry essential to biological processes including, but not restricted to, enzymes, metabolism and signal transduction. Importantly, the use of purified components and cell-free systems for these studies has been a defining hallmark of the biochemistry discipline. Dyed-in-the-wool biochemists focus heavily on defining the chemical mechanisms that drive the biology. They don't identify with a single biological system, even though they may have chosen to focus on one for their studies. It is this mechanistic focus that defines the biochemistry discipline and has resulted in key intellectual discoveries and technological advances.

It is essential to have a standalone community of biochemists. These are the people who want to understand the chemical details of life in all its forms. It is important to be able to ask a colleague about the affinity of a substrate in an obscure reaction and to have that question taken as a serious challenge and not an annoying detail. We need a space where understanding DNA repair in a deep-sea mollusk is given the same weight as in a mammalian cell.

Maintaining biochemistry as a discipline is essential to the progress of all the biological sciences. We need the intellectual and physical space to delve deeply into mechanisms. Time and again, these mechanisms have been shown to be broadly applicable. Biochemists develop the critical tools to illuminate these mechanisms that ultimately we will share with our colleagues in different disciplines. As biochemists, these tools are one of our major contributions to the broader biomedical research enterprise. If scientists want tools, they will want to keep and respect biochemistry as an independent discipline.



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Working at Manylabs, an open science space

By Gary McDowell

Leaving the laboratory can be a daunting prospect for a variety of reasons, not in the least that the lab environment itself can provide a unique stimulating experience, with people around you carrying out experiments and discussing science. For this reason, when I decided to leave my postdoctoral stint at Tufts University to begin working full time for Future of Research, known as FoR, and follow my husband to San Francisco, where he was doing his medical residency, I wondered what kind of working environment I might end up in. It could have been at home or at a rented cooperative working space. In moving away from working at the bench, I could, as long as I had my computer, work pretty much anywhere, but it was important to me to have a desk outside my home and be surrounded by other people.

PHOTOS COURTESY OF GARY MCDOWELL

Cere Davis showcasing her artwork at Manylabs

My boss, Jessica Polka, had passed along an opportunity she had spotted in a tweet for a residency in a unique working space called Manylabs. A nonprofit organization supported by the Gordon and Betty Moore Foundation, Manylabs aims to provide a space that brings together scientists, educators and "makers" who are working on open-source tools for science.

Transparency is central to the mission of FoR. Our mission is to make the research enterprise more transparent to junior researchers by analyzing and providing data on career outcomes, salaries, fellowships and training opportunities. We also are interested in fostering a scientific enterprise that recognizes, enjoys and benefits from the diverse ways science can be practiced and used to contribute to society, and in finding out more about the ways science happens outside academia. Additionally, FoR is dedicated to helping junior scientists practice open science safely, making the data, research and educational resources they produce freely available to improve transparency and assist in reproducibility. Therefore, Manylabs seemed like an ideal environment to maintain my comfort with the lab but also to explore a less conventional workspace. My application was successful. I am now in my second six-month residency.

Our space matches the stereotypical picture of a San Francisco cooperative working environment. We are housed in a warehouse on Folsom Street in the SoMa neighborhood, with desk space on the top floor, desk and lab space on the floor below, and more lab space and a large workshop space on the ground floor. The workshop space is a useful facility to have as I start to do more workshop/meeting-based work with the local scientific community. The residents hold open house events where we showcase our work and give demonstrations to the community.

There's an incredible range of work going on at Manylabs. There are people using science in art, groups installing air- and water-quality monitors around the Bay Area, computational scientists creating open-source tools to map food webs, organizations driving open data-based environmental advocacy, and groups working on local community outreach around natural history. There are those developing innovative educational tools, such as kits for students to build to learn a variety of science and engineering lessons. There's a movement toward facilitating citizen science in the lab spaces. Some of the work of the people at Foldscope takes place here too, in trying to distribute affordable devices for science around

Eric Mandu gives a lighting talk about an aquaponics system at the Manylabs Open House in October.

the world.

As it's usually billed as a makerspace, I sometimes feel a little like an imposter to be hanging around working on policy and advocacy, but at the same time, everything that is happening here is also central to helping me understand the barriers to noninstitutional science and the different incentive structures that are directed more toward local educational or environmental effects than the traditional publication structure of academia.

Of course, the one thing that feels familiar is the search for funding. Being in a nontraditional space in science means looking for nontraditional funding sources or fundraising efforts. I am currently fortunate to be supported by a grant from the Open Philanthropy Project. Some in the space have to work elsewhere to support their nonprofit work, so there are people who I see through the day regularly and some I hardly see at all, depending on who has what kind of support for their work and when they can be around.

We get together weekly for "T++," which is a reference to the computing language, but instead we have tea and discuss what is going on with our work. We also update the Manylabs website with monthly updates both for the public and for each other. It's also a chance to see how we can collaborate with each other.

The major challenge for me has been moving from a hierarchical structure like academia into a very unstructured environment. No one technically is in charge. It has benefits and downsides. But that soon will change, as the group has hired a community manager to provide support for the organization and the people within it. Because we rarely are all together, it can be something of a challenge for everyone to know what everyone else is working on in great detail. The inability to overlap in time and space may limit possible collaboration. However, it will be exciting to have someone around all the time whose job is to know what is going on and what everyone is doing and to create connections within and outside the organization.

I travel a lot for my work now. I've been to Boston, New York, Edmonton, Calgary, Chicago and Washington, D.C., in recent months, but it's great to have a home base and somewhere to keep my collection of frog mugs from my lab days. Being in an environment where people are doing things that are very different from my academic experience, where they all are looking to effect or advocate for change or educate the wider public, is really refreshing and helps me in some aspects of my work around advocacy. I'm looking forward to holding more workshops and satellite events around conferences that are in town to connect with the local scientific communities. Manylabs has been helpful in shaping my thoughts on the different ways science and society could interact and broadens my vision of a more encompassing scientific enterprise.

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Just like Boyd

Gary Weisman is inspired by his 100-year-old mentor, Boyd O'Dell

By Stephen Schmidt

In 1985, Gary Weisman had several assistant professorship offers in molecular biology after finishing his postdoctoral fellowship at Cornell University. He had the enviable problem of having trouble deciding which offer to choose.

"They said 'Don't dismiss Missouri,'" says Weisman, recalling the advice from his Cornell colleagues. "They knew Boyd O'Dell personally and how big he was in the field."

As the head of the department's hiring committee for a new cell-culture laboratory at the time, O'Dell, a professor of biochemistry at the University of Missouri, was fascinated by Weisman's work with cell-culture models, which would allow the institution's researchers to speed up the work that initially was being run with animal-based models.

Weisman ended up heading west to help transition the work of faculty in food science and nutrition at the university into the realm of cell biology.

PHOTO COURTESY OF JUSTIN KELLY

O'Dell raises a glass of wine at an event honoring his 100th birthday.

A pioneer in nutrition research

O'Dell, who turned 100 in October, has spent most of his career, which dates back 80-odd years to his time as an undergraduate student beginning in 1937, at MU. He worked primarily on animal-based nutrition research related to trace mineral deficiencies, namely of zinc and copper.

He would go on to become one of the first scientists to discover the important role that folic acid and vitamin B12 play in the development of human and animal embryos, leading to the now-well-recognized relationship between deficiencies in folate and B12 and birth defects in humans. O'Dell, with the help of his research team, also discovered in the late 1950s how phytic acid interferes with the absorption and utilization of zinc.

"That observation has caught worldwide attention, and they're still researching it in humans," says O'Dell.

Weisman, who became a professor of biochemistry at MU in 1998, was accompanied to MU by his top lab assistant at Cornell, Kevin Lustig, who now is the CEO of Scientist.com.

Lustig, along with then-graduate student Laurie Erb (now an associate professor of biochemistry at MU), cloned the first human gene for a P2 nucleotide receptor. For years, others in the biochemistry field had doubts about Weisman's research, but not O'Dell. "No one thought the receptor was real," says Weisman. "Kevin and Laurie kept me on the straight and narrow, and Boyd was here all of the time mentoring me, and I worked through the problem and basically convinced everybody that it was real."

Weisman, who lives about three blocks from O'Dell, also has adopted one of O'Dell's habits. For the first 10 years of his time in Columbia, while driving to work every day, Weisman would always see O'Dell walking to his lab. Inspired, Weisman started walking to campus in the 1990s.

"I just saw how fit he was. He was much older than me, but still he was working pretty hard and walking all of the time," Weisman says of O'Dell, who still makes his way to his laboratory on foot when the weather is good. "So I changed my lifestyle and took nutrition more seriously and started walking.

"To this day, I have not used my car at all. I walk everywhere around town. Basically, I'm always on my feet following in Boyd's footsteps. I don't mean that in a general way. I mean that absolutely literally."

PHOTO COURTESY OF STEPHEN SCHMIDT

Boyd O'Dell (left) and Gary Weisman

Forming a partnership

Weisman's lab is in the same building where O'Dell does his research. In 2014, O'Dell received word that Weisman had a fluorometer in his lab that would be perfect for his work on how zinc deficiency harms cell function by blocking the signal for calcium uptake. O'Dell was put in contact with Weisman's head technician, Jean Camden.

"We've been working together ever since," says Camden, who, like O'Dell, is now semiretired. In the fall of 2014, the two began the measurement of calcium uptake with blood platelets before it was determined that the platelets had too short of a shelf life. The following spring, Camden suggested they work with human T lymphocyte cells, called Jurkat cells, which can be produced easily millions at a time.

O'Dell has been using the Jurkat cells to measure zinc released by a mild oxidizing agent.

He adds the zinc back and releases it again. Based on the results obtained, Camden has used similar conditions to measure calcium uptake with the fluorometer. O'Dell and Camden hope to publish their results soon.

"His hearing is the only problem, so you talk loud," Weisman says of O'Dell. "But he's still thinking just as fast as we are — faster probably."

'Don't worry about your age'

Weisman, at age 65, has no intention of slowing down and becoming inactive, although at the moment he cannot wrap his mind around approaching 100.

"Don't worry about your age. Worry about how you feel today. Think about what you're going to do tomorrow, and I think you'll stay in the game longer than you think," Weisman says of his mantra. "Life goes fast, but I just don't see myself as a 65-year-old. I see myself as a teenager. And I think Boyd must be that way. He must not see himself as 100-years-old."

When people ask him about retirement advice, O'Dell provides two words: "Keep working."

"Try to pick something that you're passionate about and do it," O'Dell says. "If you like to research, as I do, that's OK. If you think that you want to spend your time making fishing lures, that's OK too. But you have to be passionate enough to really want to get up and go."

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If I could turn back time

By Eleftherios P. Diamandis

The rock icon Cher sang her hit of the 1980s "If I Could Turn Back Time." However, we all know that nobody can do this, and the Nobel laureate Bob Dylan confirmed it with his song "The Times They Are A-Changin."

At the 2016 Annual Conference of the American Association for Clinical Chemistry, I attended a session honoring three very successful clinical chemists. I asked each of them of what alternative career they would pursue if given a second chance. One kindly declined to answer. The second emphatically declared that he was completely satisfied with his current career and would follow the exact same path if he were to start over. The third one, who enjoys athletics, revealed that he would have pursued professional golf.

I believe that I have had a successful career in science and academia. I published often, made some discoveries, mentored more than 60 graduate students and collaborated with an army of other scientists during the course of my career. The beauty of our profession is that we have the freedom to investigate unanswered questions of our choice and be at the forefront of curiosity-driven research. At the same time, we have the privilege of working with brilliant young people whom we coach to develop their own paths to success. It is a very rewarding and well-paid profession. With all this in mind, I have no complaints.

PHOTOS COURTESY OF ELEFTHERIOS DIAMANDIS

The poster for Diamandis' laboratory band

However, we are all born with other passions that we do not necessarily explore when building a professional career. Over the years, I have been devoting time and energy in areas outside my profession. I suspect that these natural and spontaneous tendencies could have been used to build an alternate career. From very early on, I showed a keen interest in many sports, including soccer, boxing, basketball, athletics and so on, but it wasn't until my medical-school years that I fell in love with tennis. I was introduced to the sport in the early 1980s when I traveled for the first time to the U.S. and bought a cheap wooden racket from K-Mart before returning to Greece. It so happened that near my apartment in Athens there were two university tennis courts where I could play tennis for free. As the tennis courts were in high demand, we usually had to wait two or three hours to play for 30 minutes, as court booking was not a thing in Greece during that time. The balls we were using were beaten to death and barely bounced. If the strings of my racket broke, I would mend them myself, since I had no money to replace them. I would try to hit tennis balls anywhere, including walls, inside my apartment or even in the lab where I worked! I followed all the international tournaments and scores in newspapers (no internet then) and fell in love with the champions of the time, such as Bjorn Borg, John McEnroe and Jimmy Connors. When my children were born, I so desperately wanted them to become Wimbledon champions that I even wrote a fiction piece on this subject (1). To my disappointment, my children never really showed any interest in playing professional tennis (they play for recreation) and I was never really good at the sport either. Yet each of the two properties I own includes a tennis court, both built before the houses were erected! (Important things first.) Aside from my backhand weakness, I know the game very well.

But even before I came across tennis, as a 15-year-old from Cyprus (a former British colony), I began discov-

Diamandis with Canadian tennis player Eugenie Bouchard

ering in 1967 the Beatles, the Rolling Stones and the other rock groups by listening to English radio stations. I religiously followed "Top 20," a radio show based in England, and meticulously took notes on the ranking of the songs and the bands. Despite having no musical background in my family, I was obsessed with listening to music with my transistor radio on every occasion and became proficient at knowing every single song of that era. I was the human Shazam of my time (Shazam is the mobile app that identifies music). By 1970, my heroes included Neil Diamond, Neil Young, Led Zeppelin, AC/DC and The Who, among many others. When I was serving in the Cyprus Army as a soldier, I was punished and put in prison many times for breaking the rules and listening to music while on guard service.

My passion for music grew immensely over the years. My electronic library now has more than 300,000 songs, split equally between Greek and English. Everywhere I go, I carry my iPod as well as my iPhone, which are loaded with music. When I find five or more minutes of spare time, I listen to music. During my wife's shopping expeditions, I pass the time with music, secretly wishing she would do more shopping so I could extend my enjoyment! While I can spend 70 minutes daily on a treadmill exercising with music, I am not able to do a single minute of exercise without music. In short, music is an absolute necessity in my life. Unfortunately, I do not seem to have any talent in creating new music. My talent is only in listening.

I still am highly fascinated with the discovery process and the mentoring of young individuals, such that I could not imagine that I would leave this activity if presented a second chance. Consequently, I would prefer a composite of professions: From Monday to Friday I would operate my research laboratory, aiming toward discovering and publishing new knowledge and mentoring young students. On Saturday, I would work for a television station as a commentator for sporting events, particularly tennis. On Sunday, I would host my own radio show, during which I would play the music I like and tell the stories behind the hits!

Realistic? Probably not. Crazy? Definitely yes. From here, I suppose my only hope for a second profession (and proclaim, like Britney Spears, "Oops! … I Did It Again") is to find the "Stairway to Heaven."

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Make or break

By Vivian Tang

With lots of data in hand,
I yearn for them to make sense.
Count on no one to understand,
Publish or perish — that's common sense.

One postdoc appointment after another,
Craving for clarity in my academic prospect
And the bandwidth to go further.
If only I can turn back the clock, in retrospect.

I would perhaps take the industrial route.
Far less pressure to publish,
My hard work would still bear fruit,
Even a career I'd relish.

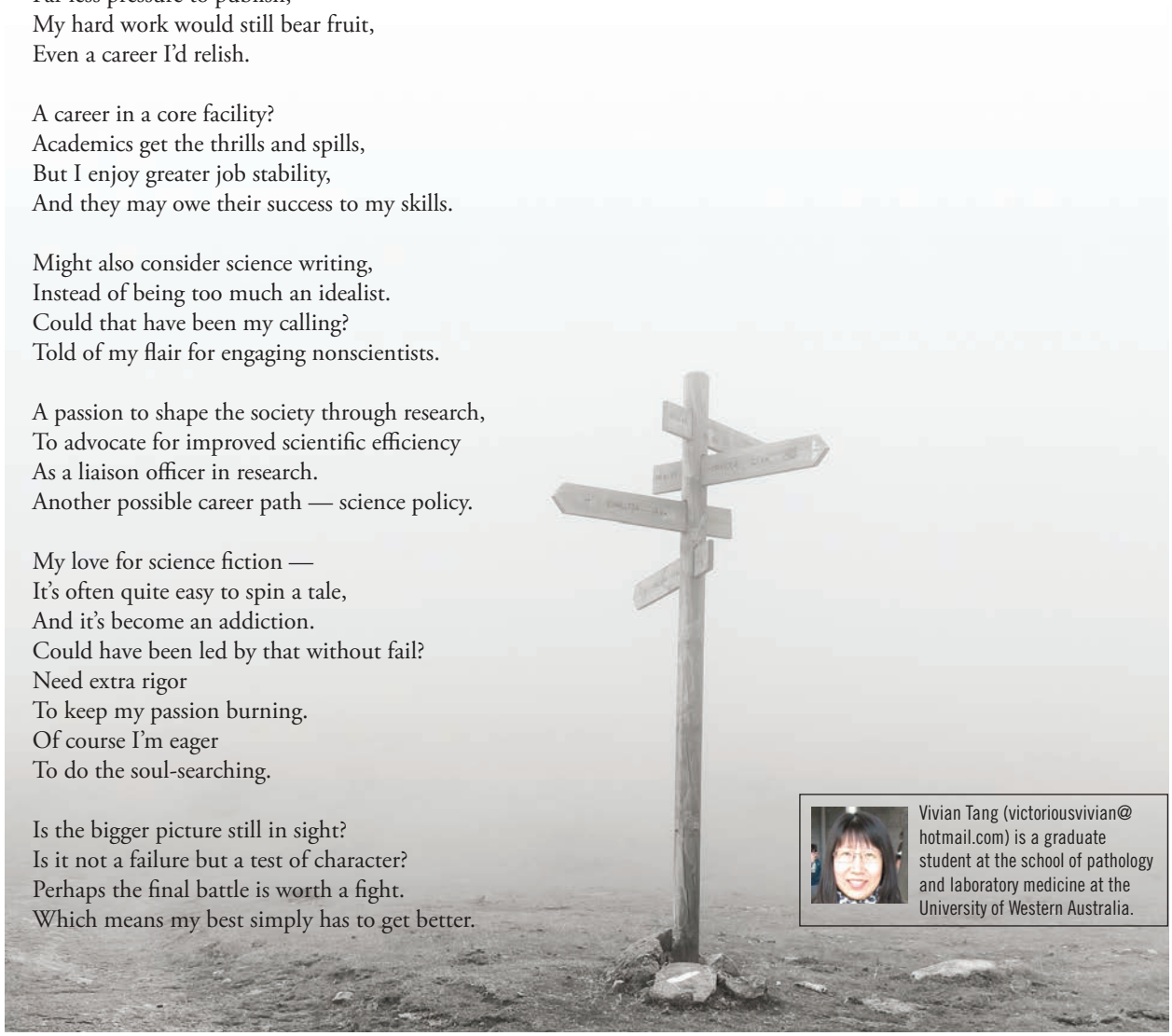
A career in a core facility?
Academics get the thrills and spills,
But I enjoy greater job stability,
And they may owe their success to my skills.

Might also consider science writing,
Instead of being too much an idealist.
Could that have been my calling?
Told of my flair for engaging nonscientists.

A passion to shape the society through research,
To advocate for improved scientific efficiency
As a liaison officer in research.
Another possible career path — science policy.

My love for science fiction —
It's often quite easy to spin a tale,
And it's become an addiction.
Could have been led by that without fail?
Need extra rigor
To keep my passion burning.
Of course I'm eager
To do the soul-searching.

Is the bigger picture still in sight?
Is it not a failure but a test of character?
Perhaps the final battle is worth a fight.
Which means my best simply has to get better.



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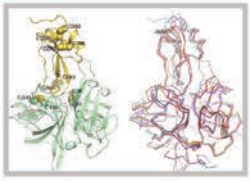

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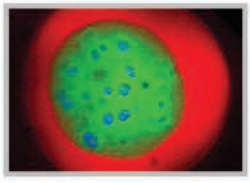

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