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ASBMBTODAY

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

THE TAXI DRIVER'S BOOK

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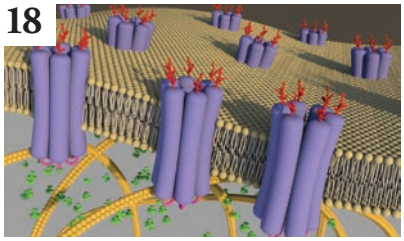

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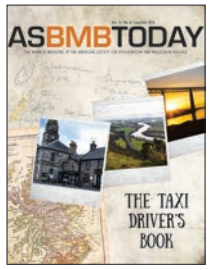
How a Scottish cab driver preserved a history of science in signatures.

Images courtesy of Pho-Tay Photography



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

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EDITOR'S NOTE

The taxi driver tip

By Lauren Dockett

Davie Douglas, the 69-year-old Scottish taxi driver at the center of this issue's cover story, may seem an unconventional subject choice for a biochemistry and molecular biology magazine. But Douglas has an exceptional relationship to the field. Over the past decade he has become an unwitting science historian by keeping a guestbook in his cab and preserving encounters with a cadre of notable life scientists.

We at ASBMB Today are happy to be the first to do a story on Douglas and to be featuring images in the piece from his photographer son David — photos we find so stunning we've decided to use them online as part of the magazine's very first immersive feature experience. Be sure to visit asbmb.org/asbmbtoday to check out this alternative version of the story.

PFEFFER

We learned about Douglas through Suzanne Pfeffer, former American Society for Biochemistry and Molecular Biology president and professor and chair of the biochemistry department at Stanford University. Pfeffer met Douglas on a work trip to Scotland and had an inkling that his story might be right for our readers. We were immediately taken with Douglas — and think you will be too — but know that we likely would not have found him on our own. (Although we consider ourselves a pretty in-the-know crew, exploratory trips to the castle-strewn hills and glens of foreign lands in search of story clues are a little beyond our means.)

We are grateful to Pfeffer for thinking Douglas would make a good profile for ASBMB Today and then actually getting in touch with us to propose the story. We encourage you to follow her lead and reach out whenever you encounter an interesting character of science or happen upon a captivating development that you think should be in the magazine. We'll look for your suggestions at asbmbtoday@asbmb.org. Who knows, you too could land a story on the cover!

Lauren Dockett (ldockett@asbmb.org) is ASBMB Today's managing editor.

PHO-TAY PHOTOGRAPHY

Our cover story about Scottish cabbie Davie Douglas was born of a tip from Stanford's Suzanne Pfeffer.

Checking out

By Steven McKnight

My tenure as president of the American Society for Biochemistry and Molecular Biology is coming to a close, and this is the last of the essays I will contribute to ASBMB Today. I've stumbled and bumbled my way through the past two years, but it has been fun. Reflecting on what has happened, I take some measure of pride in three things.

First, we have appointed Lila Gierasch as the new editor-in-chief of the Journal of Biological Chemistry. The JBC is our flagship journal. It is of utmost importance that we do anything and everything possible to ensure that the JBC sustains its reputation as the premier journal for publication of discoveries in the broad field of biochemistry.

I can think of no person better qualified to steer the journal than Lila — she has verve, style, energy and moxie. Lila is a biochemist/biophysicist of international fame. Lila also has been a successful leader of her department at the University of Massachusetts Amherst, so this will not be her first rodeo as a scientific leader. Finally, Lila is already simpatico with the fantastic Bethesda staff of the JBC headed by Nancy Rodnan.

Second, as a society, we have elected Natalie Ahn as the new president. My confidence in Natalie mirrors my confidence in Lila. Natalie's scientific accomplishments are also of international acclaim. Like Lila, Natalie is brimming with positive verve and energy. Organizations such as our ASBMB never follow a level trajectory: They are either getting better and better or getting worse and worse. I have all the confidence in the world that Natalie will lead the ASBMB north with respect to health and vitality.

Third, as an organization, I think the ASBMB is getting serious attention. Roughly a year ago, National Institutes of Health Director Francis S. Collins asked me if he could deliver a plenary talk at our annual meeting — we did not ask Francis to do this; he asked us to include him. Collins and other leaders recognize the value of the bedrock of the ASBMB — basic science.

I've stumbled and bumbled my way through the past two years, but it has been fun. Reflecting on what has happened, I take some measure of pride in three things.

This attention comes from many of our activities, including the efforts of our public affairs committee to communicate what we do as scientists to our elected representatives; the success of our minority affairs committee that has helped so many underrepresented minorities to learn how to write, submit and win research grants from federal agencies; and the many other vibrant components of our society.

The attention also comes, at least in a small part, from the essays I have written over the past two years in ASBMB Today. Yes, I ruffled many feathers with the misguided use of "riffraff." I happily accept the bruising I so rightly deserved for the essay titled "The curse of committees and clubs." I may be wrong on this but offer the speculation that the consternation associated with that essay helped elevate the amplitude of our collective voice.

For better or worse, I chose to use the bully pulpit of the ASBMB presidency to mix it up. My venue has been ASBMB Today, and I'm delighted that, by all measures, the footprint of "AToday" has expanded exponentially under the direction of Angela Hopp and her hardworking team. As I depart, I leave with some measure of confidence that the biomedical research community is paying attention to us.

Finally, I want the members of the ASBMB to know that my interactions with the headquarters staff have been fantastic. Barbara Gordon, Steve Miller, Angela Hopp, Nancy Rodnan, Ben Corb and all of the crew represent the glue that holds the ASBMB together. In this closing essay, I want ASBMB members to know that the staff running our organization is as good as it gets. Whereas elected members such as me come and go through a rotating turnstile, the staff is largely constant. The ASBMB is their livelihood, their devotion and their pride.

It has been a lively two years — thanks for putting up with me! I close with the Norwegian words "har det bra." As explained to me by my neighbor up in Montana, these words do not mean "goodbye" but instead "have it fine."

Steven McKnight (steven.mcknight@utsouthwestern.edu) is president of the American Society for Biochemistry and Molecular Biology and chairman of the biochemistry department at the University of Texas-Southwestern Medical Center at Dallas.

Advocating better, together

By Benjamin Corb

American Society for Biochemistry and Molecular Biology members spend their lives dedicated to understanding the underlying mechanisms of diseases that affect the lives of millions of Americans. Advocating on behalf of their work and the work of all biomedical researchers is an honor for me. In recent months, our message about the need for increased federal support for the National Institutes of Health has gained traction. Lawmakers are better at understanding the role the NIH plays in supporting the nation's biomedical research enterprise, and members from both political parties are publicly expressing support for increasing federal investments. Last year's $2 billion increase in funding for the NIH serves as an example of this support, and overwhelming passage of the 21st Century Cures Act last summer provides us with more proof that there is political will to support the NIH.

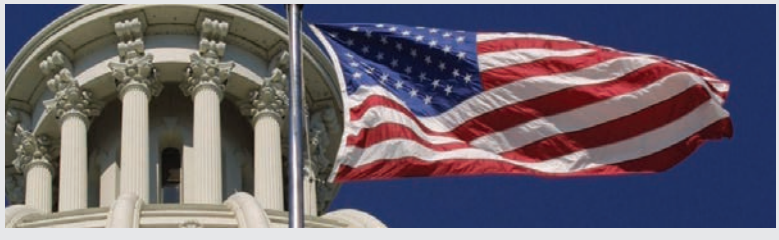
Manifesting the words of politicians into deeds — and dollars — remains the challenge of all advocates. We advocates often work together, fine-tuning our message to build momentum on Capitol Hill to support our members. As a member of the board of directors of the Coalition for Health Funding (www.publichealthfunding.org) I took part in a series of meetings with lawmakers in May that sought to break down some of the traditional silos that health and science advocates find themselves in. On that day, I was joined by advocates for disease groups, public health professionals and social scientists. We called on members of Congress to recognize how they must invest in the full continuum of health and science agencies — from the NIH to the Food and Drug Administration to the Centers for Disease Control and Prevention — if we are going to make measurable improvements to public health.

Some lawmakers already have connected these dots and are helping to spread our message to their colleagues. U.S. Reps. Rob Wittman, R-Va., and Gene Green, D-Texas, formed the Public Health Caucus in the House of Representatives last year. Caucuses are, for lack of a better descriptor, clubs in which like-minded members of Congress come together and discuss the variety of issues that affect a specific topic. The Public Health Caucus will raise awareness of public health issues and, perhaps more importantly, provide educational opportunities for lawmakers to learn more about what public health means.

When announcing the formation of the Public Health Caucus, co-chairs Wittman and Green succinctly outlined why a joint effort like this is so important. Wittman told reporters that "the field of public health is dedicated to awareness and prevention, and this caucus will be a fantastic tool to raise awareness among members and congressional staff about how public health issues can impact their districts. We must do more to reduce the cost of health care in America, and, by focusing our legislative efforts on prevention and public health, Congress can help to rein in costs in the future." Green added, "Investing in public health has led to increased life expectancies, reductions of infectious and communicable diseases, swift response to emergencies and improved health outcomes for our communities."

Since its inception, the caucus has grown to 19 members, with groups like the Coalition for Health Funding working to make more lawmakers aware of this caucus. Explaining the critical role basic research plays in the full public health continuum is a logical and needed step in continuing to bolster support for the funding scientists so desperately need.

Benjamin Corb (bcorb@asbmb.org) is director of public affairs at ASBMB.



Interested in science policy?

Follow our blog for news, analysis and commentary on policy issues affecting scientists, research funding and society. Visit policy.asbmb.org.

Scenes from Hill Day 2016

PHOTOS COURTESY OF EMILY HUFF

A Hill staffer and U.S. Rep. Steve Cohen, D-Tenn., review ASBMB materials on supporting biomedical research and listen to PAAC member Matt Gentry and postdoc Peter Mercredi discuss how federal dollars are used in their labs.

ASBMB members walked the halls of Capitol Hill for hours, meeting with 96 members of Congress to build support for the biomedical research community.

Yale's Jenna Belestrini and Garet Thompson from the University of New Hampshire join PAAC member Preston Hensley outside a Connecticut ongressman's office.

Refueling in the Longworth Office Building cafeteria, PAAC members and their students find time in their busy schedules to grab lunch.

Outgoing PAAC member Mark Lively and students from North Carolina State University and the University of South Carolina pose before a meerting.

Seven members elected to the American Academy of Arts and Sciences

The American Academy of Arts and Sciences has announced the addition of 213 new fellows and foreign honorary members. Seven of the elected fellows are American Society for Biochemistry and Molecular Biology members, including the incoming editor-in-chief of the Journal of Biological Chemistry, Lila Gierasch.

Fellows of the academy advance its mission through collaborative projects and teaching opportunities across multiple disciplines. They participate in lectures, discussions and meetings nationwide and provide scholarly advice that helps shape major decisions within both the national government and the private sector.

The 2016 academy class includes the following ASBMB members:

RICHARD H. EBRIGHT
Rutgers University

LILA M. GIERASCH
University of Massachusetts

DAVID P. HAJJAR
Weill Cornell Medical College

DONALD HILVERT
ETH Zurich

MARK W. HOCHSTRASSER
Yale University

LAWRENCE A. LOEB
University of Washington

CARL F. NATHAN
Weill Cornell Medical College

Four members elected to the Royal Society

The Royal Society announced the election of 50 new fellows and 10 new foreign members to their organization. The Royal Society is the oldest scientific society in continuous existence. Fellows and foreign members help promote the society's mission to encourage the use of science for the benefit of humanity.

The new foreign members and fellows include the following ASBMB members:

JENNIFER DOUDNA
University of California, Berkeley

HARRY GILBERT
University of Newcastle upon Tyne

MARK LEMMON
Yale University School of Medicine

LUKE O'NEILL
Trinity College Dublin

Eight members elected to the National Academy of Sciences

In early May, the National Academy of Sciences announced the election of 84 new members and 21 foreign associates.

Members are recognized for distinguished and continuing achievements in original research and lend their expertise to the academy in a number of ways, including participating in administrative or leadership roles for the academy and volunteering on study committees.

The academy class of 2016 includes the following ASBMB members:

JOHN C. BOOTHROYD
Stanford University School of Medicine

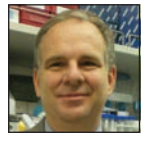
RAYMOND J. DESHAIES
California Institute of Technology

SUSAN MARQUSEE
University of California, Berkeley

KRISHNA K. NIYOGI
University of California, Berkeley

PATRICK J. STOVER
Cornell University

MICHAEL F. SUMMERS
Howard Hughes Medical Institute, University of Maryland, Baltimore County



STEPHEN G. YOUNG
University of California, Los Angeles

IAN A. WILSON
The Scripps Research Institute

Written by Bree Yanagisawa

Pagliarini wins Presidential Early Career Award


PAGLIARINI

President Barack Obama named Dave Pagliarini, director of metabolism at the Morgridge Institute for Research and associate professor of biochemistry at the University of Wisconsin, Madison, a recipient of a Presidential Early Career Award for Scientists and Engineers.

Announced in February, the PECASE awards are the highest honor bestowed by the government on young scientists and presented to researchers and engineers who have distinguished themselves through innovative research and leadership. Pagliarini's lab researches the biochemical causes of mitochondrial dysfunction and develops methods to define the functions of mitochondrial proteins. Pagliarini hopes that establishing the root causes of mitochondrial dysfunction will lead to more effective therapies for as many as 150 associated diseases.

The National Institutes of Diabetes and Digestive Kidney Disorders partially funds Pagliarini's program and nominated him for the award.

Marnett new dean at Vanderbilt med school


MARNETT

Lawrence J. Marnett, a professor of biochemistry, chemistry and pharmacology and Geddes Stahlman professor of cancer research at Vanderbilt University, has been appointed dean of basic sciences at Vanderbilt University School of Medicine. He assumed the role following the medical school's legal separation from the university at the end of April.

Marnett's responsibilities will include leading the school of medicine's basic science departments and their basic science centers, institutes and programs.

Marnett brings a wealth of leadership experience to this new position. A former associate director of basic research at Vanderbilt–Ingram Cancer Center and director of the Vanderbilt Institute of Chemical Biology, he was named associate vice chancellor of research and senior associate dean for biomedical sciences of Vanderbilt University School of Medicine in 2014.

Marnett also runs the A.B. Hancock Jr. Memorial Laboratory for Cancer Research, which seeks to develop novel and effective methods to treat and prevent cancer.

Rice receives InBev–Baillet Latour prize


RICE

Charles Rice, the Maurice R. and Corinne P. Greenberg professor in virology at The Rockefeller University and scientific and executive director of the Center for the Study of Hepatitis C, has won the 2016 InBev–Baillet Latour Health Prize for his research on the hepatitis C virus.

Hepatitis C virus, or HCV, is a major contributing factor to cirrhosis and liver failure. Rice heads the laboratory of virology and infectious disease at Rockefeller, researching how HCV infects liver cells and causes disease. Work at Rice's lab has led to a greater understanding of the life cycle of HCV. Rice's research is being applied to help develop new methods to treat the virus, such as 3-D culture and induced pluripotent stem cell culture, which can be used to grow HCV and other viruses efficiently.

Established in 1979 and awarded by the Baillet Latour Fund, this prize recognizes outstanding individual achievement in biomedical research. Queen Mathilde of Belgium presented the award, which comes with a more than $280,000 cash prize, to Rice at a ceremony at the Palais des Académies in Brussels.

Schatz and Tschudi honored by Yale


SCHATZ

Two Yale professors, David Schatz and Christian Tschudi, have been recognized by the university for their outstanding scientific achievements.

David Schatz has been named the Waldermar Von Zedtwitz professor of immunobiology and of molecular biophysics and biochemistry. A distinguished researcher, his work has increased scientists' understanding of the mechanisms that assemble and diversify antigen receptor genes, which encode antibodies and T cell receptors. Schatz is highly regarded for his discovery of the recombination activating genes RAG1 and RAG2 as well as his discoveries in the field of somatic hypermutation.


TSCHUDI

Tschudi has been named the John Rodman Paul professor of epidemiology. An expert on neglected tropical diseases, Tschudi's research focuses on the protozoan parasite Trypanosoma brucei, an agent that can lead to diseases in humans and animals in sub-Saharan Africa.

Tschudi also has received the Burroughs Welcome Fund New Investigator Award in Molecular Parasitology and the MacArthur Foundation's Research Project Award in Parasitology and Tropical Medicine.

Gustafson wins Mann award

Maggie Gustafson, a fifth-year doctoral student in biochemistry and

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molecular and cell biology at Cornell University, has won the Harry and Samuel Mann Outstanding Graduate Student Award. Created in 2011 to recognize graduate students who exhibit outstanding scientific research skills and leadership, the award honors Harry and Samuel Mann, who were instrumental in the early production of penicillin. It carries a $20,000 cash prize.

GUSTAFSON

In Chris Fromme's lab at Cornell, Gustafson works on the Golgi complex, the primary sorting organelle responsible for the distribution of proteins throughout the cell. Studying Gea1 and Gea2, proteins that turn on the molecular switch Arf1, Gufstason discovered that multiple components are involved in the activation of Arf1 by Gea.

Gustafson is also an active member of the Graduate Student School Outreach Program at Cornell, which pairs graduate students with teachers to help mentor young students. Gustafson has taught minicourses on cell biology to students at Ithaca's Cayuga Heights Elementary School for the past three years. She also works with the Cornell Institute for Biology Teachers, a program designed to help train science educators.

Written by Erik Chaulk

Zoon named interim director of new NIH office

ZOON

National Institutes of Health Director Francis S. Collins announced in April the creation of a new Office of Research Support and Compliance and named Kathryn Zoon as interim director. The new office will oversee compliance with research regulations and standards. It was created in response to a series of deficiencies within the NIH's Pharmaceutical Development Section that were identified last year by the Food and Drug Administration. The offenses include poor aseptic techniques, inadequate staff training and inappropriate ventilation in laboratory environments. In April 2015, two vials of albumin from the facility were found to be positive for fungal contamination.

Prior to this assignment, Zoon was the first woman to hold the position of scientific director for the Division of Intramural Research at the National Institute of Allergy and Infectious Diseases. An immunologist, Zoon is a member of the U.S. Institute of Medicine. She served as director of the FDA's Center for Biologics Evaluation and Research from 1992 to 2002.

Written by Bree Yanagisawa

ASBMB learned this year about the deaths of the following members:

George Acs, July 31, 2013
Peter Alaupovic, January 30, 2014
Francisco Alvarado, February 23, 2015
Richard N. Armstrong, June 22, 2015
Joel Avigan, September 6, 2014
Andrew Benson, January 16, 2015
Guido Camici, July 22, 2015
Mathew Gerakis, October 16, 2015
Alfred Gilman, December 23, 2015
John Glomset, August 28, 2015
Alan Hall, May 3, 2015
Donald Hanahan, February 4, 2016
Christian Harteneck, April, 12, 2016
Richard J. Havel, April 9, 2016
George Hess, September 9, 2015
Richard T. Jones, February 26, 2008
David B. Knaff, January 27, 2016
Robert F. Labbe, March 23, 2015
Irvin Liener, November 8, 2013
Vincent Manganiello, January 10, 2016
Donald Nierlich, June 17, 2011
David E. Ong, April 25, 2015
Charles R. Park, May 7, 2016
James T. Park, July 14, 2015
Alexander Rich, April 27, 2015
William Sacks, July 11, 2006
Melvin Santer, June 25, 2015
Richard Setlow, April 6, 2015
Marion B. Sewer, January 28, 2016
E. C. Slater, March 26, 2016
Robert Suhadolnik, January 26, 2016
Gina Sosinsky, September 2, 2015
Robert G. Spiro, May 16, 2015
Oscar Touster, February 27, 2015
Elisabetta Ullu, September 8, 2014
John Van Pilsum, November 21, 2014
George R. Waller, March 23, 2015
K. Lemone Yielding, May 3, 2014

Berg named editor-in-chief of Science

BERG

Jeremy Berg, former president of the ASBMB and a professor at the University of Pittsburgh, has been selected as the new editor-in-chief of Science. On July 1, Berg will become the 20th editor-in-chief of the publication since its founding in 1880.

"I am thrilled and humbled by the opportunity to work with the team at Science and AAAS," Berg said in a press release from the American Association for the Advancement of Science. "Effective communication of results as well as key aspects of the scientific process and culture has never been more important."

Berg brings a passion for science policy and advocacy to his new role, which also includes overseeing the Science family of journals. Before becoming ASBMB president, Berg received the Howard K. Schachman Public Service Award, and throughout his career he has been an advocate for diversity in the sciences. During his eight years as director of the National Institute of General Medical Sciences, Berg was responsible for several programs intended to diversify the scientific workforce.

Berg also has been a strong proponent for developing more sustainable models of biomedical research and continues to bring attention to this important topic by serving as a steering committee member for the new Rescuing Biomedical Research initiative.

Written by Bree Yanagisawa

Vincent C. Manganiello (1939 – 2016)

By Matthew Movsesian

Vincent C. Manganiello, a pioneer and leader in the field of cyclic nucleotide-mediated signaling, died in January. He was 76.

Vinnie, as he was known to family and friends, was born and raised in Jersey City, N.J. He graduated from two Jesuit schools, Regis High School in New York City and Saint Peter's College (now Saint Peter's University) in New Jersey, before attending Johns Hopkins University in Baltimore, where he obtained his Ph.D. in physiological chemistry in 1965 and his M.D. in 1967. A year later, after completing an internship in pediatrics at Johns Hopkins, he accepted a position as a postdoctoral fellow at the National Institutes of Health. He remained there for the rest of his life, eventually becoming chief of the Laboratory of Biochemical Physiology.

CAROLINE MANGANIELLO

Manganiello was considered the world's expert on the enzyme PDE3.

From the outset of his career at the NIH, Vinnie studied cyclic nucleotide-mediated signaling. His first papers in the area, co-authored with Ferid Murad and Martha Vaughan at the NIH, described roles for cGMP and cAMP in intracellular signaling in adipocytes. His research moved into the then-new area of cyclic nucleotide phosphodiesterases. In his first paper on the subject, he proposed that hormones could affect cAMP-mediated signaling by modulating the activity of phosphodiesterases. A year later, he showed that the inhibition of lipolysis by insulin in fat cells resulted from a decrease in intracellular cAMP content, which in turn resulted from an increase in the activity of a membrane-associated phosphodiesterase with a high affinity for cAMP. This enzyme, which was later named cyclic GMP-inhibited phosphodiesterase, or cGi-PDE, and finally PDE3, would eventually become the principal focus of his career.

Vinnie's contributions to his field were many. His laboratory cloned PDE3A and PDE3B, the only two genes identified in this family of phosphodiesterases to date. He characterized structural determinants of their intracellular distribution and showed that both PDE3A and PDE3B are regulated by reversible phosphorylation. He provided new insight into the mechanisms by which these enzymes are localized to specific intracellular domains by identifying multiprotein signaling complexes in cardiac myocytes and adipocytes to which PDE3A and PDE3B are recruited by phosphorylation in response to various extracellular signals. He generated PDE3A- and PDE3B-null mice and used these and other models to identify specific roles for each subfamily. Cumulatively, this work contributed enormously to our understanding of the role of PDE3A in regulating contractility in cardiac myocytes and the role of PDE3B in regulating insulin secretion in pancreatic beta cells and metabolism in adipocytes and hepatocytes.

Within the NIH, Vinnie was also known for his citizenship. He contributed 20 years of service on the Institutional Review Board, tasked with review, approval and oversight of human subjects research. According to his colleague Richard Cannon at the NIH, "Vinnie cared deeply about the clear explanation of risks and benefits to research subjects. He insisted that if he couldn't understand a protocol's risks, a research subject under considerable stress wouldn't be able to either. Even when discussions became

spirited, he never lost his calm and cheerful demeanor."

Health problems emerged in the last year and a half of his life. He suffered serious complications after elective surgery on an aortic aneurysm in 2014, but he recovered and published several major papers in 2015. In the summer of 2015 he was diagnosed with pancreatic cancer. He told very few people about his illness and kept working as long as he could. Colleagues were stunned at the news of his death. A recent collaborator at the University of Toronto, Peter Backx, told me, "I had no idea he had been sick. I had just talked with him a few weeks ago about new experiments. He seemed his usual self." On reflection, those who knew how much he loved research are not surprised that he would keep at it until the very end.

Vinnie was an unassuming man who never craved the spotlight. But his stature in his field is indisputable. When the Gordon Research Conference on Cyclic Nucleotide Phosphodiesterases was initiated in 1999, Vinnie was chosen as its first chairman. He served for many years on the editorial board of the Journal of Biological Chemistry. In 2012, upon his induction into the Johns Hopkins University Society of Scholars, it was noted that he was "internationally recognized for his studies of cyclic nucleotide phosphodiesterases, a multigene family that regulates many fundamental biological processes by controlling intracellular cAMP and cGMP concentrations." Jackie Corbin, emeritus at Vanderbilt University and a longtime colleague, said, "Vinnie was a wonderful pure scientist and was widely respected as such. He was considered the world's expert on PDE3, and everyone in this field came to him with questions, requests for materials and suggestions for collaborations. His presence and wisdom at international meetings were critical. He was cheerful and had an outstanding sense of humor. His contributions at a fundamental level are leading to improvements in human health."

Over his nearly 48-year career at the NIH, Vinnie mentored many people in the phosphodiesterase field and befriended many more in the scientific community. His wife, Caroline, received many letters from Vinnie's former fellows, who described him as "the best mentor one could ask for," "a father to young fellows," "warm and friendly," "generous with his time and wisdom," and "selfless." His character was perhaps best captured by a former fellow who wrote, "(H)e was an example of how scientists should be." All of these reminiscences are unified in their recognition of his personal decency. For those of us who were privileged to be his colleagues in the scientific community and, even more, his friends, the sorrow at his loss is profound and is tempered only somewhat by our gratitude for the role he was willing to have in our lives.

Matthew Movsesian (matthew.movsesian@hsc.utah.edu) is a professor of medicine at the University of Utah. He thanks Joel Moss and Eva Degerman for their contributions to this Retrospective.

Upcoming ASBMB events and deadlines

JULY

July 14 – 16: ASBMB Grant Writing Workshop, Washington, D.C.
July 20: ASBMB Webinar: Charting a course to career success
July 27 – 28: ASBMB communications and career workshops, University of Kansas City Medical Center, Kansas City, Kan.

AUG

Aug. 1: Abstract and registration deadline for the ASBMB Transcriptional Regulation by Chromatin and RNA Polymerase II symposium
Aug. 17: ASBMB Webinar: Building professional relationships: pragmatic advice for the human scientist
Aug. 19 – 21: MAC Career Workshop: Careers Beyond the Bench, Johns Hopkins University, Baltimore, Md.

OCT

Oct. 6 – 9: ASBMB Special Symposia: Transcriptional Regulation by Chromatin and RNA Polymerase II, Snowbird, Utah

ASBMB
American Society for Biochemistry and Molecular Biology

A new test for diagnosing Niemann–Pick disease

By Bree Yanagisawa

Niemann–Pick disease is a rare genetic disease with devastating effects. In one type of the disease, known as type C, defects in lysosomal storage within the cell lead to impaired neurological function. In infants, these symptoms can be especially difficult to recognize. They often include subtle changes in children's development, such as failure to meet cognitive milestones or poor balance control.

UNITED STATES AIR FORCE

A new diagnostic test for Niemann–Pick disease can use previously collected dried blood spots from heel sticks.

Until recently, the first-line diagnostic test for NPC disease involved a skin biopsy and filipin staining, which is invasive, cumbersome and expensive. Patients with NPC often go up to five years without a diagnosis, drastically limiting the possibility of early interventions.

In a paper recently published in the journal Science Translational Medicine, Daniel Ory of Washington University School of Medicine in St. Louis and colleagues lay the groundwork for a promising new diagnostic test for NPC. Importantly, the new noninvasive assay produces results within a day instead of months.

The team used mass spectrometry to analyze dried blood spots collected at various times after birth from patients known to have NPC. They found three bile acid biomarkers that could distinguish NPC patients from people without the disease.

The scientists then determined the structures of the bile acids. Ory and colleagues identified one bile acid as a trihydroxycholanic acid and another as its glycine conjugate.

Since the second bile acid helped the team distinguish NPC patients from non-NPC patients more consistently, the researchers decided to use it to develop a new diagnostic test.

Ory says the assay already is being used at Washington University in St. Louis, Mo., as a diagnostic test. He expects other centers to follow suit.

For its use in newborn screening, Ory says researchers will need to put the assay to the test in the undiagnosed newborn population to ensure its usefulness for that age group. Ory believes the testing process will take several years.

Although the U.S. Food and Drug Administration hasn't yet approved treatments for NPC, a promising drug called cyclodextrin is moving rapidly through clinical trials. To be effective, treatment interventions will need to take place early in the disease process, which is something the new test could help accomplish. "We're really trying to make an impact in this NPC community by being able to develop the therapies and being able to diagnose early," says Ory. The approach "we've taken over the last 10 years, I feel like, it's getting close to bearing fruit."

Bree Yanagisawa (breannwoelfel@gmail.com) was an intern at ASBMB Today when she wrote this story. She is currently a Ph.D. candidate in pathobiology at the Johns Hopkins School of Medicine. Follow her on Twitter at twitter.com/BreeTalksSci.

Raising postdoc overtime pay

By Benjamin Corb

The Department of Labor has issued new rules about overtime pay. Effective Dec. 1, the following changes will apply:

The overtime pay eligibility threshold will rise to $913 per week from $455 per week, or $47,476 per year.

The eligibility threshold will update automatically every three years, based on wage growth over time.

Overtime protections for salaried workers who are entitled to overtime will be strengthened.

In addition to these changes, the Labor Department has addressed an issue of immense importance to our membership: Postdoctoral fellows will be eligible for overtime pay.

These rule changes are not unexpected. In the fall, the American Society for Biochemistry and Molecular Biology submitted comments to the Labor Department on this topic. We recommended that postdocs not be excluded from the changes but also called on the Labor Department to delay implementation beyond the Oct.1 start date so that researchers, who already wrestle with tight budgets, would have time to adjust to the requirements. The ASBMB asked for a three-year delay in implementation of the rules.

In response to the rule change, National Institutes of Health Director Francis S. Collins and Labor Department Secretary Thomas Perez published an op-ed acknowledging that the time has come for the scientific community to increase postdoctoral pay. They wrote, "Our nation should embrace the fact that increasing the salary threshold for postdocs represents an opportunity to encourage more of our brightest young minds to consider choosing careers in science. Biomedical science has never been more exciting or promising than now, and we need to do all we can to support the next generation of scientists."

Collins and Perez cited the ASBMB's 2015 paper on sustaining the biomedical research enterprise in their piece and acknowledged the role that the NIH must play in ensuring a smooth transition to adherence to the new rules. Collins indicated that the NIH will increase the levels of postdoctoral National Research Service Awards, the mechanism by which many postdocs are funded.

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Ticks taste you

By Bree Yanagisawa

Ticks are notorious for spreading bacteria that can cause the potentially debilitating Lyme disease. But scientists have lacked the necessary tools to advance a proper study of the bugs and aid in the development of Lyme disease interventions. According to Cate Hill, professor of entomology at Purdue University, entomologists have been hampered by "a desperate need of molecular tools and resources to help us understand the biology of ticks."

Recently, Hill was part of a large team of scientists that worked on sequencing the entire tick genome. The team chose Ixodes scapularis, the deer tick, because of its prominent role in human infections. Results from the sequencing project, which represents the first fully sequenced tick genome, were presented in a recent Nature Communications paper.

From their genetic analyses, Hill's team identified processes that appear unique to ticks and could serve as points for targetable interventions. Among these were the function of receptors used by ticks to locate a host.

U.S. AGRICUTLTURAL RESEARCH SERVICE

Scientists have sequenced the genome of the deer tick, Ixodes scapularis, which is responsible for transmitting Lyme disease in humans.

In the 10 years it took to complete the genome sequencing, scientists uncovered many genes that may help them better understand tick biology. From their genetic analyses, Hill's team identified processes that appear unique to ticks and could serve as points for targetable interventions. Among these were the function of receptors used by ticks to locate a host.

"Their biology is very different from other blood-feeding arthropods, like mosquitos, and the way that they find a host is very different," says Hill. "We think that ticks may rely on gustatory, or taste, receptors. They use their taste receptors to smell rather than the more highly evolved smell receptors that mosquitos use."

Other important genes identified from the analysis correspond to proteins potentially specific to ticks, including a diverse range of salivary proteins and proteins involved in hemoglobin digestion. Scientists will need to validate these proteins in the ticks to understand better their feasibility as targets of intervention strategies.

Bree Yanagisawa (breannwoelfel@gmail.com) was an intern at ASBMB Today when she wrote this story. She is currently a Ph.D. candidate in pathobiology at the Johns Hopkins School of Medicine. Follow her on Twitter at twitter.com/BreeTalksSci.

Solving the riddle of the role of sphingolipids in cell signaling

By Alexandra Taylor

When Johann L. W. Thudichum, a German-born physician, discovered a puzzling class of lipids in 1881, he dubbed them "sphingolipids," a reference to the mysterious Sphinx of Greek legend who taunted travelers with her riddles. Although the structures of sphingolipids were worked out in the 1940s, their important roles in cellular metabolism were not apparent until the mid-1980s. "From the very beginning, these lipids were dubbed as enigmatic," says Yusuf Hannun at Stony Brook University Cancer Center. "No one touched them for decades."

BELL

Robert Bell, a lipid researcher, was at Duke University in the 1980s. At that time, "lipids were thought to be just components of membranes," says Bell. "Medical students hated them. Graduate students ignored them." Which is why, when the common lipid molecule diacylglycerol was found to activate the newly discovered enzyme known as protein kinase C, many in the field were astounded.

Protein kinase C phosphorylates many proteins in the cell; these proteins trigger a plethora of cellular responses, such as transcription, cell growth and immune responses. "I couldn't believe this was the case," says Bell. "Diacylglycerol was an ordinary, everyday intermediate in lipid metabolism. How could it have this special second-messenger function ascribed to it?" Bell's lab set out to refute the hypothesis that diacylglycerol regulated protein kinase C. When this proved unsuccessful, they wondered if other lipids might have an effect on the kinase.

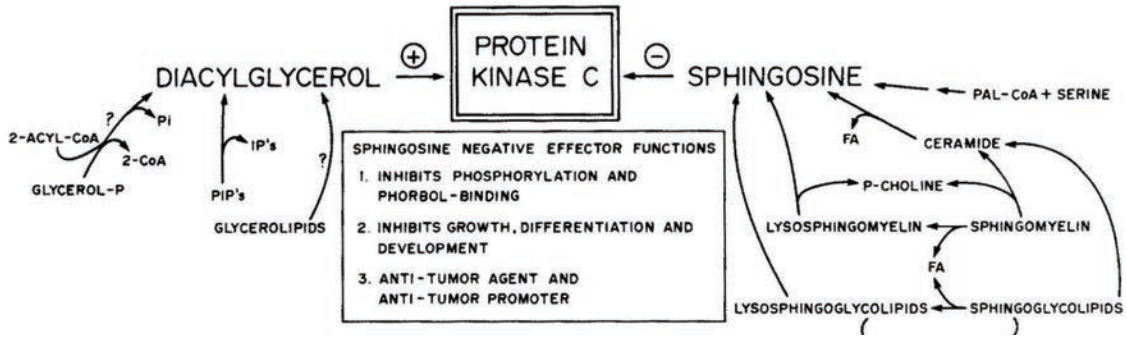
HANNUN

Hannun was a postdoctoral fellow in Bell's lab. Working alongside postdoctoral fellow Carson Loomis, he tested an array of common lipids, including sphingosine. Sphingosine can be either a precursor or a breakdown product of complex sphingolipids. "No one knew why Bob's lab had sphingosine, what it was doing on the shelf or what sphingosine even was," recalls Hannun. When the tests showed that sphingosine had the opposite effect and inhibited protein kinase C, the researchers were dumbfounded. "My mind started racing. What is this sphingolipid? What about other sphingolipids?" recalls Hannun. "I knew very little about sphingolipids. I would say most people who called themselves lipid biochemists knew very little about them."

Ultimately, Bell and his collaborators submitted three papers as a set to the **Journal of Biological Chemistry**. The first paper described the primary observation that sphingosine inhibits protein kinase C and provided examples both in the test tube and in human platelets (1). In the other two papers, other authors who collaborated with Bell's group expanded on the physiological relevance of the primary observation (2,3). Alfred Merrill, a former postdoctoral fellow of Bell's, who was then an assistant professor at Emory University, had been studying sphingolipids for several years. He describes the collaboration as "a coalescing of experiences that got everybody very excited."

MERRILL

The second and third papers reported on the roles of sphingosine and other sphingoid bases. Sphingoid bases are the building blocks of the sphingolipid backbone. The two papers explored the roles of sphingoid bases in oxidative burst, which is the release of chemicals from immune



Diacylglycerol and sphingosine are products of complex lipid metabolism that have competing effects on protein kinase C. Diacylglycerol activates the kinase; sphingosine inhibits it. This schematic originally appeared in J. Biol. Chem **261**: 12604-12609 (1986).

cells, and differentiation of bone marrow cells. The three papers together presented a more cohesive body of evidence for the important roles of sphingolipids in signaling than a one-off study would have provided. "At Duke, I worked very hard to make lipids interesting to medical students," says Bell. "Suddenly, we started to understand that sphingolipids could play roles in cell signaling. It was very exciting."

Still, their findings were controversial. Bell recalls presenting his research at a Gordon conference: "As soon as I sat down, everybody working in the sphingolipid field jumped up and started arguing," he recalls. "The pushback was instant."

Many lipid researchers had difficulty accepting that a lipid breakdown product could serve a regulatory function. "They all went back to their labs and started studying it," says Bell. "Some of them are still studying it today."

Ever enigmatic, sphingolipids proved difficult to handle for many researchers, some of whom initially had trouble verifying some of the cellular results. Once the community came up to speed, however, sphingolipid signaling proved to be fertile ground for scientific discovery. "Now there are thousands of papers on bioactive sphingolipids," says Hannun. "They do so many things — regulate blood vessel formation, cell death, cell migration, immune responses."

"You usually get the feeling that the best work you do is always resisted," says Bell, who has been retired since 2010. "People aren't going to accept it easily. I think this has now stood the test of time, which is great to know."

This article originally was published in the Journal of Biological Chemistry.

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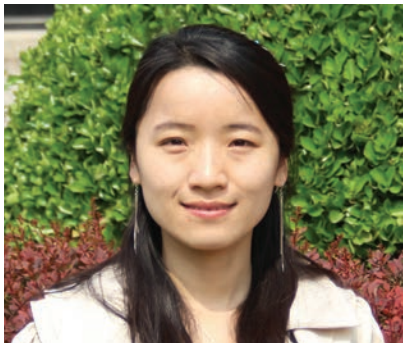
Li wins Tabor award for characterizing reader proteins

By Dawn Hayward

Yuanyuan Li, an advanced innovation fellow at Tsinghua University in Beijing, has received the **Journal of Biological Chemistry**/Herbert Tabor Young Investigator Award for characterizing novel histone reader proteins and their role in transcription.

Li combines X-ray crystallography with biochemical and biophysical techniques to explore reader proteins. These proteins recognize post-translational modifications on histones and help dictate whether genes are transcribed or kept silent. Once the reader proteins identify their target, they can recruit other proteins to allow or prevent transcription. Li has discovered several novel reader proteins and also a family of reader proteins that recognize crotonyl-lysine residues. Lysine residues on histone tails can be modified by many chemical groups, and a crotonyl-group is one such addition. The crotonyl-lysine is recognized by a so-called YEATS domain, which is common to all YEATS family reader proteins. Li says that they also can recognize acetyl groups on lysine residues and that in the future, "by combining the structure based rational design and virtual screening, my … research aims to do inhibitor design and development on these reader proteins."



Yuanyuan Li received the Tabor award from JBC Associate Editor Joel Gottesfeld at the 2016 Keystone Symposium on Chromatin and Epigenetics.

Li received a bachelor's degree in biotechnology from Shandong University in Jinan, China, and a Ph.D. in biochemistry and molecular biology from Tsinghua, where she used structural biology to investigate mechanisms of pathogens in the laboratory of Zihe Rao. She stayed on at Tsinghua for a postdoctoral fellowship in the laboratory of Haitao Li, where she began her reader protein work. She is now a Tsinghua advanced innovation fellow at the Beijing Advanced Innovation Center for Structural Biology.

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A yeast model for a severe neurometabolic disorder

By Vivian Tang

It may not be obvious why scientists sometimes use simple, unicellular organisms such as yeast to study complex human disorders. But yeast and humans share fundamental cell biology. More than 50 percent of yeast genes have a human homolog, and about 25 percent of human-disease related genes have a close yeast homolog. These strong similarities have made yeast a model system for understanding cellular processes such as protein degradation, synthesis, quality control, secretion, vesicular trafficking, oxidative stress, and cell survival and death. Rapid growth rate and ease of genetic manipulation also have made yeast useful for high-throughput genetic and chemical screening.

Yeast was chosen by Carole Linster and her team at the Luxembourg Centre for Systems Biomedicine of the University of Luxembourg and collaborators at the Luxembourg Institute of Science and Technology to study the rare neurometabolic disorder 2-hydroxyglutaric aciduria. Caused by elevated levels of 2-hydroxyglutarate, or 2HG, the disorder is characterized by delayed development, seizures, weak muscle tone and cerebral white matter abnormalities. In severe cases of the disorder, breathing and feeding problems can lead to death in infancy or early childhood. High 2HG levels also more recently have been found in certain cancers, hence the qualification of 2HG as an oncometabolite.

Although mutations have been identified in metabolite repair enzymes that degrade the two forms of 2HG, D-2HG and L-2HG, the main source of the oncometabolite D-2HG in humans has remained unclear. In a recent issue of the **Journal of Biological Chemistry**, Linster's group shares the discovery of a novel enzymatic activity that degrades D-2HG along with enzymes involved in the formation of the metabolite in the baker's yeast Saccharomyces cerevisiae.

The researchers identified Dld2 and Dld3 as the yeast homologs of the human enzyme that degrades D-2HG. They showed that deletion of DLD2 or DLD3 led to elevation of D-2HG levels of up to two-and twentyfold, respectively, in the yeast mutants. When they restored DLD3 expression in the mutant, D-2HG levels decreased almost to wild-type levels and further decreased by 50 percent when they overexpressed this gene.

The researchers subsequently proved that just like their human homolog, Dld2 and Dld3 convert D-2HG to alpha-ketoglutarate by serving as dehydrogenases. In addition, they discovered that both yeast enzymes also degrade D-2HG through a novel enzymatic mechanism by acting as transhydrogenases. Importantly, the transhydrogenase activity of the cytosolic Dld3 enzyme, which forms D-lactate as one of the products, suggests the existence of a coupling between cytosolic D-2HG metabolism and the mitochondrial respiratory chain.

Linster and her team went on to investigate the main enzymes that catalyze the formation of D-2HG. Since the expression profiles of the yeast 3-phosphoglycerate dehydrogenase Ser33 and Dld3 are highly correlated, and because Ser3 (paralog of Ser33), Ser33 and Dld3 are co-localized within the yeast cytosol, they tested the effect of overexpression and then deletion of SER3 or SER33 on D-2HG levels. They reported that overexpressing either gene further increased the accumulation of D-2HG and that deleting each gene partially reduced synthesis of the metabolite. Their observation that a double deletion of SER3 and SER33 reduced D-2HG levels by more than 80 percent compared with wild-type levels led them to conclude that Ser3 and Ser33 are major enzymes responsible for D-2HG formation in yeast.

Linster's group shares the discovery of a novel enzymatic activity that degrades D-2HG

The researchers plan to cultivate the yeast under different growth conditions and with different genetic backgrounds and then look for phenotypic changes associated with D-2HG accumulation. Since it is the primary enzyme that breaks down D-2HG, they are also keen to elucidate the roles of Dld3 in the retrograde response to mitochondrial dysfunction. Finally, they want to investigate how D-2HG accumulation may affect the structure of the yeast chromatin, its gene expression profiles and its lifespan.

According to Linster, "An obvious advantage of finding any phenotypic effects of D-2HG in yeast is that it can be followed up by high-throughput screens to pinpoint intracellular targets of D-2HG or to find chemicals that can rescue the phenotype. If conserved mechanisms are involved, the findings may then translate into advances in our understanding and treatment of human diseases characterized by D-2HG accumulation."

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Team effort to figure out a rare genetic disease

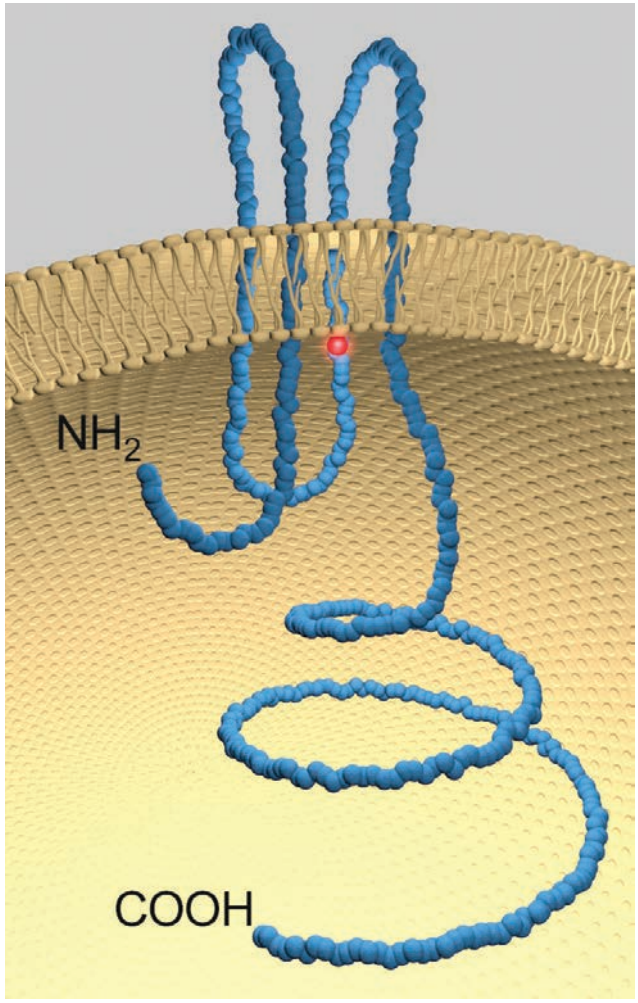
By Rajendrani Mukhopadhyay

About three years ago, Kristin Lindstrom was stumped. The medical geneticist, then based at the University of Rochester, had a patient referred to her by an endocrinologist. The endocrinologist was wondering if the patient's unusual combination of features could be attributed to an underlying genetic syndrome.

But the 17-year-old patient had features that didn't fit any descriptions of known genetic disorders. The teenager was intellectually disabled, unable to read or count past 30, and needed help with daily activities. Her ovaries failed to develop normally. She had severe hearing loss that required cochlear implants. Her back was growing hunched.

When Lindstrom's team did an evaluation of the young woman's chromosomes, they didn't see anything out of the ordinary. "We started thinking about the possibility of a single-gene disorder. I searched genetic reference books and online databases, but she really didn't fit any one syndrome perfectly," recalls Lindstrom.

So, in 2014, a year after Lindstrom first met the patient, her team decided to have the DNA taken from the young woman and her parents analyzed by whole-exome sequencing. Whole-exome sequencing searches for the parts of the genome that make proteins. Any variations in those protein-coding sequences could be mutations that cause a disease.



DALE LAIRD

A model of pannexin 1 with the location of the mutation highlighted in red.

People have two copies of every gene, one inherited from each parent. The results from whole-exome sequencing showed a mutation in a particular gene. The woman's parents each had one normal copy and one copy with the mutation. Both copies of the patient's gene had the mutation, indicating the inheritance of a disorder that only manifests when both copies are defective. The mutation was in a gene that made a protein called pannexin 1.

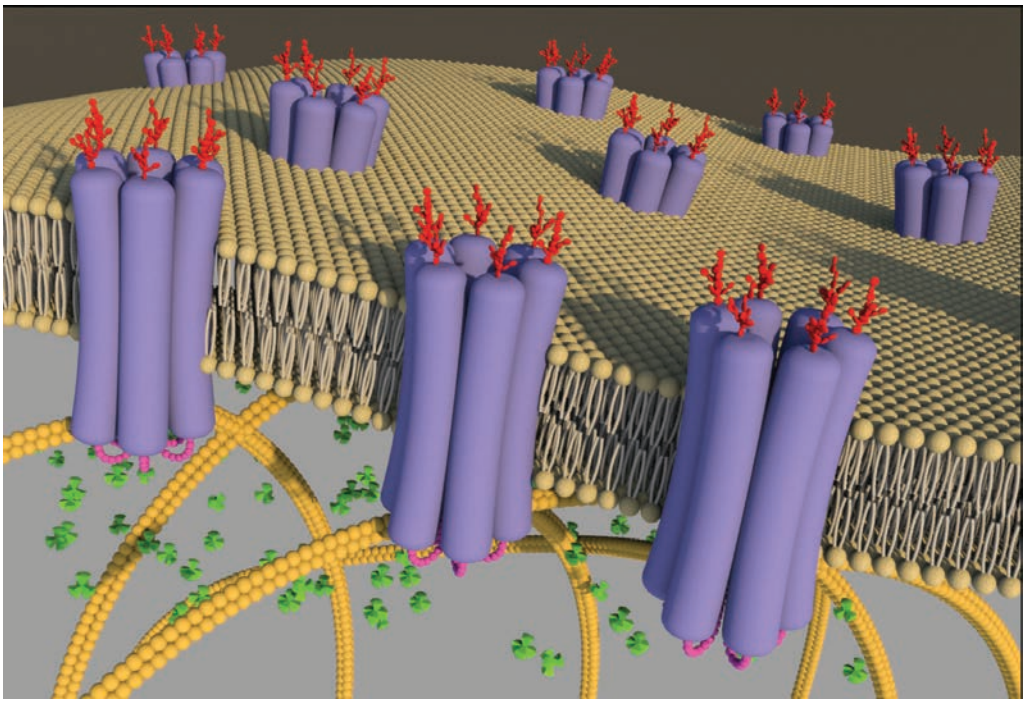
But what was pannexin 1? "Often the results of exome testing are for conditions that we already know something about," says Lindstrom, who since has moved to the Phoenix Children's Hospital. "But this was different." The young woman appeared to be the first patient to be reported with the mutation in the gene for pannexin 1.

Lindstrom had to turn to PubMed, the database of scientific papers. She began to hunt for scientists who would know about this protein and be willing to work with her team to find out more about the mutation.

That is how Dale Laird, a cell biologist at the University of Western Ontario in Canada, came to receive an email from Lindstrom about two years ago. Laird's research interests were on gap junction proteins. These proteins let neighboring cells exchange information, in the form of molecules, with one another. In 2000, a new family of channel proteins was discovered that initially seemed to be like gap junction proteins. Called pannexins, these proteins were later shown to be channels that release ATP and other small molecules.

More intriguingly, pannexins appeared to be in almost every tissue of the body. Because of their ubiquity, pannexins were implicated in diseases as diverse as Crohn's, osteoarthritis and cancer. "They've even been proposed to be hijacked by the HIV infection processes," says Laird. Laird's group became interested in pannexins, focusing in particular on the biochemical properties of pannexin 1.

After exchanging emails, Laird and Lindstrom decided to collaborate to parse out how the mutation in the pannexin 1 gene found in the teenaged patient affected the protein. The investigators found the mutation in the gene corresponded to a change from an arginine to a histidine in a loop proposed to be tucked within the channel.



Schematic image of Panx1 channels in the lipid bilayer. It shows oligomers of six glycosylated Panx1 subunits arranged into large-pore channels that are suitable for allowing the passage of ATP (green cloverleafs). Permission to reproduce image was given to Laird and reproduced from an article by Penuela. S. et al., Biochem J **461**,371-381 (2014).

By analyzing the mutant channel in cell culture, the investigators found that the protein was properly made and sent to its usual place of residence, the cell's plasma membrane. However, once it got to its destination, the channel was defective.

To characterize fully the improper functioning of the mutated protein, Laird and Lindstrom recruited the team of Michael Jackson at the University of Manitoba in Canada. Jackson's group are experts in measuring the function of channel proteins. They showed that the mutant pannexin didn't fully open, which explained why it was defective in releasing ATP.

The collaborators published their findings in the **Journal of Biological Chemistry**; their paper was selected as a Paper of the Week. But Laird says the work doesn't fully answer the questions: Does the defect in pannexin 1 actually cause all the features in the 17-year-old patient? Are there other factors at play?

The only way to answer these questions, says Laird, is to find more patients with the same or other pannexin 1 mutations. With more patients, researchers can understand better which symptoms and findings come with having mutations in both copies of the gene that codes for pannexin 1.

Here, Lindstrom has taken the charge to contact other laboratories that do genetic testing through whole-exome sequencing to see if there are other patients with two mutations in this gene who have the same features as her patient. "We can't really do much more clinically until the labs start identifying these patients. Nobody is sequencing people for this gene specifically yet, because, until our paper came out, most geneticists would have never heard about it!" she says. "You can't test for what you don't know exists."

She points out that whole-exome sequencing is a relatively new technique so "only a small fraction of people have had their exome sequenced." Lindstrom adds that she hopes that as the technology becomes more widely adopted, "we will have more people with similar mutations identified, and then it will be really interesting to see what these people all have in common."

On his end, Laird's next step is to dig deeper into the molecular and cellular effects of the mutated pannexin 1. He and his team are hoping to create a laboratory mouse that carries the mutated pannexin 1. He says, "The idea would be to see if a mutant mouse demonstrates some of the phenotypes that we see in the clinical presentations" in the teenage patient.

But Laird emphasizes the work is all about collaboration. "This is a team approach," says Laird, noting that, for the JBC paper, every team brought in different skill sets that helped the full biochemical characterization of the mutated pannexin 1.

But he saves the biggest shout-out for Lindstrom: "If she didn't contact me in the first place, we wouldn't have gotten this off the ground."

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The taxi driver's book

How a Scottish cabbie amassed a veritable history of science in signatures.

By Bree Yanagisawa

PHO-TAY PHOTOGRAPHY

Davie Douglas, a beloved driver who often transports scientists for the University of Dundee, stands before the school's life sciences building.

PHO-TAY PHOTOGRAPHY

Scientists traveling between the University of Dundee and Edinburgh in Davie Douglas' taxi are treated to views of the Forth Bridge, one of the world's first cantilever bridges.

Very early on a dark and cold January morning in Scotland, Stanford University's Suzanne Pfeffer climbs into the back of a taxi, ready to head home to the U.S. Still half asleep, she settles into her seat for the hourlong drive from the University of Dundee to Edinburgh airport. A few minutes into the ride, which winds through stone villages, past patterned green hillsides and over bridges that cross the River Tay, her cabbie — who also drove her to the university when she arrived in Dundee — makes a surprising request. He asks Pfeffer, an expert in the study of receptors in human cells, if she'd like to see his guestbook.

Pfeffer agrees, and the driver hands her a book with a simple, dark blue cover that is tattered at the edges from years of use. As she leafs through, she sees a hodgepodge of scribbled sentiments, sketches and signatures. When she looks more closely, Pfeffer realizes the signatures comprise a veritable who's who of the life sciences. Among the scrawls of notable scientific contemporaries, Pfeffer can make out the names of four Nobel laureates. There's Sir Tim Hunt, who drew an off-kilter sketch of a cell cycle alongside his note. And there are Elizabeth Blackburn, Edmond Fischer and Aaron Ciechanover.

Soon Pfeffer, who is a former president of the American Society for Biochemistry and Molecular Biology, is deep in conversation with her cabbie and struck by the depth of his knowledge about his former passengers.

Remembering their interaction some months later, she says, "He really understood how important these riders were, and in what esteem we, as scientists, hold them."

Soliciting signatures

For the past 20 years, 69-year-old Davie Douglas has worked as the driver for the School of Life Sciences

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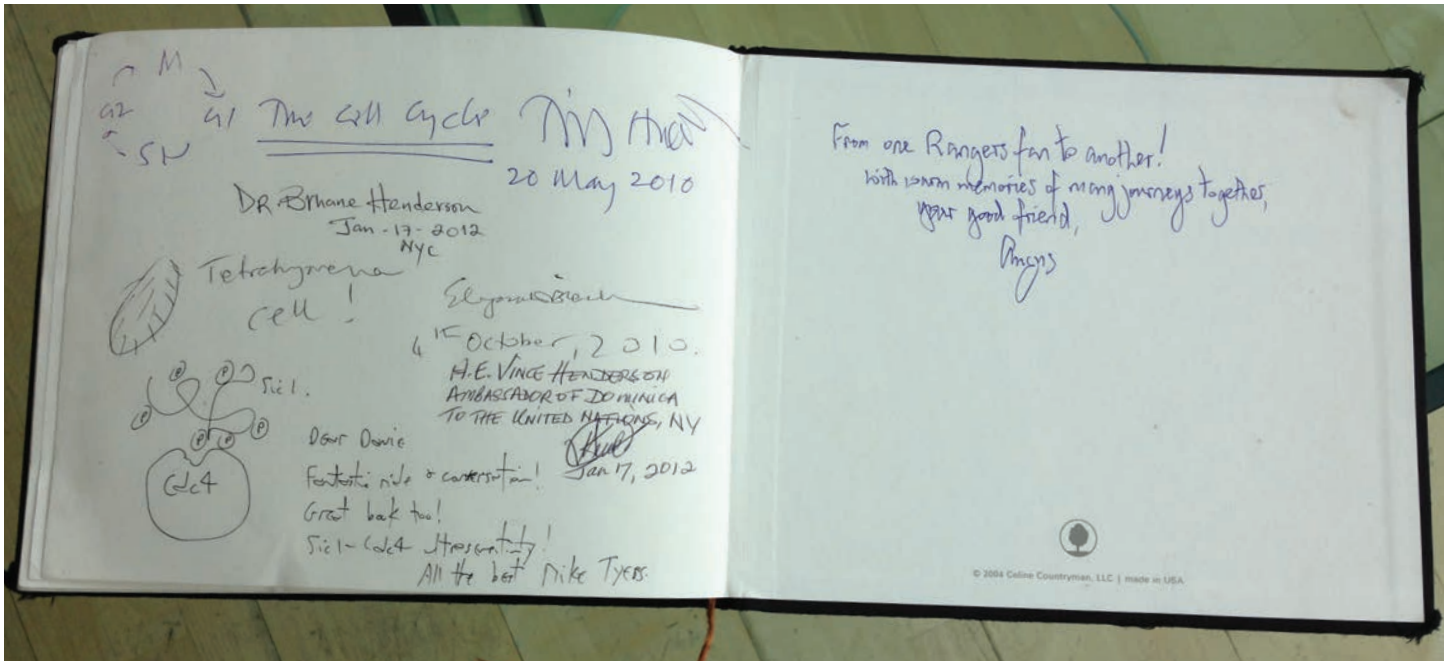

THE UNIVERSITY OF DUNDEE

Douglas asks scientists who ride with him to sign a guestbook. Below are playful drawings by Nobelists Sir Tim Hunt and Elizabeth Blackburn and cell-division expert Mike Tyers.

PHO-TAY PHTOGRAPHY

This patch of Scottish countryside near the city of Perth is on Douglas' Endinburgh airport route.

CONTINUED FROM PAGE 21

at the University of Dundee. A hub for local and international research into cell signaling and other biological processes, the university attracts a plethora of scientists to its facilities. (Nearby golf courses and whiskey distilleries are also part of the draw.) For the past nine years, Douglas has been collecting the signatures of these scientists in his guestbook.

Douglas acknowledges that some of his scientist passengers are true luminaries but is pragmatic about his interactions with them. "They're just normal people, like ourselves," he says. "They just get in your car and talk away."

It's rare, Douglas says, for his riders to talk about science, and his intuition tells him it's best not to push. "I think they're usually fed up by talking science. They'd rather just talk about football or anything else that comes to their minds."

Douglas' guest book was born of a conversation he had with Dario Alessi, director of the Medical Research Council Protein Phosphorylation and Ubiquitylation Unit at the university. Knowing how many prominent scientists were using Douglas' services, Alessi suggested a book as a means of preserving those interactions.

Douglas was hesitant and put off buying the book. But Alessi persisted.

"Dario says, 'Did you get a book yet?' I say, 'No.' He says, 'Here you are. There's a book here for you.'"

With the guestbook thrust upon him, Douglas began requesting signatures from his passengers. But not everyone was asked to sign. Joking, Douglas says, "You can't just sign the book. You've got to be invited to sign the book! If I didn't like ya, ya didn't get to sign the book."

He clarifies that scientists not being asked to sign is only ever a result of his own forgetfulness.

A treasure trove of notes from biochemistry greats, the book now contains more than 450 signatures, filling 33 pages. As Alessi says, Douglas "probably meets more eminent scientists than the average scientist would meet in their working career."

The guestbook showcases a whimsical side of some of these revered scientists. A playfully drawn tetrahymena cell sits alongside Elizabeth Blackburn's message. It's just above a sketched interaction between two cell-cycle proteins from another signee, systems biologist Mike Tyers. The whole book reads more like a yearbook, with doodles, meaningful thank-you's and promises to come back soon filling the pages.

Prelude to a cabbie

Davie Douglas wasn't always a cab driver. He started out as a seaman, a member of the British Merchant Navy. While in port in Dundee doing some shore-based work, he started taking fares on the side.

"I really hated it. I was getting ready to go back to sea," Douglas says.

PHO-TAY PHOTOGRAPHY

Passengers who have the time to be taken along Douglas' "tourist" route are treated to Falkland Village in the historic county of Fife.

Then one fateful morning, his daughter called.

It was early. Douglas was still in bed. His daughter, who was a graduate student at the University of Dundee at the time, told him the school's taxi driver had failed to show up. Douglas agreed to take the university's waiting guests to the airport.

That early-morning drive set his new career in motion. Before he knew what was happening, the university was helping him get a cell phone to make contacting him for jobs easier. A self-proclaimed technophobe, it took a while for Douglas to become accustomed to this new work. "The first mobile phone I ever had was the size of a brick," Douglas says with a laugh. "I still am really computer illiterate, and I'm quite happy to stay that way."

But Douglas manages well enough. His job now revolves around that cell phone, which he uses to coordinate transportation for staff and visitors to the School of Life Sciences.

Preserving a family record

Pfeffer says it was "priceless" to page through the book as they were driving through the Scottish countryside and see so many recognizable names together in one place. "I knew all the people," she says. "They were all part of the family of science."

Those who know Douglas say that his charm and goodwill make creating this kind of family record possible. He is known for going above and beyond to entertain his passengers. According to Alessi, "If someone's got a bit more time to get to the airport he'll take them on the more scenic route and show them around Scotland."

In caring for his passengers, the line between job and lifestyle often blur. "I don't even consider myself a taxi driver anymore," Douglas says. "I've got a meter in my car, and I couldn't even tell you how to turn it on." But he wouldn't have it any other way. He has, he says, "the best job in the world."

Alessi says that Douglas also helps keep his University of Dundee family on track. "Often if you want to find out what's going on in the university the quickest way is just to have a 10-minute conversation with Davie, who can bring you up to speed."

The book is now almost full. Douglas would like to get the pages laminated and make it available for others to read. With his 70th birthday looming, Douglas also hopes to wrap up his taxi career within the year. But for now, he continues to do the work that he loves. It's work that Pfeffer thinks could have a lasting impact. As she says, he's recorded a history of science "simply by having a guest book in his car."

Bree Yanagisawa (breannwoelfel@gmail.com) was an intern at ASBMB Today when she wrote this story. She is currently a Ph.D. candidate in pathobiology at the Johns Hopkins School of Medicine. Follow her on Twitter at twitter.com/BreeTalksSci.

PHOTO PROVIDED BY DARIO ALESSI

Douglas, left, with Dario Alessi, director of the Medical Research Council Protein Phoshorylation and Ubiquitylation Unit at the University of Dundee.

JOHN D. AND CATHERINE T. MACARTHUR FOUNDATION

In 2012, Sarkis Mazmanian received a MacArthur Foundation award.

A gut reaction

Sarkis Mazmanian is helping to change our view of gut bacteria as well as the education of Armenian graduate students

By Rajendrani Mukhopadhyay

About eight years ago, microbiologist Sarkis Mazmanian boarded an airplane in Los Angeles bound for Armenia. The future MacArthur award winner didn't know anyone there. All he had was an idea and a desire to make a difference.

Mazmanian recently had landed his first faculty position at the California Institute of Technology. "I realized I was in a privileged and unique position to help people," he says. "I felt that in some ways, it would be a shame — I'll even push it to a crime — if I don't help people who need modern scientific training desperately."

Mazmanian's idea was to bring 21st-century science to graduate students in the economically struggling former Soviet republic. "I can't help everybody," he says. "But helping people in Armenia conceptually appealed to me."

For five years running, Mazmanian, who is Armenian by blood, has spearheaded a short course in the Armenian capital of Yerevan. He and his co-lecturers teach the latest microbiology and molecular biology research to about 40 graduate students and their professors and work with them to advance their own studies.

Mazmanian says the course provides opportunities to people who are willing to work hard but who lack resources. After all, that's his own life story.

Rise up

Starting in 1915, the Ottoman Empire (also known as the Turkish Empire) began to exterminate and expel Armenians out of territories they had occupied for thousands of years. The Armenian Genocide is estimated to have claimed up to 1 million lives. Mazmanian's ancestors were among the hundreds of thousands of Armenians who escaped.

Mazmanian was born in Lebanon in 1972. When he was a year old, he and his family immigrated to the U.S., joining the large Armenian diaspora in Los Angeles.

"My parents have no education," says Mazmanian. "They've never even been to first grade. They were raised in a time when they didn't have access to education."

His father, who ran a small gift shop, and his mother, a homemaker, were determined things would be different for Mazmanian and his two sisters. Mazmanian says his parents worked hard in their pursuit of the American Dream and that they instilled in him the belief that "you can overcome a lot of deficiencies just by working very hard."

"Nobody opened doors for me. I can say that confidently. My father isn't a professional. We didn't have the financial resources to get ahead. But no one slammed doors in my face either," he says. "That's the opportunity the U.S. provides. That's 90 percent of the battle right there. You go as far as you can take yourself."

One of his sisters owns a trucking company; the other is a senior vice president of a major bank.

"There's no guarantee for success, but the way you can increase your likelihood to be successful is to work hard and make good decisions," he says.

Mesmerized by the machines

Making good decisions is something at which Mazmanian appears to excel. Many scientists, though certainly not all, cite a childhood dream of becoming a scientist. Mazmanian initially didn't know what he wanted to do.

His high-school English teachers told him he had an aptitude for writing, and, to prove it, one sent off a poem of his to a national poetry competition. Mazmanian won first place. He entered University of California, Los Angeles, as an English major.

"I was taking introductory biology classes just to satisfy prerequisites for college. In high school, I never really liked science but as I matured, I really appreciated it," recalls Mazmanian. "And then this is where serendipity comes in."

Mazmanian wanted to take a molecular biology course, but the class was full. He opted for a microbiology course. "Something really resonated with me about microbes," he says. "I thought that they were a wonderful system to study because I could learn a lot about how nature works from these self-contained machines."

Mazmanian switched majors. He graduated with a bachelor's degree in microbiology and molecular genetics. For graduate school, Mazmanian joined the group of microbiologist Olaf Schneewind, then based at UCLA. There, Mazmanian discovered sortase, an enzyme found in Gram-positive bacteria that anchors surface proteins to the bacterial cell wall.

Then he took a step that put him in uncharted scientific territory and eventually made his name as a scientist. In 2002, Mazmanian arrived at Dennis Kasper's laboratory at Harvard University as a postdoctoral fellow. At the time, Mazmanian was mulling over an unusual idea.

ARSEN ARAKELYAN

Mazmanian spearheads an annual course about microbiome research in Armenia.

Bacteria had been studied predominantly from the point of view of the harm they cause to humans. What if, wondered Mazmanian, there were ways in which bacteria help humans? And if they were capable of helping, could bacteria be used as therapies?

"The notion that bacteria could be used as treatments for disease at the time was scientific heresy," says Mazmanian, who describes Kasper as "a courageous person who took on a fellow who wanted to do something that was completely out of the box."

The idea made getting funding for Mazmanian's postdoctoral fellowship difficult, with some agencies rejecting his research proposal. Finally, he received a fellowship from the Helen Hay Whitney Foundation.

Kasper had been considering beneficial bacteria as well. His group previously had discovered a molecule called polysaccharide A, or PSA, that the symbiotic gut bacterium Bacteriodes fragiles made. PSA appeared to be a potent trigger in human cells and in the immune system of mice.

"It induced T cells to produce

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interleukin 10, which is an immunosuppressive cytokine," explains Kasper. "In conversations with Sarkis, the thought evolved that this organism which has a normal place in the microbiome — and this was before the word 'microbiome' was used and we used to say 'gut flora' — may have had something to do with immune tolerance."

Research into beneficial bacteria was the Wild West of microbiology. There was very little about it in the scientific literature. Therefore, there was little to influence Mazmanian's thinking.

Bacteria engage the immune system — and the immune system engages bacteria — during an infection. What if, wondered Mazmanian, there were scenarios in which the bacteria actively engaged the immune system and shaped it in a way that was actually beneficial to the host?

SARKIS MAZMANIAN

Mazmanian in front of a 13th century monastery in Armenia.

Mazmanian began a series of experiments. He took two sets of laboratory mice. One set were regular mice with a community of gut bacteria; the other set were mice free of any resident microorganisms. "I discovered a very striking phenotype" in the germ-free animals, recalls Mazmanian.

The germ-free mice, compared with their normally colonized counterparts, had fewer specific T cells in their immune system. But giving the germ-free mice B. fragilis reversed the defects in the T cell population. The finding suggested that microorganisms influence how important parts of the immune system develop and function.

Mazmanian next gave the germ-free mice only PSA. "I showed that this polysaccharide was both required and sufficient for inducing the T cell phenotype," he says.

The first paper with Mazmanian and Kasper as co-authors came out in Cell in 2005. It described how PSA on B. fragilis molds the mammalian immune system. "It was a concept that resonated with people who study microbes," says Mazmanian. "In addition to our own genome, additional signals from gut microbes are needed for the full maturation of the immune system."

Besides fighting off external threats, the immune system also keeps an eye on the body to fight off certain diseases, such as inflammation and cancer. Did bacteria play a role here too?

In 2006, Mazmanian started at Caltech as an independent investigator and began to look into inflammatory bowel disease, an autoimmune disorder where the body thinks there is a problem in the gut and attacks it. Mazmanian and his group eventually showed that B. fragilis has protective effects against irritable bowel disease in mouse models. The finding hinted that the bacterium could be a potential therapy for the disease, harking to the notion Mazmanian harbored when he was beginning his postdoctoral work.

"It was very gratifying (to see those reports), but I'd be lying if I said it wasn't also shocking," he says. "I firmly believe you have to ask big questions, but that also puts you in a position where the likelihood of success is lower."

Gut–brain connections

In 2010, someone made a comment that changed the course of Mazmanian's career. That someone was Paul Patterson.

Patterson, who passed away in 2014, was a neuroscientist at Caltech who studied the effects of the environment on autism. Over a casual cup of coffee, Mazmanian recalls, "I was telling him about our work in inflammatory bowel disease and how we're able to successfully restore gastrointestinal health with specific organisms. Paul said, 'You know, many autistic children have gut complications.'"

Mazmanian and Patterson began to wonder if the genetically engineered mouse models for autism carried signs of gastrointestinal damage, the kind Mazmanian's group was used to observing in mouse models for irritable bowel disease. Furthermore, the two scientists wondered if mice that develop autismlike symptoms could be treated with probiotics to ease their GI symptoms and, potentially, change their behavior.

The Patterson group usually analyzed the brains of the mice with the autismlike symptoms. Mazmanian asked for the guts. The first time the Mazmanian group looked at the guts, the signs of a proinflammatory response were there. It wasn't a full-blown chronic inflammation as seen in irritable bowel disease, but it was detectable.

Next, the Mazmanian and Patterson groups treated the special mice with B. fragilis. "We were able to show that the GI symptoms were reversed in the mice when we treated them with this human commensal bacterium," says Mazmanian. "But, even more fascinating, and perhaps surprising, was that their behavioral deficits also improved."

The collaborators dug in deeper and soon figured out that the behaviors were not being improved through the immune system. Something else was happening. Enter the "leaky gut."

The "leaky gut" idea envisions an intestine damaged badly enough to become porous. Chemicals that would normally leave the body through the excretory systems instead leach out of the damaged intestine and into the bloodstream. The chemicals get to the brain through the blood, cross the blood–brain barrier and interfere with neurological development. The hypothesis gives rise to the notion of the "gut–brain axis" where the intestine and the brain, two seemingly separate organs, are intimately connected.

JOHN D. AND CATHERINE T. MACARTHUR FOUNDATION

Mazmanian's group is now focusing on connections between the gut microbiome and brain development.

Where does a symbiotic gut bacterium come into play? In 2013, Mazmaniam's group showed in a Cell paper that giving B. fragilis to the mice with autismlike symptoms appeared to lessen the behaviors associated with autism spectrum disorder.

Mazmanian emphasizes that the work on the gut–brain axis is in its early days. "There's still a long way to go, but at least it gives us a mechanism to try to understand the potential link between the gut and the brain," he says. "If the causal events for neurodevelopmental disorders are happening in the gut, perhaps it is viable to think about treating the gut as a potential treatment for autism. We've taken a few steps, but we're not there yet."

Kasper, with whom Mazmanian has monthly phone calls, is tickled to see how his former postdoctoral fellow's research has played out. "When he

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Country name in conventional long form: Republic of Armenia

Location: Southwestern Asia, between Turkey (to the west) and Azerbaijan

Size: At 29,743 square kilometers, Armenia is slightly smaller than the state of Maryland

Climate: Highland continental, hot summers, cold winters

Population: 3,056,382 (estimate from July 2015)

Languages: Armenian (official) 97.9%, Kurdish (spoken by Yezidi minority) 1%, other 1% (estimate from 2011)

National holiday: Independence Day, 21 September (Armenia became independent of the Soviet Union in 1991)

SOURCE: THE CENTRAL INTELLIGENCE AGENCY'S WORLD FACTBOOK (HTTPS://WWW.CIA.GOV/LIBRARY/PUBLICATIONS/THE-WORLD-FACTBOOK/GEOS/AM.HTML)

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was still in my lab 10 years ago, he actually said, 'I think there is going to be a connection between the microbiome and autism,'" recounts Kasper. "I was going, 'What is he talking about?'"

'Don't take credit'

Most researchers working at the forefront of one of the hottest avenues of scientific investigation of the early 21st century likely would be satisfied with focusing exclusively on that work. But not Mazmanian.

Every year, he returns to his ancestral homeland to teach an annual four-day course with his co-lecturers, Bana Jabri from the University of Chicago and Haig Alexander Eskandarian at the Swiss Federal Institute of Technology.

The course focuses on host–microbe interactions. Mazmanian, Eskandarian and Jabri kick off the course by giving lectures about the latest microbiome research. Then they lead a journal club. For the journal-club portion, the students get into small groups, read a scientific research paper and then give presentations based on what they learned.

Arsen Arakelyan heads the Institute of Molecular Biology at the National Academy of Sciences of Armenia, which hosts the course. "There are huge differences in knowledge delivery between the Soviet system, which we have adopted here for a long time, and the Western educational system," says Arakelyan. "Students who attend the course are really amazed at how freely Sarkis communicates. It isn't just a lecture with Sarkis talking and people listening. It's interactive."

Arakelyan says, as an educator, he has learned much from watching Mazmanian. "I have grown up in the atmosphere of Soviet educational system," says Arakelyan. But these days, "when I lecture at university, I use a lot of (Mazmanian's) tricks. I have learned from him a lot about how to deliver a lecture and communicate."

The course content isn't strictly stuck on the microbiome. Students discuss their own research. "We try to have a young scientist present his or her own work in front of Sarkis and colleagues," says Arakelyan. The co-lecturers "give suggestions and advice on how to proceed with the research or ask if the interpretation of results is correct or not."

Arakelyan notes that the lecturers help faculty members by critically reading their scientific research manuscripts and recommending appropriate journals for the papers. Arakelyan also says there have been discussions about establishing research collaborations between Armenian researchers and the Mazmanian group. But he adds that not many agencies fund research collaborations between the U.S. and Armenia.

Mazmanian, for his part, wants to do more. His 2012 MacArthur award came with $500,000, which gets paid out in equal quarterly installments over five years. "I've not spent a dollar of that money," he says. "I'd like to explore how to most efficiently use those funds to help research and science in Armenia." Mazmanian notes that the Armenian research facilities lag behind the ones in the U.S. He's considering using the money to upgrade laboratory space in a research institution and installing more modern equipment.

Although he's happy to talk about his trips to Armenia and his research, Mazmanian doesn't see what he's doing as anything out of the ordinary. "Don't take credit for what you're supposed to be doing," he says.

Rajendrani Mukhopadhyay (rmukhopadhyay@asbmb.org) is the chief science correspondent for ASBMB. Follow her on Twitter at twitter.com/rajmukhop.

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Meet Natalie Ahn, ASBMB's incoming president

By Angela Hopp

Natalie Ahn, a professor of distinction at the University of Colorado, Boulder, was elected president of the American Society for Biochemistry and Molecular Biology in the summer of 2015. Her term begins July 1. Ahn previously served as a member of the ASBMB Council. Her lab uses proteomic, cell biology and biophysical approaches to uncover new signal-transduction mechanisms. She was a Howard Hughes Medical Institute investigator from 1994 to 2014. She spoke with ASBMB Today's executive editor Angela Hopp, about her background and involvement with the ASBMB. This interview has been edited for length, style and clarity.

What was your childhood like?

I grew up on U.S. military bases in Korea and Japan. My father emigrated from Korea and worked at the U.S. embassies in Seoul and Tokyo. My mother was a finance clerk at the embassy. I have one brother who's an engineer and builds airplanes.

I was always interested in science. When John Glenn became the first American to orbit the Earth, I remember riding my tricycle and thinking that I too wanted to be an astronaut. Later, when I became an assistant professor at the University of Colorado, three of my first lab members had segued into biochemistry after first starting in aerospace engineering. So I appreciate how NASA and other big-science efforts promote science by inspiring kids.

Tell us about your education and training.

After finishing a high school degree overseas, I majored in chemistry at the University of Washington in Seattle and did undergraduate research in X-ray crystallography with the late

Lyle Jensen and protein hydrodynamics with David Teller. I obtained my Ph.D. in chemistry from the University of California, Berkeley, where I studied enzymology with Judith Klinman. Judith is an incredibly deep thinker as well as a generous and courageous individual who continues to be one of my greatest inspirations.

My first postdoctoral job was with Christoph de Haën at the University of Washington, where I studied hormone receptor binding. Christoph was unable to renew his funding and had to close his lab, and that's how I learned about the importance of grants! He ended up great anyway, eventually becoming director of preclinical research at Bracco and director of the Milano Research Center. I then moved to the lab of the late Edwin Krebs for a second postdoc, where I was among the first to describe MAP kinases and MAP kinase kinases. That started my career in signal transduction.

Tell us a little about your current work.

I still work on MAP kinase and other signaling pathways. When I started at the University of Colorado, I began applying the new technology of protein mass spectrometry to address questions in signaling. This was done in collaboration with my late partner, Katheryn Resing. My lab's applications of proteomics to signal transduction have led to broad discoveries, ranging from new mechanisms for cell regulation to mechanisms for allosteric control of MAP kinases.

How did you get involved with the ASBMB in the first place?

I attended the ASBMB annual meeting during my graduate studies, and it was at this meeting that I gave my first public research talk and got to meet the leaders in enzymology. That was a spectacular experience. When at the end of my first postdoc I had no way to pay for an accepted manuscript, the Journal of Biological Chemistry generously waived page charges, allowing me to publish. Since then, I've helped organize symposia at the annual meeting and served on the ASBMB Council. It was the support by the ASBMB during the crucial early years of my career that engendered my long-lasting love for this society.

PHOTOS COURTESY OF NATALIE AHN

Ahn received her education and training in the western U.S. and now teaches at the Unviversity of Colorado.

How would you describe your leadership philosophy or style? Do you see any crossover of your lab leadership style in your approach to leading a scientific society?

I try to be involved in every aspect of my lab, but I let my students and postdocs — currently eight in all — work independently enough to discover their strengths, while following behind to support them. That's not too different from the way I view leadership elsewhere, where my instinct is to try to solve the most important problems and avoid fixing what's not broken.

The ASBMB has had some pretty outspoken, political and provocative

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presidents. It's also had diplomatic, some might even say reserved, presidents. What kind of president do you want to be?

I'm not a flashy or provocative person, although I am unafraid of taking a stand when it's important. As president, I plan to focus on gaps and weaknesses at the ASBMB and the detailed steps needed to address them.

What are your priorities as president of the ASBMB?

Right now, we have three priorities. First is to recapture the annual meeting's reputation as a must-attend event. Second is to expand our visibility and membership, especially among young investigators. Third is to restore the prominence and stature of the Journal of Biological Chemistry, where we've been fortunate to appoint Lila Gierasch as editor-in-chief and Fred Guengerich as deputy editor.

There's a lot of competition among associations today. What do you think makes the ASBMB stand out in the crowded field?

The ASBMB has profoundly influenced discovery, and the importance of BMB in our current era is growing, not shrinking. This is obvious just by looking at the approximately one-third of human open reading frames and majority of noncoding RNAs whose functions are unknown and the overwhelming numbers of new regulatory connections generated from large-scale studies. Our discipline is the cornerstone of what's needed to discover the functions of new molecules and mechanisms underlying their connections.

When you're out there in the world, how do you describe the value of being an ASBMB member?

Many researchers, especially new investigators, may not be aware of the pivotal contributions of ASBMB members to current knowledge and how much the society has done and continues to do to promote everyone's careers and to advocate for the support that allows them to do their work. That's what I want to communicate — that ASBMB has benefitted every individual and that, in order to continue doing so, it is essential for all of us to be fully engaged with the society.

It's our custom to ask senior scientists what kind of advice they offer their students. What's yours?

I tell my students to envision what they want and where they want to be, then work backward to figure out how to get there. I can't say that I've always done this myself, because I tended to put one foot in front of the other and follow my curiosity, taking opportunities as they came. It took me awhile to learn how goal-driven the rest of the world is. But it's taught me a lot, and if I were a student now I would appreciate the advice. Second, never stop believing in yourself. When everything seems to fall apart, make a list of what you have that you're grateful for, and then keep going.

Angela Hopp (ahopp@asbmb.org) is the ASBMB's communications director and ASBMB Today's executive editor. Follow her on Twitter at www.twitter.com/angelahopp.

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Cows, Wall Street and science

How applying findings on diversity and bias can help improve the research workforce

By Hannah Valantine

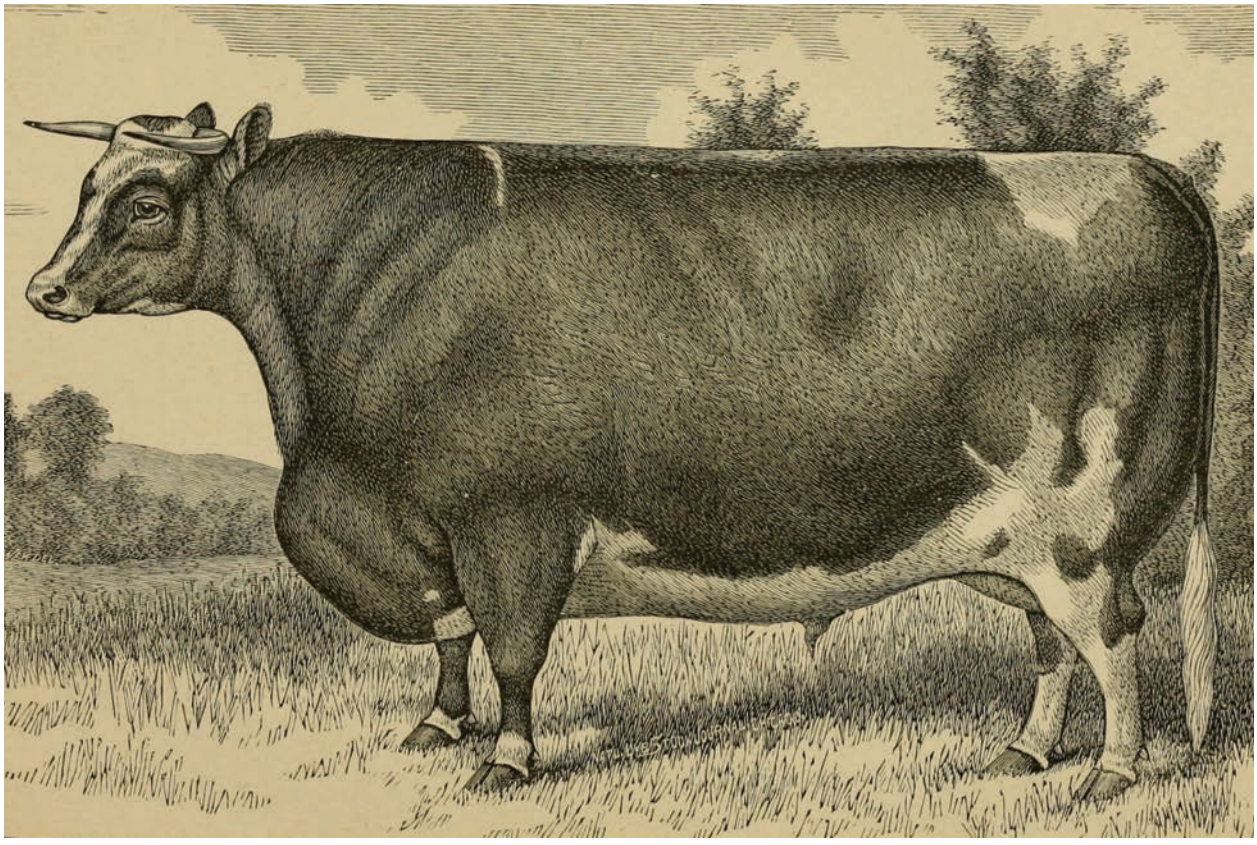
Diversifying the research workforce is inherently complex. At the National Institutes of Health, we have learned that addressing such complexity requires a rigorous scientific approach, which is consistent with the ways that we address the challenges of science discovery.

The scientific necessity of diversity

The discovery that happens at the NIH gets done by people — creative scientists of all types all across the nation. As the NIH's chief officer for scientific workforce diversity, I am keenly interested in a couple of deceptively simple questions when it comes to hiring these people. Who are the best ones for the task, and how can we be sure we're getting the best ideas? Luckily, science is helping me answer these questions. It tells me that in many ways diversity drives innovation and that we ought to be assembling diverse teams of scientists if we want to hasten discovery.

Let's consider a few real-life scenarios

It's 1906 at the West of England Fat Stock and Poultry Exhibition. People are lined up to guess the weight of a steer on display. Some know a lot about the weight of such animals. Others don't. In the end, all the guesses are averaged. That average turns out to be within one pound of the actual weight of the cow and closer than any one person's guess. This finding, which has been replicated in



At the turn of the 20th century, a diverse group of exhibition-goers were asked to guess the weight of a displayed steer. Their averaged guess was better than the guess of any single person's, be they amatuer or pro. This finding has been replicated in a variety of settings in the century since.

Research shows that mental shortcuts, or heuristics, lead to judgment errors that emerge in everyday life and also in major decisions, such as recruitment and hiring. Because the human brain is wired to process multiple pieces of information rapidly by using mental shortcuts, we are all guilty of bias.

other settings, suggests that the output of a group of different people is more accurate than any one person, be they an amateur or a pro.

In a more recent study from 2014, a group of ethnically similar financial traders were 33 percent less able to predict stock prices accurately than an ethnically diverse group. What's more, the similar group members accepted inflated prices based on speculation, contributing to a harmful financial bubble (1).

But these findings about diverse versus non-diverse groups don't just play out at stock shows and on Wall Street. They are true in science as well. In a 2013 study, economists looked at 2.6 million U.S. scientific papers across many fields, using surnames of co-authors as a proxy for assessing ethnic diversity (2). They controlled for other factors. The first result was expected. Like associated with like, and ethnically similar scientists published more often together. But a second result may surprise you. Those papers with co-authors of multiple ethnicities had more citations and were published in journals with higher impact factors.

Researchers believe that when diversity produces better outcomes, it is because friction is productive. In the face of difference, we have a tendency to take a closer look at our own assumptions, and we feel compelled to listen to others' views.

You could sum it up this way: The wisdom of the crowd is the power of diversity. But if we know this, why do scientists who pride themselves on objectively evaluating data consistently rate applications with male names higher than those with female names, even when the information contained in the applications is identical? Why are applicants whose names suggest that they might be African-American less likely to receive a job call-back compared with applicants with white-sounding names?

This essay is part of ASBMB Today's ongoing discussion of diversity and inclusion matters in biochemistry and molecular biology. Visit **www.asbmb.org/asbmbtoday** for more.

Tackling bias

Something is getting in the way of our ability to leverage the power of diversity. Is that something simply bias? And, if so, is it bias that we are aware we have or bias we don't even recognize in ourselves?

Research shows that mental shortcuts, or heuristics, lead to judgment errors that emerge in everyday life and also in major decisions, such as recruitment and hiring. Because the human brain is wired to process multiple pieces of information rapidly by using mental shortcuts, we are all guilty of bias.

Recognizing this human truth is a first step to interrupting subconscious snap judgments that may not have the best — or fairest — outcomes. At the NIH, we are working on ways to minimize subconscious, or implicit, bias in scientific settings. One way that we do this is by providing educational modules on bias to scientists leading recruitment searches. To see whether the modules are effective, we give a pre- and post-test to measure attitudes known to be associated with certain biases, and then we assess the diversity in their selected pool of candidates. At other institutions, these approaches have had a positive impact on diversity outcomes, and, if we are successful, we hope to expand the education across the NIH. Stay tuned for the results!

Hannah Valantine (hannah.valantine@nih.gov) is the chief officer for scientific workforce diversity at the National Institutes of Health. She leads the agency's effort to diversify the national scientific workforce and expand recruitment and retention.

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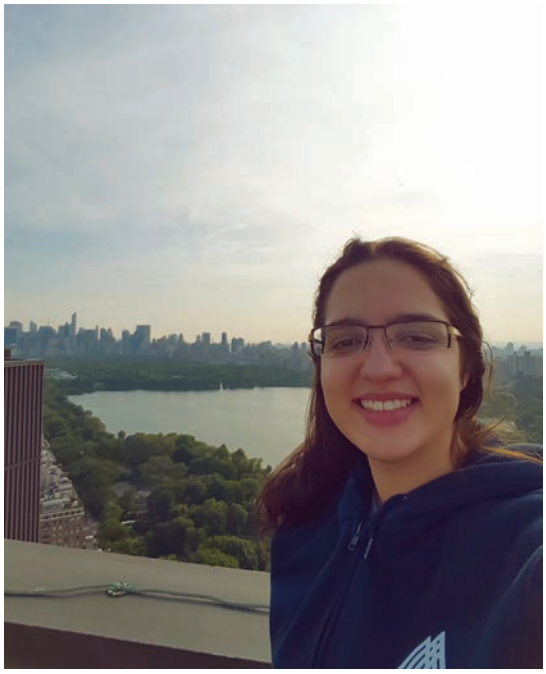
Back on the road with Coursera

By Caroline Monteiro

I'm a biologist. It's something I know in my heart. From a young age, I was completely sure of my career path and absolutely certain about what I would and would not spend my time learning.

Japan

I received a bachelor's degree in biology in my home country of Brazil and was eager to start graduate school and continue along my path to becoming a practicing scientist. I decided that I wasn't going to pursue my doctorate just anywhere, however. I was set on studying in Japan. Before applying to a Japanese graduate school, I decided to prepare myself for this potential transition and spent a few months in Japan — mainly Tokyo — getting to know the lifestyle and culture and making sure I could adapt to it all.



PHOTOS COURTESY OF CAROLINE MONTEIRO

Monteiro always wanted to be a scientist. Her journey so far has taken her to Japan, Europe, and, with the help of a serendipitous Coursera connection, New York City.

Europe

As I prepared to head home from Japan after those initial test months, I realized that for the first time in my life there would be no school waiting for me upon my return. On a whim, I decided to take that time and backpack through Europe. It was on my way home, after all!

My sense of geographical direction is nonexistent. So, to explore Europe effectively, I found myself approaching lots of strangers, asking for directions and then heading out, circling back and asking again. When this didn't work, I just kept walking until I found something — anything — interesting. If this kind of wandering worked for Columbus, I reasoned, it should work for me too. The trip reified my idea of myself as someone who possessed a carefree attitude and a personality that was amenable to change.

Brazil

When I returned to Brazil, I spent a whole year studying Japanese. I was going to take the Japanese-Language Proficiency Test and, once I'd passed, go back and apply to Ph.D. programs in Japan. It didn't worry me that this was a challenging prospect. In all the previous months of traveling, there never had been a moment when all the big and scary things everyone had warned me about actually had sunk in. Nothing, I reasoned, was going to frighten me away from this decision.

That is, until something did. When the 2011 earthquake and tsunami hit Japan a couple of months before I was supposed to go back, I discovered that in fact I was not someone who easily adapts. It turns out that the real reason I imagined I was not scared of anything, including natural disasters, was that I never had been affected by tragedy. Up until that moment, I was choosing my destiny. I was the one deciding my path. But the cataclysm that befell the island of Japan shifted all that, and I could only react.

I didn't go back. I'd grown up in the middle of Brazil in a relatively safe area with a loving family and no

worries about natural or man-made tragedies. The destruction in Japan broke my heart and, with it, my desire to go back to school.

I stayed in Brazil and started working in industry. But after a couple of years passed, I found myself missing school. Even so, I was not sure I really wanted to go back to school and had stopped thinking about a future career in research.

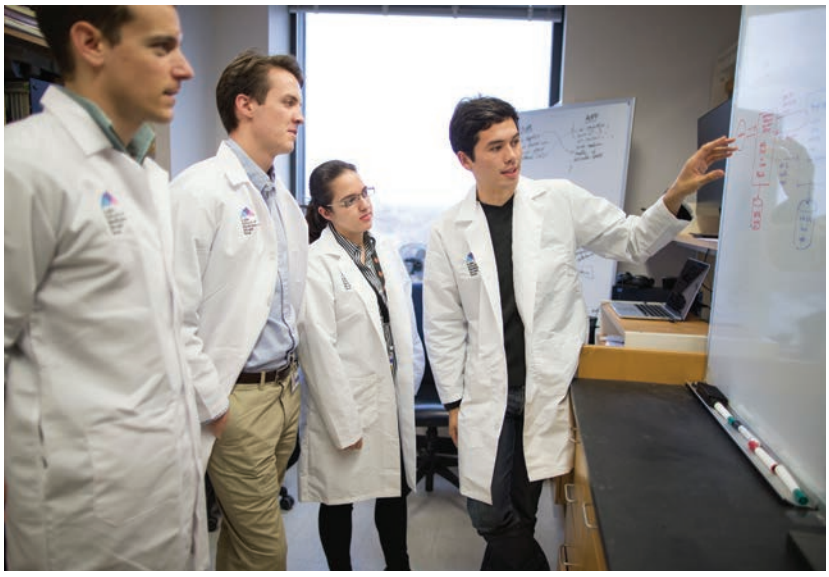
Coursera

It was around this time that I discovered Coursera, an online platform of classes taught by faculty from some of the best universities in the world. Coursera is open to anyone with an internet connection and a desire to learn. It was the perfect outlet for me. I could choose subjects to sample and get my feet wet without anyone knowing, all with the benefit of not having to pay for credits or be in a formal academic setting. It was incredibly freeing. I started taking classes in everything, including things that I, a molecular-biology-loving biologist, had never thought to try.

That is how I stumbled across the field of systems biology. Deemed a specialization by Coursera, there were six courses designed to introduce the topic. These courses were offered by the Icahn School of Medicine at Mount Sinai. I signed up, but my attitude was noncommittal. "Eh," I thought, "why not? I can stop at any time."

Who knew I wouldn't be able to stop? The more math and computation — two subjects I'd never really studied — the courses required, the more I wanted to know and do really well.

One of the courses assigned crowdsourcing challenges. I may have gotten a tad bit competitive with those — oldest child syndrome I suppose. The challenges involved combing through the Gene Expression Omnibus, or GEO, and, for example, identifying



Monteiro with her lab mates at the Ma'ayan lab at Mount Sinai in New York City. Professor Ma'ayan asked Monteiro to move from Brazil and join his lab after she took a Coursera course with him.

studies that perturbed a single gene in a mammalian system. I then had to identify the controls and condition samples and then extract the differential expression signatures from each of these studies using the web application GEO2Enrichr. The goal of it all was to build a high-quality gene set library.

As with everything in life, the more you do it, the easier it is. There were three challenges for that class, and I won all of them. It was good fun, and as far as I knew, that was all it was ever going to be. Then one day I received an email from the course's professor asking me to help him with work for another similar project. We began communicating, and I told the professor about my newfound love for systems biology and how I actually was thinking of going back to school and applying to systems biology programs. I, the wet lab enthusiast, was happily spending more and more time in front of a computer.

One day, after several weeks of progress with the project, the professor emailed to ask me whether I would consider working in his lab at Mount Sinai in New York City for a few years before applying to Ph.D. programs.

I read and re-read his email and even asked my brother to read it too. I had clearly forgotten the meaning of English words! Could the professor really be asking what I thought he was asking?

New York

Indeed he was, and I'm now in New York City working at Avi Ma'ayan's lab at Mount Sinai. It has been a surreal experience, and I find myself existing in a big blur of excitement that at times still scares me. But I'm truly glad I've done it and grateful to my kind colleagues and Professor Ma'ayan.

I understand now that I had constructed an ideal in my mind of who I was and what I could handle, and when that was shattered I was lost. It was by taking those online classes and allowing myself to try something completely new — something that didn't fit exactly into my life plan — that I finally understood that, whether we are undergoing some big changes or staying perfectly still, life gives us the chance to learn something new every day.

Caroline Monteiro (caroline.monteiro@mssm.edu) is a bioinformatician in the lab of Avi Ma'ayan at the Icahn School of Medicine at Mount Sinai.

Hey ladies, where you at?

By Jennifer Ross

The following essay is excerpted from the blog Woman of Science, written by biophysicist Jennifer Ross. It has been edited to meet ASBMB Today's style requirements.

So I just went to a big nerd conference this week. You know the one. No, not that one — I went to the other one.

I had a lot of fun talking with everyone and doing some service committee work. I met new people and connected with friends. I did a lot of mentoring. It was pretty exhausting, even though I only went for three days. And I noticed something. There were many times when I was the only one. Don't make me say it. I was the only woman in a big group of men. It wasn't all the time or every dinner, but it was noticeable and fairly often.

It got me to thinking. Where were the women? I talked about it with my husband, and we realized there were women at the meeting, but there were almost no women at my stage — the post-tenure, associate professor stage. There were lots of young women — grad students and postdocs and even undergrads! There were lots of post-tenure, associate-level men, but there were only a small handful (a couple) of associate women. There were also about two to three full professor women floating around visibly. We brainstormed about women who were missing to figure out where they were. For each one, I realized they had told me in the past that they were going to "not travel so much" after tenure. Or, in some cases, they had a baby after tenure and couldn't travel. And now they are MIA.

At dinner, I was sitting with two pre-tenure women, and I asked one of them if she would continue to travel when she (inevitably) got tenure. Her

Ross addresses the relative absence of post-tenure femaile faculty at scientific conferences.

OK, you don't think of yourself as a role model, and you didn't have a lot of women role models? Fine, but don't you think there should be more women at the big conference who are successful, normal middle-aged people who can relate to lots of different kinds of women? Because the way this conference looked, if you were a woman, you were either pre-tenure or you had very limited options.

answer was a clear no. And the reasons were multifaceted. She said she would probably travel if she were invited to speak. But she didn't want to go to as many big conferences. She has two kids at home under the age of 5, and she wanted to spend more time with them. She felt that she was a better mom when she was with them. She herself had a stay-at-home mom, and she felt a lot of guilt from being away. She also had a two-body problem, and her husband was going on the market. These are all really good reasons, but it just made me sad because it was another woman pledging to become less visible after getting tenure. Sometimes I think that it isn't that women aren't in science — it's just that no one knows we're here because we are diligently doing the science and not out there selling our science the way many men do (I realize #NotAllMen).

So I am writing this to plead with the women for them to come back to big conferences. Please. See, I asked nicely.

Here are three good reasons to return.

Do it for you

Attending, networking and participating at big conferences is important for maintaining your enthusiasm, your creativity and your visibility in the field. At the big conference this week, I saw two talks: my student's talk and part of the one before my student. They were great. I wish I could have seen more, but I was too busy networking (schmoozing) and talking science in the hallway outside of the rooms. I had several meetings with collaborators, established some new collaborations, and met a ton of students and mentored them (see below). These connections with students who will ultimately join our ranks are just as important as the ones with the older fellows. The reinforced connections with my peers and near peers lead to continued invitations to conferences, seminars, colloquia and nominations for awards. And even though I didn't see many talks, I did learn a lot of science from the network. I learned what people are excited about and interested in. I talked to a program officer or two about big new ideas from my group. I was just obviously and actively engaged in the meeting.

Do it for me

OK, that first one wasn't convincing, because you are a selfless, government-financed servant who works on science for the thrill of discovery, and you don't give a f--- what anyone else thinks now that you have tenure. I hear you. Well, how about you come back to big conferences for me? You are probably thinking that I don't need you. But I do. I need you so that I am not the only woman at dinner or after-dinner drinks three out of four nights of the conference. I need you because I can't talk to all the students myself and be the representative for my entire gender at this age and stage of career. It's a lot of pressure. I have never been so sought after for my advice. In fact, similar situations are what drove me to start my blog in the first place. And most of them didn't even know I have a blog full of advice on exactly what they were asking me!

Do it for them

And this brings me to my final reason, which piggy-backs on the last one. I shouldn't be the representative woman. There should be other examples of women who do it differently. I am navigating my career in a particular way that works for me, but I am a big-mouth feminazi. The students need examples of other types of women. What about if you are shy? What if you do theory? What if you are pregnant? What if you are conservative? What if you don't want kids? What if you are gay? I'm not many of these things. We need lots of women to go to conferences who have navigated tenure successfully and are still visible to serve as role models. OK, you don't think of yourself as a role model, and you didn't have a lot of women role models? Fine, but don't you think there should be more women at the big conference who are successful, normal middle-aged people who can relate to lots of different kinds of women? Because the way this conference looked, if you were a woman, you were either pre-tenure or you had very limited options.

So that is why I think it is important for women to continue to attend big conferences in their broad field. Your impact can be in just being yourself and reminding everyone you exist. What do you think? Will you continue to go to the big conference? Will you come back? I hope so. It is very lonely for me without you.

Jennifer Ross (rossj@physics.umass.edu) is a professor of hard science at a research-intensive university. She blogs at https://womanofscience.com.

A class on career options

By Bree Yanagisawa

Research funding crunches and a glut of young Ph.D. scientists battling for academic research jobs have forced many in the life sciences to re-evaluate their ideas of what constitutes a typical science career. Fortunately, Ph.D.-level scientists have been applying their skills to a multitude of nonacademic science-related jobs for years. Nancy Manley, director of the Integrative Life Sciences program at the University of Georgia, long has been aware of the plethora of job options for her Ph.D. students. But she says resources to prepare them for nonacademic careers have been scarce. Ten years ago, to give her students a chance to investigate their options, Manley began teaching a seminar course called GENE 8200: Careers for Ph.D.s in Life Sciences.

Bree Yanagisawa, a science writing intern for the American Society of Biochemistry and Molecular Biology, spoke with Manley about the importance of the course and its content. The conversation has been edited for length and clarity.

What's your scientific background?

I'm a distinguished research professor at the University of Georgia in the department of genetics. I have been here for 14 years now. I'm a typical faculty member who had what would be considered a typical academic career track. I went to graduate school, went to a postdoc, got an assistant professor position and then got promoted.

Why did you decide to start the course?

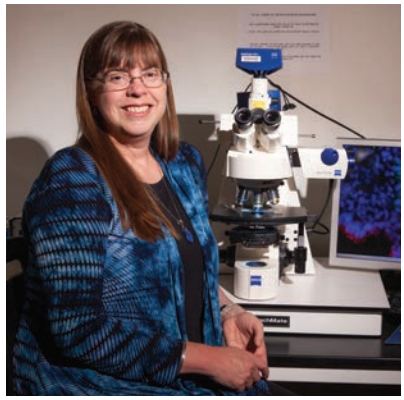
I've been teaching and mentoring graduate students and postdocs in my own lab for my whole career, but I was always mystified by faculty who told students that the best students should go into academia and everybody else should consider something else. It was like somehow all those other jobs were consolation prizes for those who couldn't cut it in academia. I never felt that was true.

I was asked to teach a seminar class and told I could teach whatever topic I wanted. I knew that students needed more information about other careers. So I decided to try it one year — to use a graduate seminar as an opportunity to have a discussion about career options.

What is the course content and how is the course run?

The specific career topics the course covers change every year. We start the first meeting talking about careers. What is a career track? What kinds of jobs are available to people who have Ph.D.s in life sciences? I usually divide the options up into academia, government and private sector, and within each of those we talk through multiple possibilities.

We generate a long list of possible careers that students could have. I have the students pick a career or maybe a couple of closely related careers. Each of the students then goes out and is responsible for getting information about what the career is, identifying people who have that career who have a life sciences Ph.D. and interviewing them. The students have to OK their question list with me, and then they contact the people and set up the interviews. The rest of the class is the students giving presentations on their findings to everybody in the class.



ROBERT NEWCOMB, UNIVERSITY OF GEORGIA PHOTOGRAPHIC SERVICES

Nancy Manley teaches a popular graduate seminar course about life science careers.

Are there any careers that are favorites each year?

There are some that we do every year, for sure. We always do patent law. We always have somebody who wants to do big pharma and large biotech companies. We always have somebody who does small biotech companies. So the standard things that you would think of as the primary careers outside of academia. We usually have somebody who does nonprofit organizations of one kind or another. We will often, though not always, have somebody who does science writing as a topic also.

I also usually (do a separate lecture) about academic careers. I think one of the problems we have even within academic careers is that students really don't know what they are like. Students have a general idea, but they don't know what faculty do with their

time. They don't know very much about the nuts and bolts of running a lab. One of the things I tell them is: You may think that academia is not for you, but what is that based on? Do you even know what it is that you're deciding you don't want to do?

Would you recommend all students take your career exploration course regardless of career aspirations?

Absolutely. The purpose of the class is to have students make conscious choices about their career decisions. But in order to make a conscious choice, you have to have information. Many students have never been full time in the workforce. There are really big differences in the kinds of benefits you get if you work in government versus the private sector versus being self-employed. The course is not just about the day-to-day content of a career but also about the nuts and bolts of making career decisions.

What is the biggest misconception students have when they come into the course?

That academic jobs are the hardest jobs and that other jobs are easier and pay more.

The real answer is that all careers have advantages and disadvantages and that pay is not always based on how hard you work, but it often is. If you want a job that isn't very difficult but pays you lots of money, it doesn't exist. Any career that you want to have that's going to be personally rewarding and financially lucrative is going to require you to work really hard.

It's not a matter of finding the easy way out. It's a matter of finding the job that is the best fit for what is important to you.

What's one piece of advice you hope your students take from the course?

At the end of the class we always do a sum-up discussion and talk about (the common elements of) the advice given by all of the people. They are: be successful in graduate school, and network, and go out there and do the best at what you're doing now, because the experience of graduate school trains you for all of these other careers. That's the reason why people who have this degree can have all these diverse careers. The degree itself is valuable.

Bree Yanagisawa (breannwoelfel@gmail.com) was an intern at ASBMB Today when she wrote this story. She is currently a Ph.D. candidate in pathobiology at the Johns Hopkins School of Medicine. Follow her on Twitter at twitter.com/BreeTalksSci.

Students reflect on Manley's course

VANDRISSE

I remember in one class Nancy told us not to confuse uncertainty with (lack of) interest, which allowed me to reflect on the true reasons why I was confused about my future career path. It turns out, I knew what I wanted the whole time. I just needed someone to help me gain confidence in my decision.

- Chelsey VanDrisse

XU

After I explored several possibilities (biotech industry and consulting) according to our discussion in the class, I decided to continue my career in academia more consciously. Now I am a postdoc (at) Memorial Sloan Kettering Cancer Center, and I enjoy what I am doing, because I know this is the area (in which) I fit very well. Thanks to the-career development class, I became very dedicated to what I am doing now.

- Jianing Xu

AVERY

Most graduate programs I am aware of groom you for postdoctoral fellowships and a life of academia. As a consequence, students and postdocs alike have a tendency to pigeonhole themselves into this niche. Limiting ourselves in this way is foolish. The key fact I took from Manley's seminar is that as scientists we have tangible and transferable skills that virtually any employer in any field is looking for.

- Jay Avery

RHAESA

I now feel confident conducting an informational interview to see if I'm interested in a career. This skill alone has made this class well worth my time and will be so valuable to my future job search. Taking this class has shown me more options for careers than I realized existed and has given me the tools to get onto my career path of interest.

- Amanda Rhaesa

Communications training near you

Expanding the reach of 'The Art of Science Communication'

By Geoff Hunt

Effective communication is critical for effective outreach. That is why communication training has been a central effort of the American Society for Biochemistry and Molecular Biology's Public Outreach Committee since its inception. Through our recently developed online course, "The Art of Science Communication," we have begun providing participants with fundamental training for presenting science to nonexpert audiences in formal settings. Now that the course has been running for several months, the committee is looking for ways to expand participation.

One way we hope to extend the course's reach is by partnering with external organizations — including universities, professional societies and private companies — that will then provide their members access to the content. We believe that increasing the number of course participants in this way will lead to a stronger, more effective community of science communicators. We are also looking for ways to recruit additional instructors who are associated with these organizations and can help lead the virtual course.

This expanded training need not occur only online. We also have begun running blended versions of "The Art of Science Communication" in which participants familiarize themselves with the course content online before meeting on a campus for facilitated class discussions. Hudson Freeze, who is on our outreach committee, led this type of blended course at his home institution, Sanford Burnham Prebys Medical Discovery Institute in La Jolla, Calif., in the beginning of 2016. "I like the blended course because all of us make a production together," says Freeze. "We get to know each other and watch personalities emerge as the students tell their own personal scientific stories." Freeze says he would "encourage anyone to feel the exhilaration and satisfaction of developing their own story in a setting like this."

The committee also is developing a series of in-person instructional workshops that are separate from but complement what takes place in the online course. Two such workshops were piloted at the 2016 Experimental Biology meeting. One version, led by committee members Susanna Greer of the American Cancer Society and Tom Baldwin of the University of California, Riverside, introduced attendees to the skill of giving an elevator pitch. Attendees learned how to describe their research pithily and had a chance to practice their pitches in front of peers. One attendee described the workshop's impact by saying, "I can better articulate my research to others, and I have learned that who I am is very important to communicate to others."

The other workshop, led by me and staff member Rajendrani Mukhopadhyay, focused on storytelling. Using examples from cinema, children's books, scientific papers and the hit Broadway musical "Hamilton," we organizers showed how narrative themes such as plot, character, tension and emotion can be applied to science

communication. Attendees incorporated these lessons into their own science stories, which they presented to the rest of the group in a great variety of styles including rhymes, poetry and even children's stories.

The high turnout and positive feedback from these pilot workshop attendees have encouraged the committee to bring live trainings to additional meetings, events and institutions throughout the scientific community. We will host workshops at career symposia at the University of Kansas Medical Center on July 28 and the University of Texas on Sept. 10 as well as during the society's Special Symposium on Transcriptional Regulation, to be held Oct. 6 through Oct. 10 in Snowbird, Utah. We also have been invited to present our workshop at the annual meeting of the American Society of Plant Biologists in July and intend to present at the Annual Biomedical Research Conference for Minority Students in Nov. 2016.

To help support these future workshops, the society has applied for funding from the National Institutes of Health's Innovative Programs to Enhance Research Training program. We also are developing additional workshops and online training modules based on different communication themes and designed to resonate with various audiences. For example, the outreach committee is collaborating with the ASBMB Public Affairs Advisory Committee to develop training related to communicating with policymakers.

Want to know how your organization can partner with the ASBMB to bring our training to your colleagues and members? Interested in having us bring our training program to your town or institution? Please contact us at outreach@asbmb.org.

Geoff Hunt (ghunt@asbmb.org) is the ASBMB's Manager of Public Outreach. Follow him on Twitter at www.twitter.com/thegeoffhunt.

Re: President's Message, May issue

Nothing new here. Maxwell's equations of electromagnetism didn't hit the big time overnight. Having said that, a short while ago a paper showing specific molecular stoichiometry (and challenging prior dogma) was published in the Journal of Biological Chemistry and received little attention. A year later a similar paper on another member of the same family of proteins reiterated the JBC observations. Here's the clincher: the second paper was in Cell and did not even refer to the earlier JBC work. The authors were either negligent or dishonest to claim novelty. The reviewers were clearly negligent. The senior author in the Cell paper is a superstar. The JBC author was told by the National Institutes of Health reviewers that he didn't publish in "high profile" journals, and he lost his funding. So, among other things, journal impact factors clearly matter.

– Little Shot

I fully concur with Steven McKnight's analysis on the untoward effect of journal impact factor, especially for our younger colleagues. The amount of time wasted on trying to send a particular work to some very high-impact-factor journal is beyond crazy. If one is going to use citation analysis for a given paper, the only thing that counts is not where the paper was published but how many times it was cited. If you take a look at your plot of citations versus year for the Konopka & Benzer paper, it seems to me that the number of citations for the first 20 years (up to 1991) is well over 100, which already make it a very high impact paper (never mind the mega-citations even further on). Even for the first 10 years up to 1981, I would estimate that the paper had reached either over or close to 50 citations, which again is really very good.

– Marius Clore

I agree with both Steven and Marius. Long ago, when I was chasing my first academic job and eventully tenure, all that was expected was that I publish solid papers in strong peer-reviewed journals such as JBC, the Journal of Molecular Biology and Biochemistry. This is still pretty much the policy of the University of California. Whatever "fame" I have achieved has come from being in science for the long term and working on basic problems that I was curious about. As for the Konopka/Benzer paper, a long time was required for the embedding of my papers in the literature. (I'm still waiting for some of them to embed!) I'm disgusted by journal publishers chasing the almighty impact factor. If publishers were to ignore it altogether, science would still prosper. And I think it would be more fun.

– Stephen White

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B1 D-Glucose-6-Phosphate	B2 α-D-Glucose-1-Phosphate	B3 L-Glucose	B4 α-D-Glucose	B5 α-D-Glucose	B6 α-D-Glucose	B7 3-O-Methyl-D-Glucose	B8 α-Methyl-D-Glucoside	B9 β-Methyl-D-Glucoside	B10 D-Salicin	B11 D-Sorbitol	B12 N-Acetyl-D-Glucosamine
C1 D-Glucosaminic Acid	C2 D-Glucuronic Acid	C3 Chondroitin-6-Sulfate	C4 Mannan	C5 D-Mannose	C6 α-Methyl-D-Mannoside	C7 D-Mannitol	C8 N-Acetyl-β-D-Mannosamine	C9 D-Melezitose	C10 Sucrose	C11 Palatinose	C12 D-Turanose
D1 D-Tagatose	D2 L-Sorbose	D3 L-Rhamnose	D4 L-Fucose	D5 D-Fucose	D6 D-Fructose-6-Phosphate	D7 D-Fructose	D8 Stachyose	D9 D-Raffinose	D10 D-Lactitol	D11 Lactulose	D12 α-D-Lactose
E1 Melibionic Acid	E2 D-Melibiose	E3 D-Galactose	E4 α-Meth Glactos								
F1 Adonitol	F2 L- Arabinose	F3 D-Arabinose	F4 β-Meth Xylopyra								
G1 Tricarballylic Acid	G2 D,L-Lactic Acid	G3 Methyl D-lactate	G4 Methyl pyruvat								
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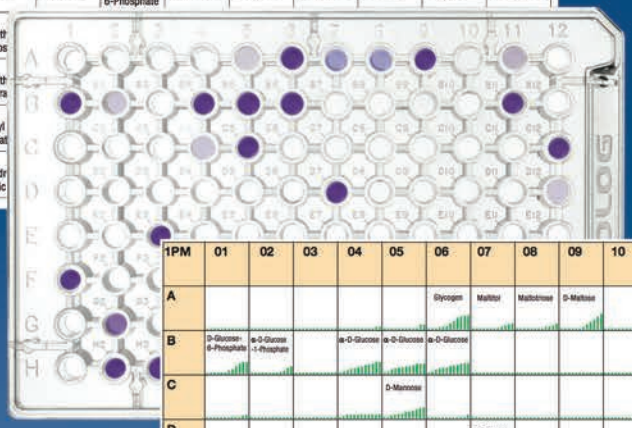

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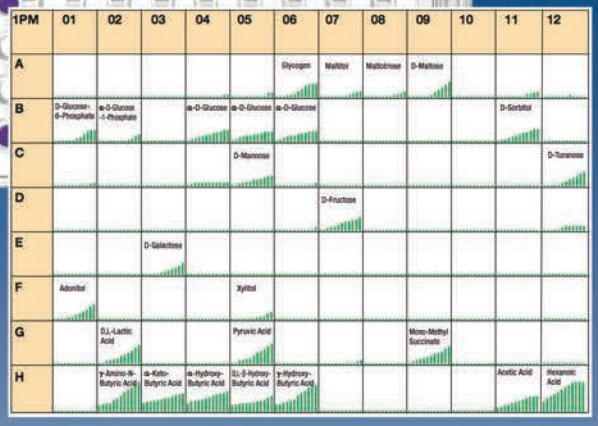

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