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ASBMB TODAY

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

AFTER MARIA, PUERTO RICO RISES

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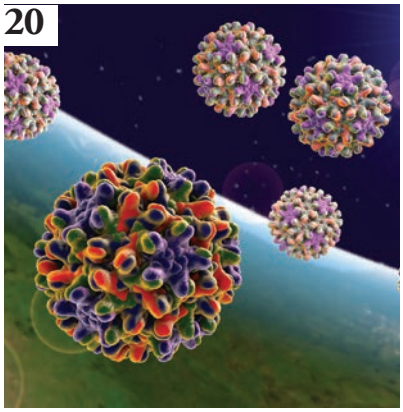

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ASBMB TODAY
THE MEMBER MAGAZINE OF THE AMERICAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY

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2018: You say you want a resolution?

By Comfort Dorn

Don't you just love making New Year's resolutions?

Me neither.

Mine always relate to getting more of something (money) or less of something (weight), and whatever the goal, it's always out of reach. No wonder we all hate February – it's more than the cold that's bringing us down. It's the iron weight of failure.

I've learned that I'm happier when I set a goal that I know I'll meet, so the year I had a contract to sell my house, my resolution was "Move out." Nailed it.

At a website called (I kid you not) happynewyear-2018.org, you can find a slew of suggested resolutions, everything from "Start a meditation practice" and "Learn a new language" to "Forget the past" and "Enjoy the little things." My favorites are "Dress more like my style" and "Tame your monkey mind." I had to look that last one up.

By the time you read this, it will probably be too late to redirect you for the moment the ball drops in Times Square, but put this suggestion under your hat for future reference: Set a reasonable goal for something that will benefit others, as well as yourself. Or as my mom always said, "Stop thinking about yourself so much."

And it's true. All this resolution-making can get a little narcissistic. I just finished reading John Arnst's story about scientists and students in Puerto Rico coping after Hurricane Maria (see page 24). These folks have lived without electricity, telephones and drinking water, but they still manage to focus on their research. Kind of makes a goal like "Watch less TV" seem pretty small.

So, by all means, think about helping someone else. Me, for example. (Send donations to Puerto Rico first, of course; then help me.)

I'm picturing a sort-of reciprocal resolution arrangement here. You resolve to send us your thoughts about this magazine — what you like, what could be better, what you don't see that you want to see — and we'll resolve to do our best to bring you what you want.

For example, many of you seem to want more career advice, but what exactly would be most helpful? Whose perspectives do you want to read? What science should we be writing about? Are we missing trends in academia or industry? The American Society for Biochemistry and Molecular Biology has a broad membership, and that doesn't even include all our wonderful online readers. We may not be able to give you everything you want, but we may be able to give you more of what you want.

So feel free to drop me a line and let me know how we can do better. Once we have your feedback, we can focus like a laser on making it happen. And I won't have to worry about my untamed monkey mind.

Comfort Dorn (cdorn@asbmb.org) is managing editor of ASBMB Today. Follow her on Twitter @cdorn56.

Planning for an active 2018

By Benjamin Corb

As 2017 turns to 2018, the Public Affairs Advisory Committee and your public affairs team look forward to an exciting year with new opportunities and programs to enhance the American Society for Biochemistry and Molecular Biology's advocacy and science policy efforts.

The public affairs staff in November launched "Pipettes and Politics," a science policy podcast that you can listen to on our blog or download from iTunes and Stitcher. In addition to our articles and blog posts, the podcast brings you inside our team conversations and strategy sessions as we discuss the federal policies that impact science. We'll have a new episode every two weeks, and we invite ASBMB members (and nonmembers) to give the podcast a listen and let us know what topics you'd like to hear us discuss.

The PAAC will launch an advocacy externship program in 2018, offering training and leadership opportunities for ASBMB members who are looking to get deeply involved in science policy and science advocacy. The program, now being developed by the PAAC's Grassroots Advocacy Working Group, will focus on monthly regional advocacy activities and will help strengthen the ASBMB's advocacy efforts in all 50 states. We expect to debut the program in the spring.

We aren't just doing new things though. We're also building on our progress in 2017, starting with working with the National Institutes of Health and sharing opinions on the NIH's Next Generation Researcher Initiative. The NIH introduced the NGRI back in August, and the PAAC's initial comments provided suggestions on what policies truly can benefit young investigators. The PAAC has been developing NGRI policy recommendations, which will be shared with NIH leadership throughout 2018.

The committee is working to make its legislative positions clear and easily accessible to policymakers and ASBMB members. The staff is beginning to develop an online library of ASBMB policy positions on topics ranging from disease-specific research funding to the use of embryonic stem cells in research. We plan to publish this library by the summer. We remain committed to advocating for robust funding for science and watching for legislation that will help to create a fertile environment for biochemistry and molecular biology research.

And that's not all. The committee continues to improve relations with and advocacy for the National Science Foundation, and we are expanding our efforts to work with other funding agencies, such as the Department of Energy's Office of Science. We are also reviewing alternative research funding models and working to better understand the role philanthropy plays in funding basic science. And we're developing webinars with policymakers and thought leaders in the life sciences community during which members will be able to participate and ask questions. Updates on these and other exciting activities will be shared in this space as well as on our blog at policy.asbmb.org.

We look forward to the opportunities that 2018 will provide.

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

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**.

Kenneth E. Neet (1936 – 2017)

By Marc J. Glucksman

Ken Neet was an extraordinary human being, a consummate academician and a professional citizen. He will be remembered as an accomplished scientist with imagination and energy, as an exceptional leader who performed selfless service for our academic and scientific community at large and as a talented teacher who inspired wonder and passion.

Ken was born Sept. 24, 1936, in St. Petersburg, Florida. He rose from humble roots to graduate from St. Petersburg Junior College and subsequently received a Bachelor of Science in chemistry (1958) and a Master of Science (1960) from the University of Florida. He served as a biochemist with the Navy in the Physical Biochemistry Division at the Naval Medical Research Institute in Bethesda and earned a Ph.D. in biochemistry (1965) from the University of Florida. There, Ken worked under the direction of the renowned protein biochemist Frank W. Putnam, seeking to understand protein folding and stability by elucidating the biophysical pathway of protein denaturation. Ken then completed a postdoctorate at the University of California, Berkeley (1965-67) with the legendary Daniel Koshland. Always ahead of his time, Ken created a "chemical mutation" (his words) by converting an essential active site residue of an enzyme from serine to cysteine. This innovative research long preceded the advent of genetic engineering in later decades.

COURTESY OF KYOUNG JOON OH

Ken Neet and his wife, Jane, pictured at his retirement in 2014 from the Chicago Medical School at Rosalind Franklin University of Medicine and Science.

After his postdoctorate, Ken joined the faculty at Case Western Reserve University, where he rose through the ranks to become a full professor by 1978. Ken studied protein conformational changes related to enzyme activity and became a world-renowned expert in this field. He authored a review in Annual Review of Biochemistry titled "The catalytic and regulatory properties of enzymes," a paper cited almost 700 times.

Ken was a firm believer in keeping yourself renewed by sabbaticals. In 1980, he sojourned at Stanford as a visiting scientist. His work with Eric Shooter

In the mid-1980s I walked into a room to serve a term on a National Institutes of Health study section, somewhat intimidated by the fact that I vaguely knew only a few people in the room. Ken Neet immediately walked up to me with a broad smile and a handshake. We chatted for a few minutes, and then he introduced me to a number of those present. I was immediately put at ease.

Six or seven years later, I, along with everyone around me, benefitted from those qualities of friendliness, kindness and thoughtfulness when he accepted the position of chair of the department of biochemistry and molecular biology at Chicago Medical School. Manifesting those qualities along with a keen intellect, Ken established himself as a much-admired teacher, a respected department leader and university administrator, and a valued participant in the national biochemical community, all while maintaining a highly productive and funded research group.

I also have fond memories of Ken from our many years of enjoyable, although usually poorly executed, rounds of golf, followed by always well-executed rounds at the 19th hole.

— Bob Kemp

resulted in a shift in his research when he applied his expertise in protein chemistry and stability to the study of nerve growth factor, a key molecule involved in neuronal growth as well as a target for ameliorating neurodegenerative disease. Ken's research focused on understanding not only structure–function correlates but also a possible treatment rationale for Alzheimer's disease. A natural outgrowth of these studies was a renewed examination of the regulation of nerve growth receptor heteromers.

In 1990, Ken moved to the shores of another of the Great Lakes, to the Chicago Medical School at Rosalind Franklin University of Medicine and Science, where he chaired the department of biochemistry and molecular biology from 1990 to 2005. At Rosalind Franklin (formerly the Finch University of Health Sciences), he was recognized for initiating a program with an academic focus on protein structure. Ken's talent for recruiting faculty with expertise in cutting-edge technologies ushered in a new era. He encouraged use of X-ray diffraction, enrolling the university in subscriptions to the then-new beamlines at Argonne National Laboratory and establishing proteomics and a panoply of spectroscopy within the department. His own research included extramural funding spanning from 1969 to 2009 that yielded some $7 million in grants ($20 million in today's dollars). He published 107 peer-reviewed papers and 20 book chapters/reviews. His most recent work evolved from his mentoring and collaboration with junior faculty and was published in Scientific Reports the week of his passing. Ken achieved emeritus professor status in mid-2014, but he maintained an office, attended seminars and continued as associate dean of research for another year. He demonstrated that he was a prolific thinker and active contributor to the university and department.

Ken also had a worldwide reputation for his breadth of knowledge, as reflected by the professional services completed throughout his career. He was a founding member of the Protein Data Bank Advisory Group and was on the editorial boards of Protein Science, Molecular & Cellular Proteomics and, most notably, the Journal of Biological Chemistry, where he was senior associate editor for 17 years. All this attests to his recognition and stature as an expert. His service for the American Society for Biochemistry and Molecular Biology included being treasurer and council member from 2000 to 2006 and Finance Committee member from 2009 to 2015. He was secretary for three years of the Association of

It was with great sadness that I learned of Ken's passing. Not only was he a dear friend, but he was also something of a kindred spirit. Our careers intertwined at many places; we were both treasurers and long-term members of the American Society for Biochemistry and Molecular Biology Finance Committee, both associate editors of the Journal of Biological Chemistry, both departmental chairs and, perhaps most importantly, both aficionados of protein chemistry, with particularly reference to nerve growth factor and its role in biology and neuroscience (on which we published a paper together in the JBC). This resulted in many opportunities to interact, including some lovely dinners (especially with our wives, Jane and Penny), and to discuss science, publishing and the fascinating topic of departmental politics.

Although not normally loquacious, Ken had a broad range of knowledge and a marvelous insight into everything from protein structure to human nature, and he was thoughtful in expressing his opinions and judgments. I always paid attention when Ken had something to say. Indeed, when the society launched Molecular & Cellular Proteomics in 2001, I recruited Ken and Tom Vanaman (both JBC associate editors at the time) to the new editorial board to give it the benefit of their deep experience and extensive wisdom as preeminent protein chemists. I knew they would be invaluable in building credibility early, and indeed they were.

Ken was a protein physical biochemist of the old school — he knew all the ins and outs of the complex arsenal of biophysical techniques — but he was also au courant with the rapidly developing methodologies and applications of molecular and cellular biology, and his research, particularly in the later years, was very much directed to biological questions. Thus, I always looked forward to our discussions of the latest findings in the world of NGF and to our friendly arguments as we dissected our separate views (some in accord and others not) about our favorite molecule. He invariably had ideas and explanations that I had not thought of, and I never finished a conversation with him without feeling that I had learned something new.

Simply put, Ken was a delightful person — unassuming, generous and kind. He was the type of person that when you called upon him for a favor, he would always agree and would do his best to help with whatever issue you were struggling with. He was a gentleman, a scholar and a scientist of the finest caliber. There is no question that he was a leader and a mainstay at Rosalind Franklin Medical School and they will miss him very much, as will his many friends, colleagues and family — and as will I.

— Ralph A. Bradshaw

EMILY HUFF/ASBMB

Ken Neet, right, talks to Stephen Miller, chief financial officer of the American Society for Biochemistry and Molecular Biology, at a meeting in 2013.

Medical and Graduate Departments of Biochemistry, an organization of academic department chairs. His commitment to the research community was exhibited by his participation in numerous study section panels, including four at the National Institutes of Health, three at the National Science Foundation and one at the Office of Naval Research as well as his 15-year tenure reviewing grants for the Alzheimer's Association.

In addition to serving as a department chair at the Chicago Medical School, Ken was the associate dean for research from 2004 to 2015. One of his major initiatives was tripling the number of relevant research experiences for our medical students, a crucial value-added factor for successful residency applications. As a concerned local resident, he was recognized in 1995 by the Illinois State Board of Education with the Those Who Excel Award for service

I met Ken Neet in 1997 by phone as I was running out of my office to give a medical pharmacology lecture at the University of South Alabama College of Medicine where I was then a professor. I had responded to a job ad, and this was a "let's see if there's some interest" call. From the outset, I sensed in Ken many of the traits that I admire in a mentor and to which I would aspire.

On my second interview for a professorship in his department at the Chicago Medical School, Ken held my three-month-old daughter gently, but confidently, as he led my wife and me on a tour of potential lab spaces. Soon after I signed a contract to join the faculty, I ordered a -80 freezer, and Ken volunteered to set it up so I could avoid another long-distance trip. I later learned that Ken always said the way he knew he had hooked a new faculty member was not when they signed the contract, but when they shipped their -80 samples — that indicated they were really coming.

Ken was a valued mentor, colleague and friend through the rest of my career. When it became apparent, to both of our surprise, that I had talent and interest in science administration, he helped me develop in that area without sacrificing my research program. In 2002, Ken made me his vice chair and then allowed me to observe and to participate in some of the difficult decisions regarding allocation of department resources, etc. In 2005, he put my name forward as his successor as chair of the department of biochemistry, an action subsequently endorsed by the school and the university. In January 2006, I followed in Ken's footsteps as chair. Because of his mentorship, I was familiar with the fine balance of time allocation among teaching, research, grant writing and administration. In subsequent years, Ken provided much advice, especially during the financial crisis of 2008-2009 when difficult resource decisions needed to be made.

When my career trajectory was intercepted by the medical school's need for a vice dean for research (2010) and then the university's for a vice president for research (2011), the latter necessitating that I step down as chair, Ken continued to provide the experience-based humor, data-driven wisdom and unencumbered advice that influenced my deliberations.

Ken vicariously experienced the growth, achievements and tribulations of my children over the past 20 years with his avid attention to stories about them. He exhibited his genuine interest in their lives with his unsolicited and frequent queries.

In the course of my career, I have had the good fortune to come across several individuals who shaped my path. Ken was one of them. I am proud to have called him my friend, colleague and mentor. I will miss him dearly.

— Ronald S. Kaplan

to a local school district with a large population underserved in science and medicine, predating what we now know as STEM (science, technology, engineering and mathematics). Ken also believed in developing talent. A former postdoc and two faculty members he hired at Chicago Medical School became department chairs themselves.

As a teacher, Ken Neet was legendary. Just prior to his retirement, he was the most prolific teacher in our department with the largest number of hours. He taught in every medical and graduate school course (n = 7) offered through our department. He directed medical genetics for 10 years and was course director of physical biochemistry for 12 years. He was revered by both students and postdocs under his tutelage. The outpouring of grief and stories shared upon learning of his demise is remarkable in the communal response by all generations of scientists.

Ken embodied the ideals of scientific and professional leadership, service and teaching. We all miss him tremendously and attempt to model what he represented for us as an exemplar of respect, scientific integrity and exceptional leadership.

Ken died April 12. He was a modest and private person who enjoyed his family; their pictures adorned his office along with mementos of his scientific accomplishments and honors. He is survived by his wife of 57 years, Jane, and four children, Kerrie, Kellie, Kirk and Kyle.

(The author thanks the people at Rosalind Franklin University of Medicine and Science who offered up factoids of Ken Neet's life and career.)

Marc J. Glucksman (marc.glucksman@rosalindfranklin.edu) is professor and chair of the department of biochemistry and molecular biology, director of the Center for Proteomics and Molecular Therapeutics and director of the Midwest Proteome Center at Rosalind Franklin University of Medicine and Science/Chicago Medical School.

Ken E. Neet was my chair for 16 years and my friend for 27 years. I was proud to be a member of his department as he made significant contributions to science and was recognized among the scientific community in many ways. As chair, Ken transformed a broad-based department into one focused on protein structure and function, with an emphasis on X-ray crystallography. This emphasis continues today. Ken contributed to the education of students, from didactic lectures to medical and graduate students to the teaching of undergraduate through postdoctoral students in his laboratory. I have heard nothing but exceptional praise for Ken as a mentor. Serving on many of his students' committees, I can attest that their educational experience was exceptional. I don't know if all who worked in his lab recognized Ken's accomplishments and history, but now they are part of them.

And Ken was more than that — he was a good person. He was not pretentious, but rather modest, sincere and honest. These attributes made Ken very approachable, and I frequently interacted with him in both professional and social settings. We often talked about new hypotheses I had and new directions in my research. We played golf in a quietly competitive way but rather humorously, since neither Ken or I were very good.

Ken's big loves were his family and his science. He lived modestly and chose to spend his money and free time with his family. Ken and Jane gave their four children first names that started with K and middle names that started with E, so his and their initials were all KEN. As for his love for science, Ken once told me that he knew why scientists do not get caught up in sex scandals; scientists are too interested in experiments, he said, and thus distracted from everything else around them. This statement, valid or not, illustrated Ken's sincere love for science. It is impossible to evaluate the impact one person has on another, but I am certain that I am better off because of my relationship with Ken, both in and outside the lab.

— David Mueller

When I was recruited and received several tenured job offers, including one at Chicago Medical School, Ken, playing surrogate real estate agent, drove me around Chicagoland and shared his vision of the department with me, including the usual start-up packages and the creation of the Midwest Proteome Center. As I spoke to one of my mentors, who was familiar with Ken Neet, he remarked that Ken was "about 65" and "you should be certain that he will be around after your hiring." Ken assured me he was not planning to retire soon and ended up on the active faculty until he was almost 80. He was always a man of his word.

In faculty, strategic planning or other meetings, Ken was always the quiet one, taking in the environment and assiduously hearing everyone's points of view. Then he would ask the questions that drove to the core of the discussions that facilitated arrival of the ultimately wisest decisions. This involved his anatomic structure function correlate of why we have two ears and one mouth — so we can listen much more carefully than we speak.

— Marc J. Glucksman

WELCOME, NEW ASBMB MEMBERS

Yamilex Acevedo–Sánchez, University of Puerto Rico, Mayagüéz
Olubisi Adeyi, Federal University of Agriculture, Abeokuta
Ashraf Ahmad
Femi Alakija, Stephen F. Austin State University
Karlbuto Alexandre, Rowan University School of Osteopathic Medicine
Aktan Alpsoy, Purdue University
Abdulraof Alqrache, King Abdulaziz University
Jordan Alvarez, Skidmore College
Yukitomo Arao, National Institutes of Health
Cheryl Arrowsmith, University of Toronto
Allison C. Augustus–Wallace, Louisiana State University Health Sciences Center, New Orleans
Bolatito Ayo–Lawal, Worldwide Commercial Ventures Limited
Ryan Baker, UWorld
Jackson Baumgartner, San Francisco State University
Stephen Bell, Massachusetts Institute of Technology
Morris Birnbaum, Pfizer
Douglas Borchman, University of Louisville
Daniel Bouvard, Centre National de la Recherche Scientifique
Harrison Bracey, Texas State University
Joel Brind, Baruch College, City University of New York
Amanda Casey, University of Texas Southwestern Medical Center
Mingzhu Cai, University of Illinois at Urbana–Champaign
Nicholas **Cairl**, University of Toledo
Brandaise Callahan, University of Alaska Fairbanks
Elaine Camacho–Hernandez, University of Puerto Rico at Ponce
Kelly Chacón, Reed College
Jeannine Chan, Pacific University
Stephanie Chaousis, Griffith University
Kelsey Chen, University of California, Santa Barbara
Keming Chen, Lanzhou General Hospital
Andrew Christofferson, Royal Melbourne Institute of Technology University
Hak Suk Chung, Korea Institute of Science and Technology
Jens Coorssen, Brock University
Zaida Cordero–MacIntyre, Loma Linda University
Garland Crawford, Mercer University
April Darling, University of South Florida
Matthew de Cruz, University of Southern Mississippi
Alejandro De Santiago Perez, University of California, Riverside
Alexandra Deaconescu, Brown University
Magdalena Delgado, University of Arkansas for Medical Sciences
Michael Detweiler, Boise State University
Valentina Di Biase, Medical University of Innsbruck
Jacqueline Drak, Bellevue College
Francesca Duncan, Northwestern University
Steven Eastlack, Louisiana State University Health Sciences Center
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Barnita Haldar, University of South Alabama
Marlin Halim, California State University, East Bay
Shelby Havel, Boise State University
Siran He, Emory University
Samanthi Herath Gedara, BASi
Genaro Hernandez, University of Texas Southwestern Medical Center
Ethan Higginbotham, Marshall University
Maryam Honarbakhsh, Rutgers University
Elham Honarvarfard, Clarkson University
Mandi Hopkins, University of Colorado Anschutz Medical Campus
Ronggui Hu, Shanghai Institute of Biochemistry and Cell Biology
Wei Huang, Southwest University
Ankita Bachhawat Jaykumar, University of Texas Southwestern
Miji Jeon, University of Maryland, Baltimore County
Vikash Jha, University of Western Ontario
Yuxuan Jiang, Western University
William Johnson, Oklahoma State University
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Sobhi Kazmouz, Monmouth College
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Joan Montaner, Vall d'Hebron Institut de Recerca
Pavitra Murali, University of Arkansas for Medical Sciences
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Karthik Nadendla, University of Missouri–Kansas City
Anita Nag, Furman University
Deepika Nambiar, University of Tennessee, Knoxville
Madhavan Narayanan, Mercy College
Matthew Nebiyou, Towson University
Xuan Nguyen, Scuola Internazionale Superiore di Studi Avanzati
Emily Niedziela, Bay Path University
Wilson Nieves, Stony Brook University
Nour Noaman, Western Sydney University
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Nicole Oberlie, Bradley University
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Adua Rahman, Tennessee Technological University
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Savanna Ratky, University of Alaska Fairbanks
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Dormarie Rivera Rodriguez, University of Puerto Rico at Ponce
Rebecca Rodell, Cornell University
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Elodie Roussarie, Centre National de la Recherche Scientifique
Thusitha Rupasinghe, Metabolomics Australia, The University of Melbourne
Brooke Seamans, Rochester Institute of Technology
Mark Shaaya, University of Illinois at Chicago
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Xingyi Shi, Denison University
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Daniela Simcikova, Charles University in Prague
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Sathish Srinivasan, University of Miami Miller School of Medicine
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Thaiesha Wright, Miami University
Bin Xia, Peking University
Guanying Xu, University of Illinois at Urbana–Champaign
Hideaki Yano, National Institute on Drug Abuse
Nikhil Yedulla, Wayne State University
Yuxia Zhang, Kansas University Medical Center
Li Zhou, Abbvie

Member update

By Erik Chaulk

Tauro wins first place for undergrad research

TAURO

Marymount Manhattan College student Tracy Tauro won a first place award at the 20th annual Undergraduate Research Symposium in the Chemical and Biological Sciences at the University of Maryland, Baltimore County. She won first place in biochemistry and molecular biology for her presentation, "In silico exploration of the mechanism of translesion synthesis by DinB."

Tauro, a double major in dance and biomedical sciences and a member of the school's ASBMB student chapter, has been doing research on the protein DinB (short for DNA polymerase IV) under the supervision of Benedetta Sampoli Benitez, a chemistry and biochemistry professor, over the past year.

The competition drew more than 250 students from 45 universities, showcasing novel student research in the chemical and biological sciences.

Tauro will present additional research at the 2018 ASBMB Annual Meeting, held in conjunction with the Experimental Biology conference in April in San Diego.

In memoriam: Milton J. Schlesinger

Milton J. Schlesinger, professor emeritus of molecular microbiology at Washington University School of Medicine in St. Louis, passed away Oct. 27 at the age of 89.

SCHLESINGER

Schlesinger studied physics at Yale University, graduating in 1951. He earned an M.S. in biophysics from the University of Rochester in 1953 and a Ph.D. in biochemistry from the University of Michigan in 1959.

Schlesinger worked as a research associate at the University of Michigan and at the Massachusetts Institute of Technology before joining the faculty at Washington University in 1964, where he stayed for the rest of his career. He was named professor emeritus in 1999.

Schlesinger was a professor in the departments of microbiology and molecular microbiology at Washington. He did research on viral assembly and replication, authoring nearly 200 papers and books throughout his career.

He is survived by his wife of 62 years, Sondra.

AAMC's Nickens Award goes to Vanderbilt's Hill

HILL

Vanderbilt University School of Medicine professor emeritus George C. Hill is the 2017 winner of the Herbert W. Nickens award.

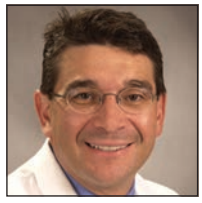
Presented by the Association of American Medical Colleges, the Nickens award recognizes an individual who has promoted justice in medical education and health care equality in the United States.

Hill has spent his career supporting diversity in higher education. He was associate dean for diversity in medical education at Vanderbilt from 2002 to 2011. In 2015, he was appointed as the first vice chancellor for equity, diversity and inclusion; he retired from that position in July.

Hill is a decorated microbiologist whose research explores the molecular biology and biochemistry of African trypanosomiasis.

He received the award, which includes a $10,000 prize, in November at AAMC's annual meeting.

American Heart Association honors Temple's Koch



KOCH

Walter J. Koch, professor and chair of pharmacology and director of the Center for Translational Medicine at Temple University's Katz School of Medicine, received the 2017 Basic Research Prize from the American Heart Association.

With a $5,000 award, the prize recognizes an individual who has contributed significant research in cardiovascular science.

According to the association, Koch was recognized "for basic cardiovascular studies that have identified novel molecular targets for treating heart failure and advanced prospective therapy to the doorstep of clinical trials."

He received the prize in November.

Pufall shares Dorfman award


PUFALL

Miles Pufall, assistant professor of biochemistry at the University of Iowa, has won the Donald D. Dorfman Research Award.

Presented by the Holden Comprehensive Cancer Center at the university, the award recognizes the most significant research paper in the study of leukemia or lymphoma.

Honored for his paper, "Suppression of B-cell development genes is key to glucocorticoid efficacy in treatment of acute lymphoblastic leukemia," Pufall received a $10,000 prize.

The Dorfman award usually recognizes a single individual, but the 2017 award also went to Han-Hui Xue, a UI microbiology professor, for a separate paper.

In memoriam: Bruce Murray Anderson


ANDERSON

Former Virginia Tech University biochemistry professor Bruce Murray Anderson passed away Oct. 29. He was 88.

Born in Detroit, Anderson attended Ursinus College, graduating with a B.S. in chemistry in 1953. He earned an M.S. in biochemistry from Purdue University in 1954 and a Ph.D. in biochemistry from Johns Hopkins University in 1958.

Anderson held several academic positions throughout his career, beginning as an assistant professor at the University of Louisville School of Medicine. He also served on the faculty at the University of Tennessee, Knoxville, and Virginia Polytechnic Institute and State University.

In 1982, Anderson settled at Virginia Tech, where he remained for the rest of his career. He served as professor in the department of biochemistry until becoming professor emeritus in 1999.

He is survived by his daughters, Marcia Anne and Nancy Louise, and his son, David Bruce.

12 members named ASCB fellows

Twelve American Society for Biochemistry and Molecular Biology members are among the 67 new fellows appointed by the American Society for Cell Biology. To be eligible to become an ASCB fellow, a person must be an ASCB member for at least 10 years and have a significant impact in the field of cell biology.

Congratulations to the following members:

Raymond Deshaies, California Institute of Technology/Howard Hughes Medical Institute
Scott Emr, Cornell University
Marilyn Farquhar, University of California, San Diego
Carol Greider, Johns Hopkins University School of Medicine
Susan Gerbi, Brown University
Erika Holzbaur, University of Pennsylvania
Thoru Pederson, University of Massachusetts Medical School
Suzanne Pfeffer, Stanford University
Martin Schwartz, Yale University School of Medicine
Zu-Hang Sheng, National Institute of Neurological Disorders and Stroke
Jeremy Thorner, University of California, Berkeley
Kenneth Yamada, National Institute of Dental and Craniofacial Research

In memoriam: Leonard E. Mortenson


MORTENSON

Leonard E. Mortenson passed away at his home in Willow Street, Pennsylvania, on Oct. 30. He was 89.

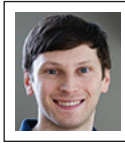
Born in Melrose, Massachusetts, Mortenson studied microbiology and chemistry at Rhode Island State College, graduating in 1950. He earned an M.S. and Ph.D. in bacterial biochemistry from the University of Wisconsin–Madison.

Mortenson held a research position at DuPont from 1954 to 1961 before joining the faculty at Purdue University, where he stayed until 1981. He later took positions at the Exxon Research Engineering Company and on the faculty at the University of Georgia.

Mortenson was highly respected for his research, which focused on bacterial nitrogen fixation. He received the Hoblitzelle National Medal and Award for the discovery of ferredoxin.

Among his many honors, Mortenson was elected as a corresponding member of the French Academy of Sciences in 1965 and a fellow of the American Institute of Chemists in 1968.

He is survived by his wife, Patricia.


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

21 members elected as AAAS fellows

The American Association for the Advancement of Science has elected 21 members of the American Society for Biochemistry and Molecular Biology as fellows for distinguished scientific achievement during their careers. Chosen by their peers, these members have demonstrated outstanding achievements in scientific research, education or leadership.

The AAAS is a nonprofit organization dedicated to the promotion of science through enhancing communication among scientists as well as promoting scientific education and policy. These new fellows will be recognized at the AAAS annual meeting in February.

Congratulations to the following ASBMB members, listed by section affiliation:

Agriculture, food and renewable resources

Autar K. Mattoo, U.S. Department of Agriculture, Agricultural Research Service

Biological sciences

David A. Bernlohr, University of Minnesota
James J. Champoux, University of Washington
John A. Cooper, Washington University in St. Louis
David Cortez, Vanderbilt University
Sudhansu K. Dey, Cincinnati Children's Hospital Medical Center/University of Cincinnati
Iqbal Hamza, University of Maryland
Reuben Stewart Harris, Howard Hughes Medical Institute and University of Minnesota
Gail P. Jarvik, University of Washington Medical Center
D. Borden Lacy, Vanderbilt University
Tomas A. Lindahl, The Francis Crick Institute (UK)
Yoshinuri Ohsumi, Tokyo Institute of Technology (Japan)
John W. Peters, Washington State University
K. Sandeep Prabhu, Pennsylvania State University
Johannes Frederik Gerardus (Hans) Vliegenthart, Utrecht University (Netherlands)

Medical sciences

Blossom Andrea Damania, University of North Carolina at Chapel Hill
Richard L. Gallo, University of California, San Diego
Anil K. Rustgi, University of Pennsylvania

Chemistry

Marvin H. Caruthers, University of Colorado Boulder
Jung-Ja Park Kim, Medical College of Wisconsin
Susan T. Weintraub, University of Texas Health Science Center at San Antonio

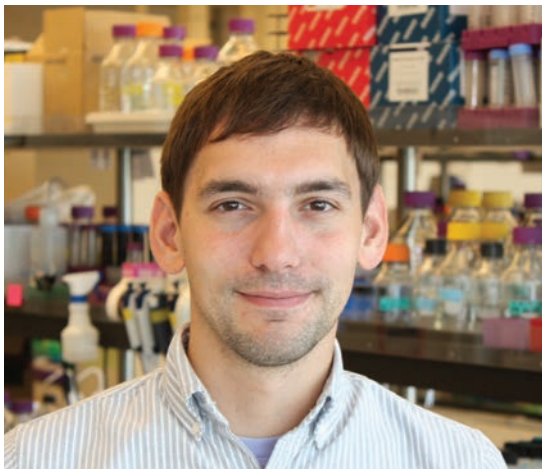
Send us your news

Have you recently been promoted or honored? Do you have good news to share with your fellow ASBMB members? Email it to us at asbmbtoday@asbmb.org — and don't forget to include a photo!

Postdoc wins Tabor award for lipid membrane research

By Dawn Hayward

Lipid membranes surround and protect each of our cells. They serve as a first line of defense, allow for intracellular signaling and keep subcellular compartments separate. The lipid composition must therefore be diverse and distinctive enough to keep a cell running smoothly. Figuring out which lipids are needed where and when can be challenging, however. Itay Budin, a postdoctoral fellow at the University of California, Berkeley, studies lipid properties and why certain ones belong in specific cell membranes. For his research, Budin received a 2017 Journal of Biological Chemistry/Herbert Tabor Young Investigator Award.



COURTESY OF ITAY BUDIN

Itay Budin is a Miller Institute junior fellow at the University of California, Berkeley, and works at the Joint BioEnergy Institute.

Budin investigates how lipid composition affects membrane properties and the consequences of altering particular lipids in model organisms. Lipids make up cellular and subcellular membranes and help maintain integrity and compartmentalization. However, manipulation of these lipids to understand their roles has been done primarily in vitro, and tools to recapitulate findings within living organisms are difficult to develop. Budin uses metabolic engineering to explore lipid composition and functionality. He explained that he does this by altering the genes that give rise to particular lipids. He is then able to "rewire" these pathways within the organism to understand which lipids are necessary for a particular membrane and why. Budin and colleagues have learned that a cell's membrane can act as an environmental sensor, and a particular set of proteins then responds to maintain homeostasis. This work was published in May 2017 in the journal Metabolic Engineering.

JBC Associate Editor Dennis Voelker presented the award to Budin in August at the 2017 Gordon Research Conference on Molecular and Cell Biology of Lipids. Receiving the award was a "great honor and real thrill," Budin said, and receiving it from Voelker, a lipid biologist, reinforced the importance of his work. The award committee thought Budin's work was instrumental in "assigning a crucial mechanistic role for unsaturated lipids in serving as molecular signals that liberate transcription factors from the endoplasmic reticulum in response to a variety of stimuli," they wrote with input from Voelker.

Budin earned his Ph.D. in biochemistry and physical biology from Harvard University in the laboratory of Jack W. Szostak investigating the changes in lipid composition throughout evolution. He then came to the University of California, Berkeley, on a Miller Institute Junior Fellowship. He works with Jay Keasling at the Joint BioEnergy Institute, a research center in Berkeley focused on synthetic biology and metabolic engineering.

The Tabor award is changing

The selection process for the Journal of Biological Chemistry/Herb Tabor Young Investigator Award will change in 2018. A panel of JBC associate editors will select a handful of first authors on JBC papers for the award. The winners will be invited to give short talks at the annual meeting of the American Society for Biochemistry and Molecular Biology. To recommend a first author for the award, contact JBC Associate Editor George DeMartino at george.demartino@utsouthwestern.edu.

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

Ion channel researcher receives Tabor award

By Courtney Chandler

Voltage-gated calcium and sodium channels are involved in numerous biological processes. Sodium channels support fast propagation of electrical impulses. Calcium channels, in response to the electrical impulses, convey calcium ions into cells, and this can have different effects based on the type of cell. One effect is changes in gene expression in both neuronal and cardiac cells, which is mediated by the influx of calcium and a variety of down-stream partners. The molecular properties of both channels are fine-tuned by a number of proteins, allowing them to perform their wide-ranging biological roles.

Manu Ben–Johny, a postdoctoral researcher at Johns Hopkins University, has been researching the molecular and biophysical properties involved in channel fine-tuning, leading to insights into how channel dysfunction may be involved in specific diseases and how synthetic proteins could be used to modulate channel function in a specific manner. For his work, Ben–Johny won a 2017 Journal of Biological Chemistry/Herb Tabor Young Investigator Award. "I am truly humbled to receive such an honor from the American Society for Biochemistry and Molecular Biology," Ben–Johny said. "The young investigator awards are an important way to encourage, inspire and promote creativity and scientific rigor in young scientists."

The award honors Herb Tabor's contributions to science and recognizes innovative new researchers who exemplify scientific creativity. Ben–Johny was selected by a panel of three experts during the Federation of American Societies for Experimental Biology 2017 Conference on Ion Channel Regulation. "The quality of his poster was outstanding," said Robert Colbran, a member of the panel. "He presented the poster with great enthusiasm, displaying an outstanding depth of knowledge in responding to questions."

COURTESY OF MANU BEN–JOHNY

Manu Ben–Johny won a 2017 Journal of Biological Chemistry/Herb Tabor Young Investigator Award during the Federation of American Societies for Experimental Biology's 2017 Conference on Ion Channel Regulation.

Ben–Johny began his graduate studies at Johns Hopkins with the goal of engineering medical devices. However, his focus changed after meeting his eventual graduate studies mentor, the late David T. Yue. "He spoke elegantly about the voltage-gated calcium ion channel as though it were an electrical device," Ben–Johny said. "Instead of building devices, I got to apply the same quantitative thinking to unravel deep mysteries about ion channels and their molecular mechanisms."

Ben–Johny stayed at Johns Hopkins for his postdoctoral studies to continue his research on voltage-gated calcium and sodium channels, including how the calcium-binding protein calmodulin, or CaM, is involved in tuning channel function. He and colleagues have described a mechanism by which CaM signaling to the two ion channels is selectively silenced by different proteins, providing insights into how aberrant signaling may lead to disease.

Ben–Johny's current research focuses on the interplay of ion channels, CaM/calcium modulation and disease. Calcium channel misregulation has been implicated in human diseases, including cardiac and neurological disorders. "Resolving these pathogenic mechanisms and devising novel strategies to rescue channel function in the context of disease is critical and a worthy endeavor," Ben–Johny said.

This year Ben–Johny will become an assistant professor at Columbia University, where he plans to continue his research on calcium-regulated ion channels and disease. "As I embark on this new leg of my scientific journey," he said, "the incredible accomplishments of Herb Tabor both scientifically and in terms of enriching the scientific community serve as an exemplar in my own journey."

Courtney Chandler (cochandl@umaryland.edu) is pursuing her Ph.D. in biochemistry at the University of Maryland, Baltimore.

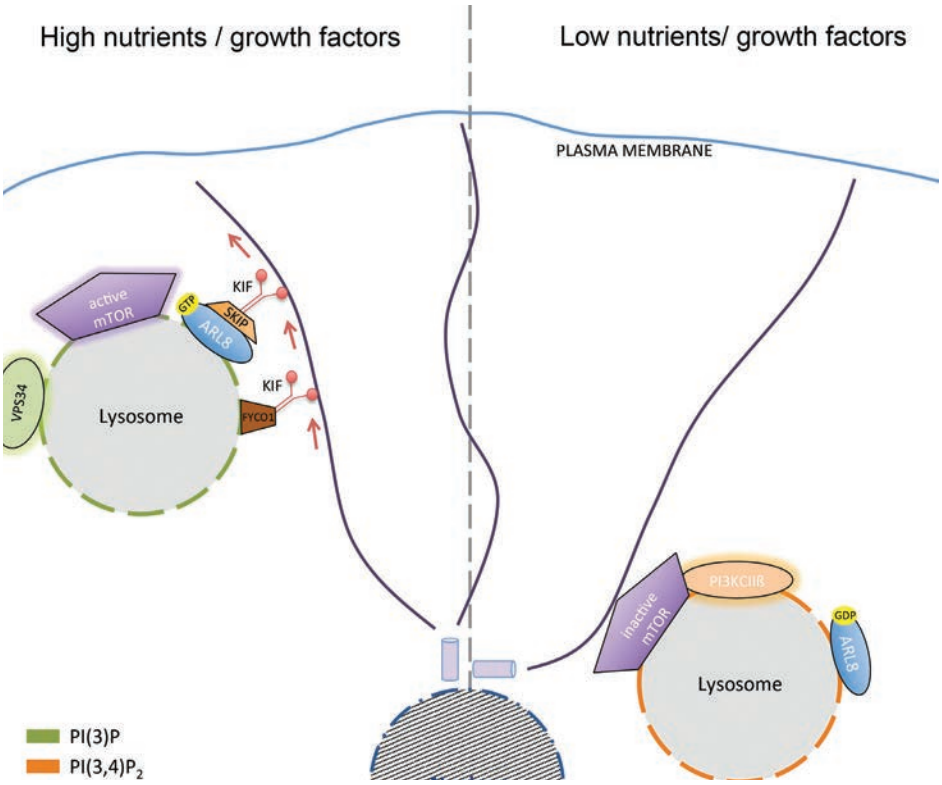
Lipid control of nutrient signaling

By Alexander Wallroth & Volker Haucke

The study of phosphoinositides, or PIs, has been in vogue for some time, as this minor class of short-lived membrane phospholipids regulates many of a eukaryotic cell's physiological functions, including cell polarity, cytoskeletal dynamics, and membrane traffic and signaling. Recent data reveal a mechanism based on local PI generation that couples cell signaling to cellular nutrient status (1).

Different PIs display distinct subcellular distributions, with PI 3-phosphates such as phosphatidylinositol 3-phosphate, or PI(3)P, and phosphatidylinositol 3,5-bisphosphate, or PI(3,5)P_2, being found predominantly within early and late endosomes or lysosomes. The lysosome, via the mechanistic target of rapamycin complex 1, or mTORC1, acts as a central metabolic control hub that integrates extracellular growth factor signals with the cellular nutrient and energy status to direct the cell into either an anabolic or a catabolic state (2, 3).

Disruptions in mTORC1-mediated lysosomal signaling are implicated in diseases such as diabetes, cancer and neurodegeneration. Lysosomal mTORC1 activity depends on growth factor signals that trigger the class I-PI3K-mediated synthesis of plasma membrane PI(3,4,5)P_3, which stimulates mTORC1 via its effector Akt (2, 3).



COURTESY OF VOLKER HAUCKE

Left: Kinesin-mediated dispersion of lysosomes containing PI(3)P synthesized by Vps34 under conditions of high nutrient and growth factor availability activates mTORC1. Right: Perinuclear concentration of lysosomes containing PI(3,4)P_2 synthesized by class II PI3Kbeta under conditions of nutrient and/or growth factor deprivation represses mTORC1 activity.

Researchers now have uncovered control mechanisms elicited by local PI signals at late endosomes/lysosomes that integrate lysosomal mTORC1 activity with cellular nutrient status. Growth-factor deprivation causes the late endosomal/lysosomal recruitment of class II PI3Kbeta, which locally produces a late endosomal/lysosomal pool of phosphatidylinositol 3,4-bisphosphate, or PI(3,4)P_2, that suppresses mTORC1 activity (1). How exactly such suppression occurs is only partly understood, but one important element appears to be the recruitment of inhibitory 14-3-3 proteins to the mTORC1 subunit Raptor by local PI(3,4)P_2 (1).

These findings are surprising given the established function of plasma membrane PI(3,4)P_2 synthesized downstream of class I-PI3Ks in the

CONTINUED ON PAGE 17

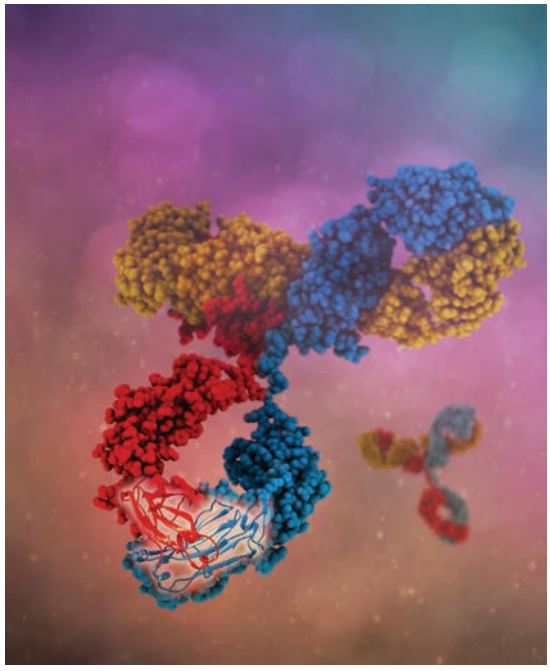
Expanding the reach of therapeutic antibodies

By Sasha Mushegian

A group of researchers has developed an approach to efficiently produce antibodies that can bind to two different target molecules simultaneously, a long-desired innovation in the field of cancer immunotherapy.

Antibodies are proteins produced by the immune system that specialize in recognizing and binding to molecular targets unique to bacteria, viruses or other foreign cells. Because antibodies are stable and long-lasting in the human body and can recognize specific targets precisely, they have been exploited to develop new treatments for diseases. For example, modified antibodies can be used to bind to targets in cancer cells, recruiting the immune system to attack the cancer or preventing the cancer cells from multiplying. Because of their precision and capacity to stimulate the body's immune response, antibody-based therapies typically have fewer side effects than chemotherapy or radiation.

Antibodies are Y-shaped and typically bind a target, or antigen, through the tip of each arm of the Y. In naturally produced antibodies, both arms of a single antibody typically are the same and bind to the same target. One approach to increasing the versatility of antibody therapies is to engineer what are called bispecific antibodies, in which each arm binds to a different molecule. This expands the range of what antibodies can be used for. For example, a bispecific antibody could target a cluster of proteins made up of multiple protein types, or it could bring two different molecules or cell types together.



COURTESY OF MERUS N.V

A single bispecific antibody can bind to multiple antigens, resulting in more specific targeting, novel mechanisms of action and higher clinical efficacies.

One bispecific antibody-like drug — the leukemia drug blinatumomab — is currently on the market. But development of more therapies based on bispecific antibodies has been hampered by technical challenges. For example, certain bispecific antibodies deviate from the standard Y shape and tend to be less stable than conventional antibodies, falling apart easily. Further, certain bispecific antibody formats have tended to be difficult to produce at industrial scales, because they can require specialized engineering processes.

A team overseen by John de Kruif, the chief technology officer of clinical-stage research company Merus N.V., engineered improved bispecific antibodies by making a few key changes to the structure of natural human immunoglobulin G, or IgG, antibodies and showed that they could be manufactured readily. They reported their work in a paper in the **Journal of Biological Chemistry**. IgG is well-studied and is the most abundant antibody produced in the human body.

"We have made, in a complete IgG molecule, only four changes to get from a normal monospecific antibody into bispecific antibody," de Kruif said. "The great thing is that it looks so much like a normal antibody that we can produce it well and we believe we know how it will behave."

The four mutations were in the "heavy chain" protein components of the antibodies. Typically, two identical heavy chains pair up in each antibody. The challenge in creating bispecific antibodies was to induce nonidentical heavy chains to pair — creating "arms" capable of binding different antigens — while discouraging pairing of identical ones.

The team's idea was to introduce amino acids with opposite charges to the two different heavy chains so that identical heavy chains would repel each other, whereas the positively and negatively charged heavy chains would attract. With computational simulations, they identified the right locations in which to introduce these charges, using virtual screening software followed by validation in the lab.

"Using the virtual screening software provided a baseline," said Linda Kaldenberg-Hendriks of Merus, who led the testing of the antibodies. "We identified potential good candidates for design choices in the heavy chain sets, then generated the proteins and characterized them thoroughly. When we saw that they were behaving the way we wanted them to, it was really

satisfying."

The team also investigated the molecular structure of the bispecific antibodies and confirmed that the mutations resulted in only very subtle changes in the "backbone" of the heavy chains, which may explain the stability of these bispecific antibodies.

"A strong point (of this study) was to combine different approaches, the computational tools with the biochemistry and structural biology," said Camilla De Nardis of Merus and Utrecht University, who was a co-lead author on the study.

The proteins that worked paired up to form bispecific antibodies, with very few to no monospecific antibodies in the mixture. The team next subjected them to a battery of tests, confirming that they were as stable as normal IgG antibodies and had similar pharmacokinetic properties.

Because production and purification of IgG antibodies is a well-established industrial process, the team simply could provide manufacturers with the protein sequences modified with the key changes that allowed the proteins to form bispecifics. "We believe we can make virtually any bispecific antibody we want," Kaldenberg-Hendriks said.

The team's bispecific antibodies targeting cancer cell growth factor complexes are now in clinical trials, with more still in the preclinical pipeline. The team is enthusiastic about the potential for the versatile format to be adapted to different types of therapies.

"Antibodies are capable of being so specific, and you can tweak and tune them," Kaldenberg-Hendriks said. "With bispecific antibodies, we believe we can choose the affinities of both arms and balance them so that you can more specifically target tumors, and also recruit other cells or molecules to attack the tumor cells without many side effects. We really think it's the way forward."

Sasha Mushegian (amushegian@asbmb.org) is scientific communicator for the Journal of Biological Chemistry.

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activation of mTORC1 via Akt in growth factor-activated cells. These findings suggest a model whereby local PI switches at late endosomes/lysosomes sense and control cellular nutrient status to regulate mTORC1 activity (1). Consistent with this, late endosomal PI(3)P (4) and PI(3,5)P2 have been shown to activate mTORC1 locally — for example, by association of $PI(3,5)P_2$ with the Raptor subunit of mTORC1(5) — under conditions of ample growth factor and nutrient supply.

As always in a fast-moving field, many open questions remain. For example, recent studies have revealed a surprising, though mechanistically poorly understood, link between lysosome position and nutrient signaling via mTORC1. Peripheral lysosomes display elevated mTORC1 activity compared with perinuclear lysosomes (6). How local pools of PI(3)P or $PI(3,4)P_2$ couple lysosome position to the activity status of mTORC1 is essentially unknown. One possibility is that some of the factors that control lysosome position and/or mTORC1 signaling are regulated by the local PI content. PIs also may modulate the association of mTORC1 components with the lysosomal transport machinery. For example, PIs might regulate the recently described interaction between the Arl8-activating BLOC1-related complex and the mTORC1-associated Ragulator/LAMTOR complex (7).

Interestingly, PI(3)P synthesis by the class III PI3K Vps34 has been shown to facilitate the recruitment of the kinesin-1 adaptor FYCO1 to late endosomes/lysosomes (4). How the resulting dispersion of late endosomes/lysosomes to the cell periphery then causes elevated mTORC1 signaling is unclear. As lysosomes contain a variety of other PI lipids, including PI(4)P and $PI(4,5)P_2$, further unanswered questions are whether and how these lipid pools may be subject to nutrient regulation or, conversely, contribute to the coupling of lysosome function and position to nutrient signals.

We thus face exciting times for lipid biochemistry, as lipids have taken center stage in many aspects of cell physiology. Understanding how local lipid signals sense and control cellular nutrient status at distinct subcellular locations will remain an important area for future studies.

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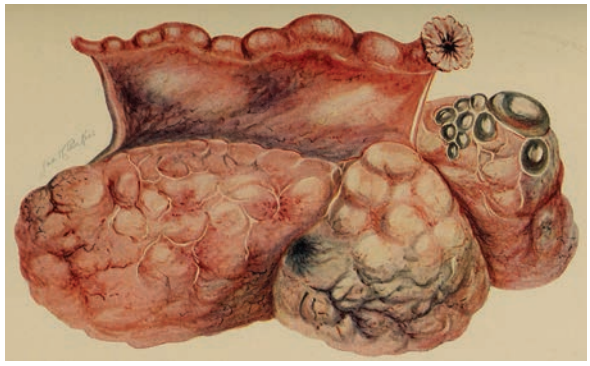
Volker Haucke (haucke@fmp-berlin.de) is a professor for molecular pharmacology and director at the Leibniz-Institut für Molekulare Pharmakologie in Berlin.

Decoding ovarian cancer's dark signaling pathways

By John Arnst

Ovarian cancer is considerably rarer than lung and breast cancer, but it is the seventh-most common cancer in women, and in 2012, 239,000 new cases were diagnosed worldwide. The most common subtype of ovarian cancer, high-grade serous ovarian adenocarcinoma, or HGSOC, is also the most lethal, with a five-year survival rate of less than 40 percent despite high vulnerability to early treatment with chemotherapy.



COURTESY OF WIKIMEDIA COMMONS/THE LIBRARY OF CONGRESS

This image from the 1905 text, "The diagnosis of diseases in women" by Findley Palmer, shows an adenocarcinoma of the ovary in which, according to the caption, "(t)he ovary is enlarged to the size of a child's head."

The mortality rate is high largely because HGSOC tumors tend to shed small spheroids early and prolifically, spreading through the peritoneal fluid in the abdominal cavity to the pelvis and nearby organs. The spheroids, together with tumor-associated T cells and macrophages derived from nearby tissues and the circulation, then manufacture a microenvironment of signaling factors that promote cancer progression, immunosuppression and resistance to chemotherapies. This hostile bubble is maintained by obscure signaling mechanisms between the different cell types, and disrupting them with existing drugs may be a way to fight this notably aggressive form of cancer.

In a paper published in the journal **Molecular & Cellular Proteomics**, researchers at Philipps University in Marburg, Germany, have analyzed the proteome and transcriptome of the microenvironment of tumors in the abdominal fluid taken from women with HGSOC. Using state-of-the-art proteotranscriptomic techniques, the researchers have thrown light on the signaling networks and uncovered associations between factors expressed by the tumors and the likelihood of patient survival.

"We came up with a signaling map between tumor cells, macrophages and T cells," said Rolf Müller, senior author on the paper, "and could now analyze and determine which cells secrete which mediators and on which cells these mediators act."

Müller and colleagues previously had developed a signaling map based on the RNA, or transcriptome, expressed in tumor cells and related macrophages, but they wanted to expand their analyses to encompass the proteome and the aggregate of secreted molecules known as the secretome. Analyses of the transcriptome, proteome and secretome, Müller said, "all have their limitations on their own, (but) you can combine them to obtain something really meaningful."

With a signaling map, Müller and colleagues were able to confirm several known signaling pathways and identify two new subgroups of macrophages, which they named B and G for their respective presence in patients with bad or good prognoses.

Müller and colleagues found that tumor spheroids and macrophages taken from patients with an estimated short survival time, based on the presence of additional factors, produced proteins that support remodeling of extracellular matrices and immunosuppression, which are both key for further cell proliferation. In contrast, macrophages taken from patients with an estimated longer survival time expressed cytokines linked to the activation and attraction of tumor-fighting effector T cells.

While clinical trials are still beyond the horizon, Müller and first author Thomas Worzfeld said they and their colleagues are interested in exploring the gamut of available pharmaceuticals that can interrupt the signaling within tumors as an alternative to disrupting extracellular communication pathways.

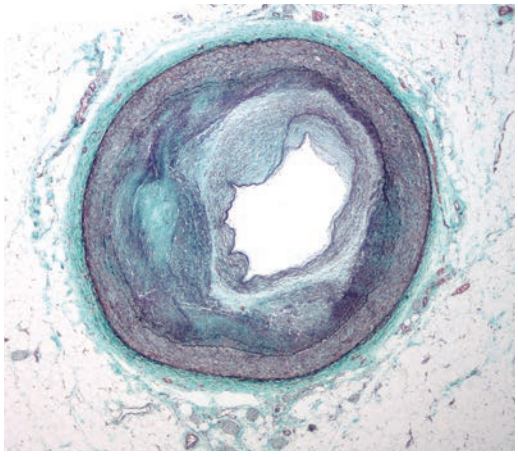
"There's a drug to block almost any important intracellular signaling pathways nowadays," Müller said, "and it would be interesting to see what the important signals are and on what intracellular signaling pathways the signals converge so that we can also block these interactions. That is something we really would like to work on in the future."

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

Arteries and bacterial lipids don't mix

By Alexandra Nail

Perhaps you have heard that brushing your teeth can be good for your heart. Bacteria of the Bacteroidetes phylum may be the main culprits behind the association between periodontal disease and atherosclerosis. In a recent paper in the **Journal of Lipid Research**, Frank Nichols and colleagues from the University of Connecticut reported on their investigation into how intestinal and oral Bacteriodetes may contribute to the development of atherosclerosis.



COURTESY OF WIKIMEDIA COMMONS/NEPHRON

This low magnification micrograph shows the distal right coronary artery with complex atherosclerosis and luminal narrowing. The artery's three layers can be seen as well as features of atherosclerosis. The section of the artery shown has only 25 to 35 percent of the cross-sectional area it once had.

Atherosclerosis is a disease in which plaques build up in arteries. The plaques contain fat, cholesterol, calcium and other substances found in the blood. Over time, these plaques cause arteries to narrow and harden, and this leads to an increased risk of cardiovascular disease. Many factors can contribute to the development of atherosclerosis, including genetic predisposition, lipid metabolism and inflammation.

For their work, Nichols and his team first focused on a specific oral Bacteriodetes organism, Porphyromonas gingivalis. This organism has been associated with an increased risk of developing atherosclerosis in rodents and periodontal disease in humans (1, 2). In addition, Nichols' group has shown that both oral and intestinal Bacteroidetes produce specific serine dipeptide lipids. The group also has shown that these bacterial lipids activate macrophages and immune cells that promote inflammation in tissues.

Two serine dipeptide lipids are produced by P. gingivalis, Lipid 654 and Lipid 430. A chemical bond in Lipid 654 is cleaved to yield Lipid 430.

For this study, the group relied on high-performance liquid chromatography, or HPLC, and tandem mass spectrometry to isolate and quantify Lipid 430 to Lipid 654 ratios. HPLC technology can resolve component lipids of a complex lipid mixture using a column containing particles of silica gel. These particles can interact with, in this case, lipid components. As the lipid components elute from the HPLC column, the eluting lipids can be introduced into a mass spectrometer to identify specific metabolites and quantify their levels in a given sample. Using this technology, the researchers saw not only that Lipid 654 was present in human carotid arteries but also that the Lipid 430 to Lipid 654 ratio increased by at least tenfold in diseased arteries compared with artery samples from young subjects. This suggests that Lipid 654 hydrolysis is increased in diseased carotid arteries. "Many think that atherosclerosis is caused by eating fatty foods," Nichols said, "but it is now apparent that other lipids produced by oral and intestinal bacteria accumulate in diseased arteries."

In addition to finding an accumulation of Lipid 430 in diseased human carotid arteries, the group identified a mechanism for cleavage of Lipid 654. They tested a panel of common lipase enzymes, some of which are associated with atherosclerosis, for their ability to hydrolyze Lipid 654 to Lipid 430. They tested human, pig or honey bee enzymes. After incubation with the enzymes, the amounts of Lipid 654 and Lipid 430 were measured by mass spectrometry. They found that phospholipase A2, an enzyme associated with macrophage activation, was the only enzyme class in the panel that was able to cleave Lipid 654.

The research by Nichols' lab suggests that patients with atherosclerotic disease have an elevated level of Lipid 430 resulting from Lipid 654 cleavage mediated by phospholipase A2. Furthermore, Lipid 430 potentially can promote inflammation at the site of plaques. "For the future," Nichols said, "it will be important to identify the enzymes responsible for the synthesis of these lipids and target specific enzymes with pharmacological inhibitors and/or dietary modifications."

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From the journals

By Sasha Mushegian, Angela Hopp & Saddiq Zahari

We offer a selection of recent papers on a variety of topics from the **Journal of Biological Chemistry, the Journal of Lipid Research and Molecular & Cellular Proteomics**.

A chemical cocktail that reprograms mouse cells

Scientists can reprogram differentiated somatic cells using transcription factors or small molecules, but the types of cells they've created thus far using chemical cocktails have been limited. In the **Journal of Biological Chemistry**, Shangtao Cao and colleagues from the South China Institute for Stem Cell Biology and Regenerative Medicine identify a combination of small molecules and growth factors that can reprogram mouse embryonic and adult fibroblasts into epithelial-like cells, an intermediate cell type capable of further differentiation. These chemical tools may enable additional advances in regenerative medicine.
doi: 10.1074/jbc.M117.812537

Malaria vaccine development

The only available vaccine against Plasmodium falciparum, one of the causative agents of malaria, shows limited efficacy. Malaria vaccine developments face major challenges due to the significant sequence diversity of the known parasite antigens. In a study published in the journal **Molecular & Cellular Proteomics,** Matthias Marti and colleagues at the Harvard T.H. Chan School of Public Health aimed to overcome this problem by performing a mass spectrometry technique to profile the surface proteome of red blood cells infected with P. falciparum. The investigators identified a set of single-copy surface antigens with low sequence diversity, some of which further were validated to be immunogenic and thus potentially could be developed as vaccines.
doi:10.1074/mcp.RA117.000076

How RNA proteins modulate phase separation

RNA granules are spots in the cell in which RNA molecules and RNA-binding proteins aggregate to carry out RNA modifications that affect gene expression. Rather than being bound by a membrane, these compartments are delineated by differences in viscosity from the surrounding cytoplasm. Phosphorylation of intrinsically disordered regions of RNA-binding proteins contributes to the assembly of RNA granules. Yuan Lin and colleagues from the University of Texas Southwestern Medical Center at Dallas write in the **Journal of Biological Chemistry** that tyrosine residues in these RNA-binding proteins modulate their ability to induce the liquid-liquid phase separation process that leads to RNA granule formation and that the phase-separation threshold depends on the number of phosphorylated tyrosines. These insights show how proteins can modulate the properties of fluids inside the cell, potentially influencing how genes are expressed.
doi: 10.1074/jbc.M117.800466

What prion protein does when it's not wreaking havoc

Misfolding of prion protein, or PrP, causes prion diseases, including bovine spongiform encephalopathy (more commonly known as mad cow disease) and the human Creutzfeldt-Jakob disease. However, the physiological function of normal, non-pathogenic PrP is poorly understood. Gui-Ru Wu and colleagues at the Chinese Academy of Sciences examined the role of normal PrP in two cancer cell lines and showed that it was critical for signaling through the tumor necrosis factor alpha pathway. Their findings were published in the **Journal of Biological Chemistry**. PrP sequestered the tumor-suppressor protein CYLD, resulting in proinflammatory cytokine production. Thus, normal PrP may be involved in both immune responses and tumor progression.
doi: 10.1074/jbc.M117.787283

The cholesterol connection to rare muscle-wasting disorders

Rare inherited mutations in the DYSF gene cause deficiencies in the protein dysferlin, resulting in debilitating limb-girdle muscular dystrophy type 2b. To date, there are no approved therapies for dysferlinopathies. Drug development has been slowed, in part, by the lack of animal models that closely mimic human symptoms. A new paper in the **Journal of Lipid Research**, however, reports the creation of a mouse that comes closer than its predecessors and contemporaries. A research team led by Pascal N. Bernatchez at the University of British Columbia set out to create the new mouse model after observing that elevated so-called bad cholesterol levels correlate with disease severity in some muscular dystrophy patients. While mice with the DYSF gene knocked out display muscle degradation, they usually don't lose the

ability to walk, as human patients do. Bernatchez's team knocked out both the DYSF gene and the APOE gene in their mice. Loss of apolipoprotein E function increases bad cholesterol. The team's double-knockout mice developed more dramatic muscle wasting than mice with only DYSF knocked out and eventually were unable to walk. The researchers hope their new model will help scientists better understand the human disease. Importantly, the researchers wrote, though it's unclear at this stage precisely how bad cholesterol exacerbates muscle damage, their model revealed "a striking correlation" that deserves further investigation. The work was supported by the Jain Foundation.
doi: 10.1194/jlr.M079459

Splice variants with opposite effects on metastatic breast cancer

Triple-negative breast cancers lack the three receptors that the most effective treatments today target, making a triple-negative diagnosis quite devastating. In the **Journal of Biological Chemistry**, James DeLigio and colleagues from the Virginia Commonwealth University write that they examined the effects of downregulating two forms of a protein in triple-negative breast cancer cells. Cytoplasmic polyadenylation element binding protein 2, or CPEB2, is a translational regulator that has two isoforms derived by alternative splicing, one of which is upregulated in aggressive metastatic breast cancers. In the researchers' study, the two isoforms had opposite regulatory effects on pathways involved in epithelial-to-mesenchymal transition and hypoxic response, processes that contribute to invasiveness, with the cancer-associated isoform upregulating these pathways' activities and the other isoform downregulating them. The cancer-associated isoform of CPEB2 appeared to exert this effect by enhancing the translation of the transcription factors TWIST1 and HIF-1alpha, both of which have been implicated in cancer.
doi: 10.1074/jbc.M117.810127

MAPping the kinase targets in Arabidopsis

The mitogen-activated protein kinases, or MAPKs, play important roles in regulating cellular processes. In the plant Arabidopsis, little is known about the functions of MAPK family members MPK3, MPK4 and MPK6, especially in cell division, growth, development and innate immunity. In a study in the journal **Molecular & Cellular Proteomics**, Heribert Hirt and colleagues from King Abdullah University of Science mapped the substrates of these kinases by performing a phosphoproteome analysis on wild-type plants and plants that have mutations in any of the three MAPK genes. The investigators identified and validated a number of novel MAPK targets that either were shared by or specific to each of the three MAPKs, increasing the repertoire of known MAPK substrates.
doi:10.1074/mcp.RA117.000135

COURTESY OF ALBERTO SALGUERO QUILES/WIKIMEDIA COMMONS

Arabidopsis thaliana

How malaria parasites get around in multiple hosts

The malaria parasites, which belong to the genus Plasmodium, have multiple stages in their life cycles in both mammalian and insect hosts. Judith Green and colleagues at the Francis Crick Institute in London are studying the life-cycle stages of Plasmodium berghei, which causes malaria in certain rodents and is used as a model for the human malarias caused by four of its parasitic kin. Green's team reported in the **Journal of Biological Chemistry** that it had characterized the components of the glideosome — a membrane-associated molecular

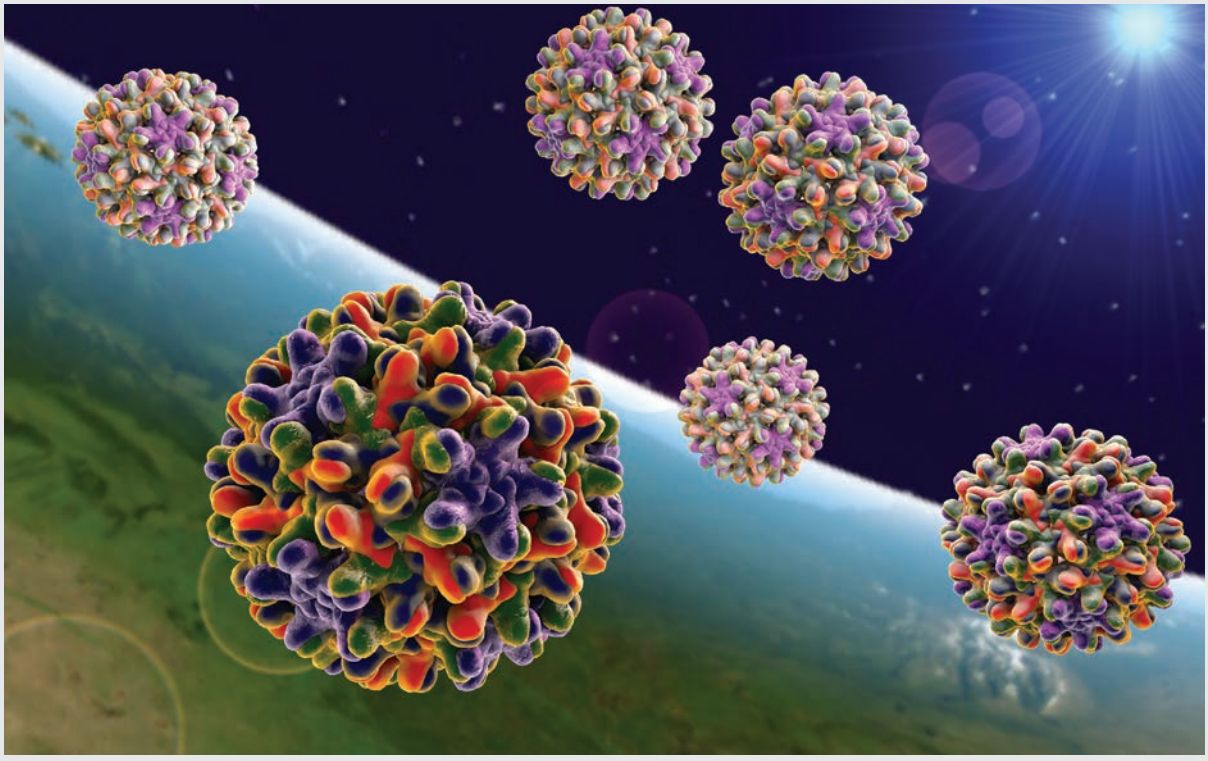
motor that P. berghei uses for motility and to invade hosts— across all the life cycle stages. The researchers identified a myosin light chain in the glideosome that previously was unknown in malaria parasites and showed that the presence of this chain doubled the parasite's sliding velocity in vitro.
doi: 10.1074/jbc.M117.802769

Understanding arginine phosphorylation

Phosphorylation has been studied widely in terms of its modifications on serine, threonine and tyrosine amino acids. Even though phosphorylation is known to occur on arginine residues, its extent and effects are a lot less studied. Investigators at the University of Greifswald in Germany, led by Dorte Becher, examined the arginine phosphoproteome of the Gram-

Alcohol consumption plus hepatitis B raises cholesterol in mice

Though a safe and effective vaccine for hepatitis B is available and used worldwide, the World Health Organization estimates that still some 257 million people are living with the viral infection. The virus is treatable but causes liver scarring and in some cases liver cancer. Intravenous drug users who share needles are especially at risk of infection and are likely to abuse multiple substances, including alcohol, which causes liver damage as well. Given that damage to the liver affects how the organ creates and clears cholesterol and given that studies have found that hepatitis B alone and alcohol alone affect cholesterol homeostasis, a research team led by Qin Ning at Tongji Hospital at Huazhong University of Science and Technology in Wuhan, China, wanted to learn more about the combined effects of hepatitis B and alcohol consumption on cholesterol deposition in the liver. In a new paper in the **Journal of Lipid Research**, the team reports that the virus and alcohol have a "synergistic effect" on cholesterol metabolism. That is to say that the combination increases cholesterol biosynthesis, decreases cholesterol utilization and impairs cholesterol uptake. To figure this out, the team created a mouse model with chronic hepatitis B and gave it ethanol. This resulted in increased cholesterol accumulation in the liver. Later experiments, using only cells, produced results that the researchers say indicate it is the HBc protein specifically that activates cholesterol biosynthesis and inhibits cholesterol degradation in the presence of alcohol.
doi: 10.1194/jlr.M079533



The hepatitis B virus can live outside the body and remain infectious for about a week.

positive bacterium Staphylococcus aureus artificially lacking the PtpB gene, a putative arginine phosphatase. Using a classical mass spectrometry database search and spectral libraries of synthetic arginine phosphorylated peptides, the investigators identified 207 arginine phosphosites in S. aureus, which could allow further investigation of the physiological relevance of arginine phosphorylation in bacteria. The study was published in the journal **Molecular & Cellular Proteomics**.
doi: 10.1074/mcp.RA117.000378

A silver lining to obesity-induced neuronal damage

Melanocortin-4 receptors are expressed in the brain and are part of the insulin- and leptin-sensing pathways that regulate appetite and energy expenditure. MC4R agonists cause overweight mice to consume less food, but prolonged agonist binding leads to desensitization. Kimberly Cooney and colleagues from the University of Arkansas for Medical Science write in the **Journal of Biological Chemistry** that palmitate-induced neuronal injury, a common complication of obesity, impaired the endocytosis of agonist-bound MC4R, decreasing desensitization. This suggests that neuronal stress experienced during obesity may unexpectedly increase the effectiveness of MC4R agonists.
doi: 10.1074/jbc.M117.785758

Nipping a cancer-related protein in the bud

Matrix metalloproteinases, or MMPs, are enzymes involved in signaling and tissue remodeling. They frequently are dysregulated in cancers and other diseases, but attempts to find selective MMP inhibitors have been largely unsuccessful. In the **Journal of Biological Chemistry**, Robert Scannevin and colleagues at the pharmaceutical company Janssen Research and Development report a new inhibitor that targets MMP-9 by allosterically inhibiting activation of the MMP-9 zymogen. The inhibitor reduced the severity of autoimmune encephalomyelitis in mice.
doi: 10.1074/jbc.M117.806075

A ubiquitous signaling protein regulates alternative splicing

Glycogen synthase kinase-3, or GSK-3, is a ubiquitous, constitutively active signaling protein whose targets have not been characterized comprehensively. Mansi Shinde and colleagues at the University of Pennsylvania performed unbiased phosphoproteomic characterization of mouse embryonic stem cells with and without the GSK-3 gene and found that, among other targets, GSK-3 consistently targeted RNA splicing factors. Absence of GSK-3 changed the splicing patterns of hundreds of RNAs, revealing a previously unknown role of the enzyme. The results were published in the **Journal of Biological Chemistry**.
doi: 10.1074/jbc.M117.813527

Upcoming ASBMB events and deadlines

JAN
2: Art of Science Communication course open for applications
25: Student Chapters travel-award deadline for ASBMB annual meeting

FEB
5: Art of Science Communication course begins
8: PROLAB application deadline
12: Outreach poster deadline for ASBMB annual meeting
16: Outstanding Student Chapter Award deadline for ASBMB annual meeting
27: Late-breaking abstract-submission deadline for ASBMB annual meeting
27: ASBMB annual meeting early registration deadline

MAR
15: Accreditation application deadline

APR
21–25: ASBMB annual meeting, San Diego

ASBMB
American Society for Biochemistry and Molecular Biology

A battered island rises

Hurricane Maria damaged labs and destroyed equipment, but scientists in Puerto Rico persevere

By John Arnst

More than 100 days ago, Hurricane Maria tore across Puerto Rico with 113 mph wind gusts, torrential downpours and flash flooding. In the storm's immediate aftermath, island residents found themselves without electricity and running water. Help from the mainland United States came slowly. After an initial humanitarian crisis and now on a grinding road to recovery, the people of Puerto Rico have come together in ingenious and innovative ways — fetching groceries on farm tractors and holding university lectures in tents.

The situation has improved somewhat in the last two months; as of December, 93 percent of the island had access to boil-advisory water, 73 percent of cell tower sites were running and 66 percent of electrical power on the island had been restored. Among the efforts of a host of nonprofit organizations, the American Society for Biochemistry and Molecular Biology awarded $76,000 in grants to 52 scientists in Puerto Rico to help mitigate the damage to their laboratories and lives across the island.

Lost with the freezers

José A. Rodríguez-Martínez, an assistant professor of biology who started his lab at the University of Puerto Rico-Río Piedras in 2017, lost all his frozen supplies when the building's backup generators failed to start, leaving the lab without power for four days.

RODRÍGUEZ-MARTÍNEZ

"All of my freezers lost temperature, so I lost everything — all my molecular biology reagents, all my enzymes, all my PCR master mixes," he said. "The week before Maria, we successfully purified our first protein … then Maria came and took it away."

COURTESY OF HECTOR JIRAU

This service station in Utuado was demolished by Hurricane Maria.

The protein that Rodríguez-Martínez's lab had purified was a transcription factor, OPTIX, that is linked to the rise of red color patterns on the wings of Heliconius butterflies. Despite these setbacks, Rodríguez-Martínez's lab, which examines the DNA binding preferences of transcription factors, sequence-specific DNA-binding proteins, got back on track in the final months of 2017.

While electricity returned to his lab within days via the backup generators, it took weeks to come back to other parts of the university and the surrounding areas of San Juan. Rodríguez-Martínez, who lives close to the campus, had to wait nearly two months for his power at home to return.

"We had to figure out where to get ice," he said. "I was freezing my water bottles in the minus-80-degree freezer that survived … Since it was completely empty and clean since I lost everything, it was perfect."

Research in Rodríguez-Martínez's lab picked back up by late November. "(We are) moving basically at the same pace, we just have to redo

Facing page: Ruined buildings are seen from a U.S. Customs and Border Protection, Air and Marine Operations, Black Hawk helicopter during a flyover of Puerto Rico after Hurricane Maria, Sept. 23, 2017.

COURTESY OF WIKIMEDIA COMMONS /U.S. CUSTOMS AND BORDER PROTECTION/PHOTO BY KRIS GROGAN

COURTESY OF HECTOR JIRAU

Many trees were uprooted by Hurricane Maria's strong winds.

a lot of things," he said. "I was super lucky … a big portion of my reagents were from New England Biolabs and Promega, and both of them replaced my entire purchases." Rodríguez-Martínez plans to use his ASBMB grant to cover the remaining reagents that were lost in the downed freezers.

"I think a main concern now is getting electricity back to people," he said. "The government is reporting mostly on the percentage of electricity generated, (but) they don't report the number of customers or households, the percentage of households … people are desperate to get power, mostly outside of the metropolitan area."

Flooding and faltering

In addition to widespread electrical outages, many labs flooded after roofs were damaged by Maria's heavy winds.

JIRAU

Hector Jirau works in the laboratory of the environmental toxicologist Braulio D. Jiménez and currently is investigating the effects of particulate matter on asthma and other respiratory diseases. Jiménez's lab is on the second floor of the 10-story Guillermo Arbona Building at the university's medical center. When the hurricane's winds damaged the building's roof, the upper floors flooded and water leaked down to the second floor.

"When I went to the lab, it was flooded, and most of our instruments were gone," Jirau said. "We have a cell culture room which pretty much doesn't work right now."

The computer that contains data for Jirau's thesis also was damaged; he hopes his ASBMB grant will cover the repair.

"Right now I have no access to my computer," he said. "I need to at least save the hard drive and extract all my data (for) my thesis project."

Many students working in storm-damaged labs, including Jirau, are moving to labs at mainland U.S. institutions, including Harvard University and Brown University, where they can continue their research alongside experts in their fields. The nonprofit Ciencia Puerto Rico, which aggregates and disseminates sources of scientific aid available to Puerto Rican researchers, shared information about this opportunity and many others, including grants from the ASBMB, the American Association for the Advancement of Science and similar associations.

"I'm talking with a professor from Harvard School of Public Health, and we're trying to collaborate so I can go see the campus and continue my thesis over there," Jirau said.

MARTINEZ

Public transportation was also shut down in San Juan, according to Jorge Martinez, another graduate student in Jiménez's lab. Martinez lost a number of reagents to the downed freezers, and his car broke down in the aftermath of the hurricane; he plans to cover the cost of a transmission repair and some of the lost reagents with his ASBMB

grant.

Martinez lives in San Juan on the northern coast, but he rode out the hurricane with his family at their home in the middle of the island. "The day after Maria, we tried to visit some family members that were close by, but it was a bit too much and it broke the transmission of the car," he said. "We had to go over trees, over debris, over boulders — it's a four-wheel-drive car, but it can't do magic."

Between his vehicle's broken transmission and the shutdown of public transportation in San Juan, Martinez had to carpool both to the lab and to his work auditing biomedical studies at the Fundación de Investigación, a part-time job he took to help his family make ends meet while the hurricane recovery is under way.

"Puerto Rico has always been an island where every Puerto Rican matters for every other Puerto Rican," Martinez said. Many residents near the center of the island are farmers, he said, and some of them used their tractors and similar equipment to get supplies to people who were stranded. "They improvised transportation methods from one side of the broken bridge to the other to at least send food to those that couldn't access any supermarkets."

COURTESY OF HECTOR JIRAU

A Bio-Rad multi-imaging machine in Braulio D. Jiménez's lab, used to capture images of various types of fluorescent samples, was damaged beyond repair by flooding.

Beyond San Juan

Jirau weathered Maria with his family in the mid-island municipality of Utuado. "The center of the island was hit pretty hard," Jirau said. "In Utuado, every 10 or 15 kilometers or so, you will see a lot of houses without roofs. You could actually see a lot of people living outside or having tents nearby their houses."

BONANO

The recovery efforts appear to be moving more slowly outside the capital and surrounding metropolitan area. Forty-five minutes to the southeast of San Juan, Puerto Ricans in the municipality of Cayey still lacked basic amenities in early December, said Keisy Bonano, an undergraduate at the University of Puerto Rico at Cayey.

"Cayey still has many areas where there's no electricity, there's no water," said Bonano, who does research at the University of Puerto Rico's medical sciences campus in San Juan. She lost lab supplies due to power outages, and her zebrafish larvae died after their incubators shut down.

Even the parts of the island that weren't directly hit by the hurricane,

COURTESY OF HECTOR JIRAU

Many roads in Utuado were damaged or obstructed by the storm.

such as the western municipality of Mayagüez, lost electricity and access to running water.

RIVERA

Before the hurricane hit, Andrea Rivera, an undergraduate student at the University of Puerto Rico at Mayagüez, split her time between a lab at the Mayaguez campus and a lab in San Juan that she became involved in through a Step-Up internship with the National Institutes of Health. In the aftermath of Maria, Rivera has continued to commute two hours to the lab in San Juan, where she works from Friday mornings through the weekend.

"I, fortunately, have electricity and my water running, but many of my friends don't right now," Rivera said. "Everyone is sharing their rooms … we're students, we don't have a lot of money so we're trying to help each other out because it's been a rough couple of months." Because areas of Mayagüez still lacked power, Rivera said, the university picked up some of the slack by keeping libraries open until midnight.

Tents and flashlights

In late October, classes resumed at the University of Puerto Rico in San Juan. With electricity still out in many buildings, Rodríguez-Martínez said, a number of the larger lectures were given outside in tents. "It was an interesting scene, looking at the professors teaching under a tent and especially doing genetics courses and organic chemistry courses outside of the classroom."

Classes also resumed at the campus in Cayey, Bonano said, with similar workarounds. "The science and math buildings are the only two buildings (at the Cayey campus) that have electricity, so we're taking classes with flashlights pointing to the board so we can see," she said.

Across the island, a sense of normalcy has started to return, even as many residents still wait for the promised recovery.

"We're still here, we're still striving," Rivera said. "We want the world to know that we weren't wiped off the map. We are here and we will succeed and we will come out of this."

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

Membrane maestro George Carman returns to the JLR

Veteran associate editor will work to draw specialized papers to the Journal of Lipid Research

By John Arnst

What a cell does with its lipids is a subject of deep inquiry. George Carman's laboratory at Rutgers University examines the genetic and biochemical control mechanisms that cells use to balance the synthesis of triacylglycerol, or fat, for stasis and synthesis of membrane phospholipids for growth.

Carman received his Ph.D. from the University of Massachusetts in Amherst after attending Seaton Hall University and William Patterson University in New Jersey. He did his postdoctoral research with the biochemist William Dowhan at the University of Texas medical school in Houston and is the founding director of the Rutgers Center for Lipid Research.

A seasoned editor and reviewer, Carman has been involved in various capacities with the Journal of Biological Chemistry, as well as Biochimica et Biophysica Acta-Lipids, Applied Environmental Microbiology, Analytical Biochemistry and the Journal of Bacteriology, since the mid '90s. He previously served as an associate editor of the Journal of Lipid Research from 2003 to 2006.

Carman re-joined the ranks of associate editors at the JLR in January 2017. He spoke with John Arnst, ASBMB Today's science writer, about his lab's work and his longtime interest in lipids and cell membranes. The interview has been edited for clarity and length.

What is your group focused on?

Our experimental system is baker's yeast, and our current focus is on the phosphorylation and dephosphorylation of the phosphatidic acid phosphatase enzyme, whose activity plays a key role in partitioning phosphatidic acid between the synthesis of fat and phospholipids. We identified the gene for this enzyme, which allowed us to make mutations in the gene; and then, by looking at phenotypes of the mutants, we could understand how the enzyme was involved in lipid metabolism. The enzyme plays a key role at a branch point in lipid metabolism, controlling whether cells make membrane phospholipids for growth or take the phosphatidic acid and make triglyceride.

This is something that we've worked on for a number of years, and when we discovered the gene for this enzyme in 2006, there was a

IMAGE COURTESY OF GEORGE CARMAN

One of the walls in George Carman's lab reflects his appreciation of Star Wars.

whole explosion in studying how this enzyme works. It's so important in governing whether cells make fat or membrane phospholipids that we're trying to understand how it ticks.

How did you become involved in working with membranes?

After graduation I looked for postdoctoral positions, and one of the places that I applied to was the University of Texas medical school in Houston with William Dowhan.

In 1972, Singer and Nicholson came out with their famous model of what the membrane looks like that has membrane phospholipids and proteins and the proteins moving through it. (Author's note: Seymour J. Singer and Garth L. Nicholson's fluid mosaic model is regarded as a cornerstone of cell biology.) After that publication, everybody in the world was working on membrane structure and function; that was Dowhan's interest, and that's one of the reasons why I wanted to go to work with Dowhan. That's why I became very interested in the biosynthesis of membrane phospholipids, because these enzymes that make the phospholipid membranes, they're also part of the membrane themselves, so they're kind of synthesizing the environment that they live in.

Did anything occur in a milestone sort of way that made you choose science as a career?

I grew up in Jersey City, where the teachers were more concerned with making sure somebody didn't get killed or get into drugs ... it was a horrible way that I grew up, and I always did well in chemistry and biology, so I gravitated to that. I had biology teachers and chemistry teachers in high school that really encouraged me to work hard, and I think those are the things that kept me going. My mother is the only one in the family who finished high school before my older sister, and I was the first one in my family to go to college.

I wanted to be a high school teacher like the biology teacher and the chemistry teacher that encouraged me. Then when I went to college I met other chemistry and biology professors that encouraged me, and I wanted to be like them, so instead of being a high school teacher, my goal was to become a college professor. I didn't know anything about research.

When I wound up going for my master's degree, I had a research project working with enzymes and bacteria, and I said, "This is fun." Then I went to the University of Massachu-

setts, and I met this microbiologist who was studying enzymes, and I really liked it. Doing the independent research is very different from doing research in a laboratory course, and this was where I began to really think that this is my calling.

I'm still a teacher, and I spend a lot of time with my students — my research students but also my classroom students — and I try to do for them what my teachers did for me, so part of my motto is pay it forward. Sometimes you can't pay back your teachers that helped you, but you pay it forward by helping people that are coming up … I often call Dowhan up and ask him for advice. I'm 67 years old and he's 75, and the learning doesn't end. The training begins early, but it keeps going.

What does it mean to you, on a personal level, to be an associate editor at the JLR?

One thing that I'd like to do is help facilitate the growth of the journal. If you have a paper that's focused and it doesn't really belong in (a broad journal like) JBC, the JLR is the place that you want to go. What I want to do is get out there and convince all of the people in the field that the JLR is the best place for that specialized lipid paper.

What do you do outside of the lab? Do you have any hobbies?

I play racquetball or I go swimming every day. Exercise is really important in my life to keep healthy. I also play tennis in the warmer weather, and in the cold weather we have family ski trips.

One thing that's really critical in balancing your life is your family, and it's really important that family comes first. I have three children, and one grandchild as of July. My oldest daughter works for a flavor company as a marketing analyst, so she really didn't go into science, and my older son is a pilot in the Coast Guard. My third child just started graduate school at the University of Pennsylvania in biochemistry.

All we wanted them to do is be good citizens and that kind of thing. We spent a lot of time with our kids, and it's paid off, because they haven't gotten in trouble and they're just really good kids.

Do you have any words of wisdom or a favorite motto?

One thing that I teach my students is to always give other people credit for what they do. If you give other people credit, it doesn't detract from you. There's a lot of politics in science, and if you always promote the work of others, you're not going to make enemies. My motto is be fair to the people who made the discoveries; don't forget the past.

The other thing that I say is just because something's published doesn't mean it's true; it doesn't mean it's fact. There's a lot of stuff out there that's fabricated; a lot of stuff out there — it's just not true. So I always impress upon my students to question what's published, and if you have to do the experiment over to make sure it's correct, do it over, because you want to get the science right.

"I always impress upon my students to question what's published, and if you have to do the experiment over to make sure it's correct, do it over, because you want to get the science right."

– GEORGE CARMAN

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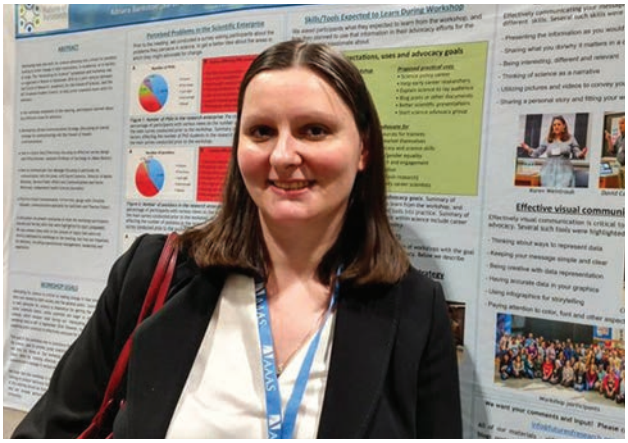
Attending conferences as a science policy researcher

By Adriana Bankston

Over the last 11 years, I've attended multiple scientific conferences where I presented my work. I started going to these conferences as a graduate student. This continued even after I left academia but in a different capacity. And I know I'm not alone. Of the 3,600 people who attend the American Society for Biochemistry and Molecular Biology annual meeting, a percentage do not currently study or work in academia. If you are one of them — or if you're considering going to scientific meetings as a non-bench scientist — here are a few lessons from my experience.



COURTESY OF ADRIANA BANKSTON

Adriana Bankston presents a poster for Future of Research, a nonprofit organization supporting early-career scientists, at the 2017 American Association for the Advancement of Science meeting.

Away from the bench

The transition out of academia can be daunting for many reasons, including the lack of structure and support when you no longer work in a lab. You may have to start over in a new field, create a new professional network and learn how to sell your work to a new type of audience. Attending scientific conferences can provide opportunities to practice these skills in your new role.

As a former bench scientist, you will probably attend the same types of conferences as when you worked at the bench. However, given your new role, you might engage in different types of activities. I personally have transitioned from my bench work as a skeletal muscle biologist to being a science policy researcher (volunteer) at the nonprofit organization Future of Research. This experience has allowed me to discover my own professional identity outside of academia and to present our organization's work at conferences across the country and get a sense of how we best can advocate for graduate students and postdocs.

Specific activities

Our work at Future of Research is classified in the science policy or education category at scientific meetings. This has allowed me to showcase our projects, advertise the organization and build a network with people working in these areas. I have presented posters on our advocacy and career workshops at scientific conferences. While summarizing a different type of data (essentially like social science) wasan interesting challenge in itself, I also found these presentations to be very exciting and motivating, because I was able to engage more people in our work.

I also have co-moderated workshops on career development for junior scientists, enhancing connections for advancing postdoctoral training, and resources to address challenges for international graduate students and postdocs. These workshops have solidified my interest in learning about and working toward addressing the needs of graduate students and postdocs across the country. This experience also helped me become more comfortable and actually begin to enjoy talking in front of a crowd about my work. Although I was fairly shy while at the bench, doing these workshops came very easily to me because I was discussing a topic that I felt very passionate about.

I also participated in discussions related to science, technology, engineering and math, or STEM, graduate education during an open forum at a national meeting, and I summarized sessions from various meetings on training and policy topics, including economic implications of scientific training, tracking postdoc trends and outcomes, rethinking graduate education in the 21st century and others.

While attending these conferences as a science policy researcher, I've also tried things I was afraid to do. I volunteered to speak about the importance of science advocacy (and engaging graduate students and postdocs in advocacy) in a short video at a meeting to support the advocacy efforts of the host society. Because I wanted to help others interested in transitioning to science policy by sharing my experience, I also co-moderated a science policy roundtable at that meeting. I was excited when multiple people asked for my advice. I am also always glad to help others — I've had mentors who pointed me in the right direction, and I want to pay it forward now by helping the next generation.

General advice

Drawing from my own experience, I offer some general pointers to help you make the transition into a new field and present that work at scientific conferences:

Immerse in your new field. You may be an expert in your former scientific topic at the bench, but you are a novice in your new field, so learn as much as you can about it from as many people as possible during these conferences. The best way to do this is to attend lots of sessions that pertain to it (workshops, talks, and so on). This is particularly relevant in science policy, where the focus can shift quickly from one topic to another. Seek mentors and peers who can help; in my experience, people generally are willing to mentor someone else, especially if they have gone through a similar transition.

Be open to learning. If you are attending a large scientific conference as a science policy professional, for example, there may be fewer sessions in your field at that conference. Embrace this as a way to learn something different but still valuable (a new skill, topic or resource) that you can use for your own professional development. This includes meeting professionals in your field or developing interdisciplinary relationships by bringing your new expertise to bench scientists.

Talk to people. Meet as many people as possible in your area of interest at conferences. Present your work to experts in your field but also to those who have no idea what you do; both can be valuable experiences. Attend talks given by experts in your field at conferences, introduce yourself to them after the event, and set up one-on-one meetings to continue the conversation. You will learn from them, and they also will get to know you as a professional in the field. In addition, you can help others navigate a career transition by co-moderating a discussion table related to your particular area during a large conference.

Maintain connections. If you make a connection at a conference, follow up with that person; keep these connections going and seek to meet them again the following year. This way you can create a network of people who can help you advance in your career, tell you about the hot topics, recommend literature and other resources, as well as connect you with other experts in the field.

Be bold. If you are intimidated by a large conference, roundtables or poster sessions can be a great way to get to know people in your field and talk with them one-on-one. But ultimately, if you want to succeed as a professional, you need to be bold and do things you are afraid to do. You must take every opportunity to tell people about your work. This is the only way to grow and begin to make yourself and your organization known in your new field.

Engage at a broader level. Beyond conferences, get involved in national committees to find out about the hot topics in your area and make further connections. As an example, I joined committees that were relevant to my interests in training and policy, which has connected me with colleagues I can learn from and call on for my needs in the future.

Resources

These general resources related to training and policy may be useful during your career transition or for presenting at scientific conferences:

American Society for Biochemistry and Molecular Biology Grassroots Advocacy Network: asbmb.org/Advocacy/GrassrootsNetwork

American Association for the Advancement of Science Career Development Center: careerdevelopment.aaas.org

Graduate Career Consortium Ad Hoc Committees: gradconsortium.org/ad_hoc_committees.php

American Society of Cell Biology: ascb.org/advocacy

Conclusion

Attending conferences as a science policy researcher (or in any new role) can be very rewarding. You may find that your new field is not so different from your past academic life, especially if you are still collecting data, writing papers and presenting posters (as in my case). Although I left the bench, I never really left science (or even research). I have continued to grow professionally by presenting our work at Future of Research during scientific conferences to a new audience and in a variety of settings.

Adriana Bankston (abankston81@gmail.com) is a former bench scientist with a passion for improving training and policies for junior scientists. She is also a member of the board of directors at the nonprofit organization Future of Research.

Battling imposter syndrome and anxiety in the quest for an academic science career

Kelly Chacón is a third-year assistant professor in the chemistry department at Reed College in Portland, Oregon. She talks about her nontraditional career path, confronts the stigma surrounding mental health in academia and shares the experiences that fuel her scientific pursuits.

How did reach your current position?

I had a nontraditional path to science and academia. I dropped out of high school at 15 and went into food service. I did not obtain my general equivalency diploma until I was 22.

Once I caught the learning bug, a key decision was not to allow any "what-ifs" or naysayers to deter me from continuing to learn and progress. I had to begin my journey with prerequisite community college courses in math, writing and science. I decided to make a simple but firm commitment to do my best, to, above all, treasure the privilege of education, and, honestly, to see just how far I could go before it got too hard. As it turned out, it took a fair bit of time, but the sky was the limit. This is true for many of us if we stay positive, ask for help from allies and work really hard.

From a young age, I did not identify as science-minded. However, now looking back to childhood, I should have known that my love of inventing household shortcuts, identifying and foraging for edible local plants, and cooking were indicators of a scientific mind. But, as a society, we may overlook these types of traits in children (especially those from underrepresented groups and/or of low socioeconomic status) and neglect to cultivate those interests into a future science, technology, engineering or math career. So I first learned about biochemistry as a field when I was 24, in a prerequisite community college biology course, during a discussion of the Miller–Urey spark chamber experiment. I was fascinated by the idea that life could have arisen from just a pool of chemicals and wanted to learn more.

Were there times when you failed at something you felt was critical to your path? If so, how did you regroup and get back on track?

Sigh. Yes, there have been some tough spots. I very nearly failed one portion of my qualifying exam as a third-year graduate student and was very harshly criticized for it. The professor who administered that portion said a score so low was an indicator that I should not be in science. This came on the heels of trying to write my first scientific manuscript for publication, which also wasn't going so well and also had received some harsh criticism. Although many know about imposter syndrome, few realize how particularly intense and devastating it can be for those from underrepresented groups. I immediately went into a tailspin because I had worked incredibly hard on both tasks and it hadn't been enough. The most important thing that I did at that point was to seek out my university's mental health resources and find a qualified therapist to talk to. I was scared to seek help, and, in my family, mental health was not something people talked about. But getting help changed my outlook, and I still see a therapist from time to time, specifically to deal with imposter syndrome and anxiety as it affects me as a minority in academia. I speak out about this now to reduce the stigma.

What advice would you give to young persons from underrepresented backgrounds who want to pursue a career in science similar to yours?

A) Start doing manageable, small things that matter to you in your community, even though you think you have no time. Make the time. It will pay off in feeling good in the moment and in opportunities down the road.

B) Practice presenting your science in every venue that will have you. High schools, local meetings, national meetings, departmental seminars, courses and lab group meetings are all fair game. Ask for honest feedback

COURTESY OF KELLY CHACÓN

Kelly Chacón didn't think of herself as a science-minded child and earned her GED at the age of 22, but she is now a third-year assistant professor in the chemistry department at Reed College in Portland, Oregon.

and incorporate that feedback into your presenting skills.

C) Practice applying for small research grants, scholarships, travel grants and self-nominating awards. It feels difficult for those of us in underrepresented groups to toot our own horn, but those from privilege do these things without a second thought. Start getting comfortable with honestly and positively assessing your own strengths as a scientist and a person.

D) If you really, deep down, with all of your heart, wish to be in academia but are feeling insecure based on what other people have told you about the job market, stop waffling and make a firm decision to pursue your dream. Make it real by sharpening your focus: What schools/areas would you like to work in? What kind of research would you like to pursue? Then, look at those schools and what they value and represent. Start doing things early on that will contribute to your CV toward the goal of appealing to those schools. And then, when a job comes up, apply for it. Don't overthink, just try. And don't be afraid to ask for help from those who supported you along the way.

What are your hobbies?

Sewing and altering clothing (I am short), cooking exotic meals, playing steel-tip darts, drawing and painting, and playing video games like the Final Fantasy series and Fallout.

What was the last book you read?

"Wizard and Glass" by Stephen King. The Dark Tower series by King is some entertaining fiction.

How have heroes, heroines, mentors or role models influenced you?

My mentors and role models are the strong women in science who gave me blunt advice when I needed it. My heroine is mi abuela, Mama Cande. My grandmother was married at 14, could not read, and gave her life to her eight sons and her pueblo. She was so full of love, curiosity and acceptance, and I am driven to make her proud of me even though she is no longer with us.

What keeps you working hard every day?

Real talk: An app called Self Control that keeps me from playing on the internet while I work. But, intrinsically, what keeps me going is that I still, even now, want to see how far I can go before it gets too hard. And I want to contribute something small, before I leave this Earth, toward understanding how our amazing universe and life works.

The Research Spotlight

The American Society for Biochemistry and Molecular Biology's Research Spotlight highlights distinguished biomolecular and biomedical scientists from diverse backgrounds as a way to inspire up-and-coming scientists to pursue careers in the molecular life sciences. Eligible candidates include Ph.D. students, postdoctoral fellows, and new or established faculty and researchers. To nominate a colleague for this feature, contact education@asbmb.org.

The leap from two-year to four-year institutions

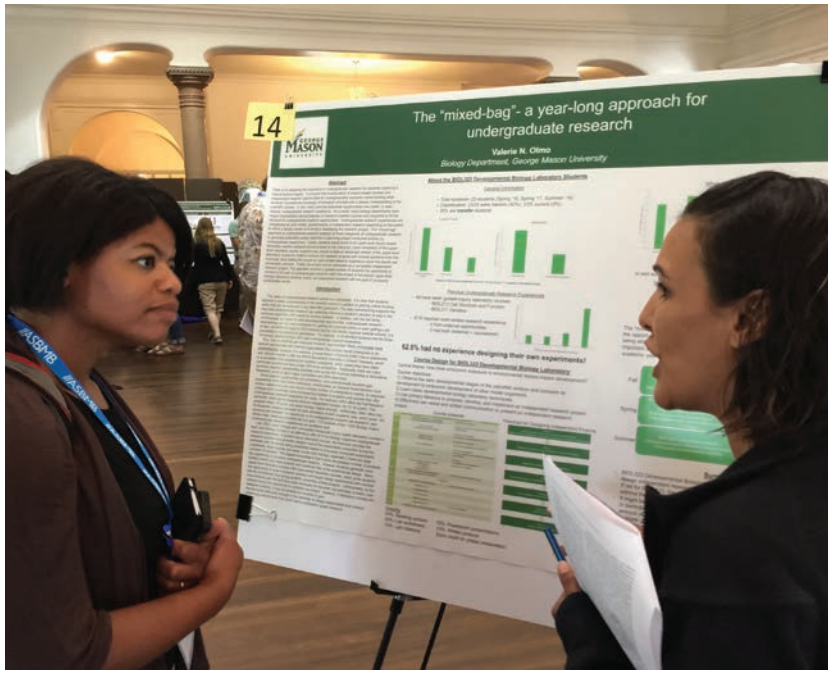
Building bridges in molecular life science education

By L. Michael Carastro, Jim Lawrence, Regina Stevens-Truss & J. Ellis Bell

Many students begin their undergraduate careers at community colleges, later transferring to four-year institutions, and science educators face a range of issues posed by these transitions. In the 2013-14 academic year, 46 percent of students who completed a degree at a four-year institution had been enrolled at a two-year institution at some point in the previous 10 years, according to the National Student Clearinghouse Research Center, and in 14 states, more than half of four-year degree recipients previously were enrolled at a two-year institution.

During a biennial symposium, "Transforming Undergraduate Education in the Molecular Life Sciences," sponsored by the American Society for Biochemistry and Molecular Biology, a diverse group of faculty, graduate students and postdocs addressed these and other issues. One panel discussion, "Bridging Community College to Four-Year College/University Transitions," included Jessica Schrader of Eastern Florida State College, James Wysong of Hillsborough Community College and Ellis Bell of the University of San Diego. The panel educators and administrators identified and explored transition issues in undergraduate bioscience education.

Challenges for these transfer students include the clustering of science classes in the latter half of a four-year degree. Many students who start at two-year colleges already have taken all, or most, of their general education courses. After transferring as science majors into four-year schools, however, they must take a daunting number of science classes in their last two years. Also, courses at two-year schools such as general or organic chemistry might not cover the same breadth and depth of material as the same courses at some four-year schools, which can result in these students being underprepared for subsequent science classes, such as biochemistry. In addition, whatever their majors, some students experience a type of cultural shock after transferring into a four-year school.



COURTESY OF L. MICHAEL CARASTRO

Valarie Olmo of George Mason University explains her research to Traci Addy of Yale University during the poster session/reception of the molecular life sciences symposium at the University of Tampa.

These issues must be addressed by increasing coordination and collaboration among educators and administrators at both two-year and four-year institutions to develop effective strategies and mechanisms. This goal initially must be approached through meaningful dialogue, such as that in the panel discussion at our ASBMB-sponsored symposium. Ideas voiced during our panel discussion included increased coordination between two-year and four-year institutional academic advisers serving students in molecular life sciences to address

Feedback

Organizers surveyed participants after the symposium in Tampa. Responses were anonymous and comments included the following:

"This is a great meeting! The community is very welcoming and kind to new attendees. The meeting also provides a lot of valuable information for improving education."

"I felt it offered great ideas of teaching styles. I told my sister who is a high school chemistry teacher she needs to attend in 2019!"

"Great meeting that is focused on undergraduate education for BMB fields. I haven't found anything else like it and look forward to attending the next one."

"Very informative. I learned different teaching techniques I was not aware of and how to possibly reach students that can be difficult to reach."

the issue of clustering classes in the last two years; collaborations among faculty involved in science courses to ensure content is more consistent at the different types of institutions; and helping provide students at two-year institutions with research opportunities at four-year schools prior to their transferring so these students are not expected to begin and complete their (sometimes requisite) research projects in a compressed time frame.

More topics

The four-day symposium, held in July at the University of Tampa, was the fifth iteration of "Transforming Undergraduate Education in the Molecular Life Sciences." These symposia focus on cutting-edge, high-impact teaching practices and strategies in molecular life science. These themes were woven into three meeting foci:

Classroom Undergraduate Research Experiences, or CUREs — Expert speakers presented a session on implementation of CURES and promoting and developing CURE faculty networks.

Early Career Faculty and Postdoctoral Fellows Teaching Development — Programs that have evidence-based records of good teaching practices were presented to encourage and prepare young scientists to consider becoming educators.

Student Chapters and Science Outreach Programs — Informal sessions focused on undergraduate outreach programs and the work of ASBMB Student Chapters to help build communities of science educators and science students who work to solve community problems.

The symposium attendees came from two-year community colleges, primarily undergraduate institutions and large research universities. Nearly one-fifth were graduate students, postdoctoral fellows or community college faculty.

The organizers decided early in the planning that postdocs and grad students should be included. This group typically does not receive training for a career in science education. In fact, there is a paucity of resources and opportunities for grad students and postdocs in molecular life sciences to gain experience in educating undergraduates. This symposium was designed to provide a career-changing opportunity to these young science educators. More than $30,000 in National Science Foundation funding helped pay for travel, lodging and registration for grad students, postdocs and local two-year college faculty.

Speakers and workshop presenters came from academia, government and the biotechnology industry, providing a range of perspectives. In addition to the discussion of transitions from two-year to four-year institutions, the symposium included an interactive workshop presented by Sherri Andrews of Bio-Rad, "The Student Lionfish Project: Facilitating Student Understanding of Methods and Data," designed to teach students the principles and utility of Sanger sequencing. Joseph Provost of the University of San Diego and Michael Pikaart of Hope College presented "CUREs: Building Communities to Support and Sustain Protein Biochemistry Research in the Teaching Laboratory."

To continue the conversations that began at the symposium and help implement change in attendees' careers, the organizers offered individual action plans and the development of mentor-mentee relationships. Those who expressed interest were encouraged to pair up and form a plan to continue their mentor-mentee relationship, including regular conference calls, site visits and sharing teaching experiences.

Undergraduate education in the molecular life sciences depends on our community working to develop future educators in the field as well as addressing the issues surrounding student transitions from community colleges into four-year educational institutions. Special symposia such as this one are a first step toward meeting these goals.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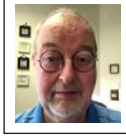
J. Ellis Bell (jbell@sandiego.edu) is a research associate professor in the department of chemistry and biochemistry at the University of San Diego.

I had my 'crazy' surgically removed

By Theresa Abell

Every doctor must know how to deliver bad news. As a patient, your reaction is unpredictable. It depends heavily on the severity of the diagnosis; upon being told you have cancer, fear, dismay and even disbelief may battle for dominance in your mind. For the doctor, perhaps the most unexpected response to telling someone they have a serious illness is a simple "OK," a nod and a moment of quiet thought. Even, secretly, a quiet sigh of relief.

About a year ago, this was my reaction when a skillful endocrinologist delivered the news. Luckily for me, my diagnosis was not as grave as cancer. I learned I had Cushing's disease, essentially an intense overproduction of cortisol by my adrenal glands resulting from a tiny noncancerous tumor pressing on my barely larger pituitary gland, located at the base of my brain. It may seem crazy to feel calm or relieved to learn that you have what amounts to a brain tumor. "Pituitary adenoma" is not a phrase little girls dream of hearing when they get older. However, this 7-millimeter blob of cells had been making me feel crazy for quite some time.

If you are a scientist, a certain way of thinking is indelibly ingrained into your being: identify a problem, determine its cause, and determine the most practical and efficient way of solving it. This process becomes hopelessly twisted when the problem lies within you, beginning with a vague feeling that something just isn't right. Vagueness is not appreciated in a scientific setting; the issue must be investigated, pinned down into something concrete or, likely to your own detriment, ignored.

COURTESY OF THERESA ABELL

Theresa and Matt Abell were married in September, six months after she had surgery to remove a tumor from her pituitary gland that caused Cushing's disease.

I had recently begun graduate studies in biology at Tufts University when the symptoms of Cushing's began to present themselves in earnest. I was constantly nervous, to the point that a tremor in my hands was noticeable when I was simply handing over my credit card in a checkout line. Well, of course I was nervous. This was a new, grand adventure for me, and I had no idea how it would go or where it would lead. I told myself this trembling must simply be a manifestation of an anxious personality. As the months passed, I began to gain weight. Again, I assigned a perfectly reasonable explanation: I was a grad student and I didn't have as much time as before to make sure I was eating well and exercising regularly. On top of that, I worked in a bakery/sandwich shop/pizzeria to pay the bills, and this being the source of an inordinate proportion of my meals was not doing any favors for my waistline. About six months into my grad-school career, I quit my bakery job in favor of a part-time job at a

biotech company to support myself during my studies, allowing me to keep one foot in industry and possibly leading to future career opportunities. I assumed that, with a more secure and interesting job and a little more comfort in my studies, things would return to normal.

As all bench scientists know, the results of what appear to be simple, rational, straightforward steps are often not what was expected. My anxiety worsened and progressed into a deeper moodiness and depression. I dieted and exercised to the limits of my ability and just barely maintained my weight. The slightest indulgence resulted in seemingly instant additional roundness to my abdomen and face. Shortly after beginning my new job, I began seeing a new primary physician and was grateful to have the sympathetic ear of someone who could give me what I truly wanted — tangible, quantifiable data that could be analyzed by experts to reach a conclusion. My own online research had yielded some potential causes for my symptoms, including hormonal disruptions that could result in emotional or physical changes, but none completed the puzzle neatly enough to be convincing. A metabolic panel to measure hormone levels, a blood pressure cuff to document the disconcerting spike in blood pressure that was the first obvious (to others) physical sign that my body was not behaving the way a normal 26-year-old body should, and conferring with a specialist in endocrine disorders: these were the missing pieces that could stitch together this mysterious malaise.

So with a peculiar gusto, I charged forward into blood tests that led to CAT scans that led to MRIs, each methodically eliminating one possibility and leading to the only sound conclusion to be drawn from a mounting pile of evidence. When I received a DVD containing the MRI scans of my head, I immediately sat down with my then-fiancé (now husband) to scroll through them all, fascinated by the literal peek into my own brain. I only wished that I was better equipped to interpret what I was seeing. He once asked me if I worried about the radiation I was being exposed to as a consequence of all these tests. Though I appreciated it, I waved away his concern — what a small price to pay for the answers for which I was so impatiently waiting. Once I was able to give it the name of Cushing's disease, a precise and meaningful summation of a long and arduous journey, I did not balk at the fact that the only viable treatment was brain surgery. In fact, I spent many hours doing my own research on the technique, endonasal endoscopic transsphenoidal pituitary surgery. I found YouTube videos of the procedure and shared them with (at times rather less fascinated) friends and family. I found out that my neurosurgeon had performed this surgery over 100 times and that it was relatively simple and noninvasive as brain surgeries go. It would involve a light, a camera and a surgical instrument going through my nose to reach the offending tumor that was to be removed from my pituitary gland.

From then on, everything went as smoothly as I was assured it would. After being diagnosed in December, the procedure was done in March, six months before my wedding and right in the middle of my last semester of school. The timing was stressful, but the results were worth it. The weeks immediately following the surgery found me utterly depleted of energy and thoroughly terrified of sneezing, lest I implode the delicate healing process. However, once my body recovered from the shock of the surgery I began to feel like my old self again. My energy returned, I lost a lot of weight, and I once again enjoyed my work and my studies rather than stressing out about every tiny thing. My wedding in September was everything I ever dreamed it would be.

My only complaint was that I did not get a surgery video of my own.

Theresa Abell (farrell.t89@gmail.com) recently received a master's degree in biology, with a concentration in molecular biology, from Tufts University. She works in the biotech industry as a molecular biologist.

WHEN SCIENCE MEETS SICKNESS

DEADLINE EXTENDED

Are you a scientist who became a patient? ASBMB Today wants your story.

Does your understanding of biology make the diagnosis and treatment easier or more difficult? To share your story, be honest and true. Be open to editing and coaching. Your essay must be unpublished and between 500 and 1,000 words. Submit at asbmbtoday.submittable.com, "Science meets sickness."

2018: Back to the lab

By Daniel Pham & Andrè Porter

Hey there, grad students. Hope you had a wonderful winter break — eating, drinking, sleeping late, catching up on all those Netflix shows. But now it's over. And here you are at the bench again. Ouch. How do you climb back on that horse? As a public service, we offer 15 helpful tips for your first day back to lab in 2018.

1. Do: Gather that mental strength from the holiday break to analyze the Excel file with 2,000 rows of data and 38 tabs. **Don't:** Assume the lab elves have completed your analysis.

2. Do: Talk to your PI/advisor about your project's progress. **Don't:** Put that discussion off until your parents are planning your graduation party.

3. Do: Check if your cells are still alive before subjecting them to your experiments. **Don't:** Use that yeast plate from two weeks ago without checking. It could be making beer by now. (Mmmmm … beer.)

4. Do: Clean out your email inbox, saving any messages from biotech companies offering free 2018 calendars. **Don't:** Delete all unread messages from the holidays. Think of all those beautiful fluorescent cell images!

5. Do: Talk science with other disciplines. **Don't:** Retreat back to your department when that voice in your head says, "You know nothing, Jon Snow."

6. Do: Use the new laptop you got

during the holiday sales before your spilled coffee breaks the space bar, again. **Don't:** Use your new drone to deliver glove boxes to the neighboring labs.

7. Do: Practice explaining your research to a nonscientific audience. (We can help you at asbmb.org/Outreach.) **Don't:** Get frustrated when people ask you why they don't have a clone of themselves yet.

8. Do: Practice the new breathing exercises you learned while dealing with the holiday family stress. **Don't:** Plan your revenge on that aunt who exclaimed loudly at the holiday party that she couldn't believe you still haven't graduated.

9. Do: Take down the Star Trek-themed holiday lab decorations. **Don't:** Hang all the lab decorations around your own desk in a desperate attempt to relive the holidays.

10. Do: Reflect on whether your family actually understood your explanation about your research project. (See No. 7.) **Don't:** Send them scientific reviews and papers for "light reading," assuming they are now experts in your field (unless they actually are).

11. Do: Plan for life after you complete your degree. **Don't:** Plan to become a professional student. That 401K isn't going to appear by magic. (See No. 1.)

12. Do: Bring desserts and treats from your travels to share with the lab. **Don't:** Bring the leftover eggnog to share with the lab.

13. Do: Look for professional development opportunities. (Check out resources at asbmb.org/careers/careerdevelopment.) **Don't:** Neglect opportunities to socialize with multicellular organisms outside of the lab.

14. Do: Find a constructive/silly hobby to give your brain a break. **Don't:** Become a petri dish zombie.

15. Do: Start writing that review you were supposed to have finished by the end of 2017. **Don't:** Think about how many New Year's resolutions you've already broken.

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Andrè Porter (aporter@asbmb.org) is the science policy analyst in the public affairs department at the ASBMB.

I AM A

COLLABORATOR

And I advance my research when I exchange ideas with scientists in related fields.

The 2018 ASBMB Annual Meeting provides countless occasions to step outside your niche and collaborate with scientists with different strengths. Thanks to the ASBMB's affiliation with the Experimental Biology conference, you'll have the opportunity for interdisciplinary learning and exchange. The event represents five host societies and multiple guest societies and attracts more than 12,000 scientists and exhibitors from throughout the biology research sphere.

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